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Obesity: Body Relief Surgeries before Bariatric Surgery for Risk Reduction

José Humberto Cardoso Resende[1], Ana Letícia Pinto Guimarães[2], Brunna Abreu Perillo[2], Ana Carolina Melo Maluf[2], Laura Ribeiro da Costa[2], Rafaella Cristina Gomes Bernardes[2], Arthur Camargo Pires[2], Thalles Gonçalves Souza Menezes[2], Murilo Calil Alves[2], Emídio Silva Falcão Brasileiro[3]

[1]UNIFAN, Work Coordinator, Aparecida de Goiânia, Brazil
[2]UNIFAN, Medicine Academics, Aparecida de Goiânia, Brazil
[3]UNIFAN, Research Coordinator, Aparecida de Goiânia, Brazil
Email: jresen99@hotmail.com

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Abstract

Depending on the treatment and weight of the breasts or abdomen, they may exceed volumes considered giant and morbidly obese. In these cases, and when the patient's BMI is high above 40 kg/m^2, the weight of the breasts or abdomen produces what we consider suffocation when the patient is placed in horizontal position on surgical tables, decreasing his respiratory capacity and increasing the difficulty in treating respiratory or embolic risks. An 8-kg breast on the patient's chest prevents normal breathing. An abdomen with a volume of 30 kg causes difficulties in all senses, making the physiological expansion of the lungs impossible and even preventing surgical assistance to patients. These patients are almost always customers who sleep in the sitting position to breathe better. The gigantic extirpation of the surgical parts facilitates a better respiratory expansion reducing by a large percentage the risk of death, what we call body relief. This relief does not free the patient from bariatric surgery for a possible weight loss, which is vital for the proper functioning of the organs and decreasing arterial hypertension and diabetes.

Keywords

Obesity, Disease, Surgery, Bariatric, Risk, Hypertension and Diabetes

1. Introduction

The risks and impossibilities of the human being are directly linked in alarming percentages to cases of morbid obesity, when the Body Mass Index (BMI) reaches values far beyond normality or beyond the phase when the person is called

"chubby" [1]. The largest amount or surgical part we removed was of 32 kg, but the average in cases of extirpations for body relief is of 8 kg (**Figure 1**). These surgeries require a larger number of surgical assistants, a prepared nursing team, a proper operating table, a larger surgical team, and surgical preparation under a thorough and complete anamnesis [2]. In all cases, we always monitor a weight loss by own free will of 8 to 10 kg before surgery. Only then does relief surgery take place. After 6 months, we release the patient for bariatric surgery or intestinal and gastric bypass to achieve the desired weight loss (**Figure 2** and **Figure 3**) [3]. All patients followed the preoperative protocols of the Plastic Surgery Service, such as: hematocrit greater than 38%; blood pressure less than 150 × 100 mmHg; blood glucose less than 100 mg/dL; normal chest X-ray; surgical risk and electrocardiogram approved by the cardiologist.

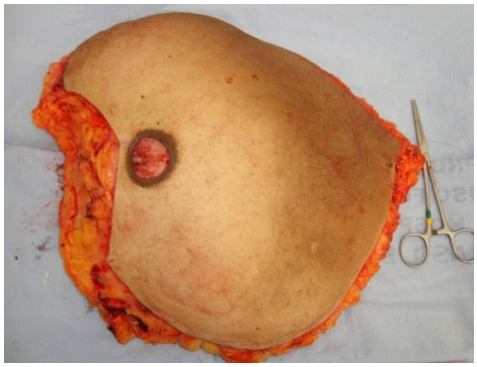

Figure 1. Surgical specimen.

Figure 2. Pre-surgery.

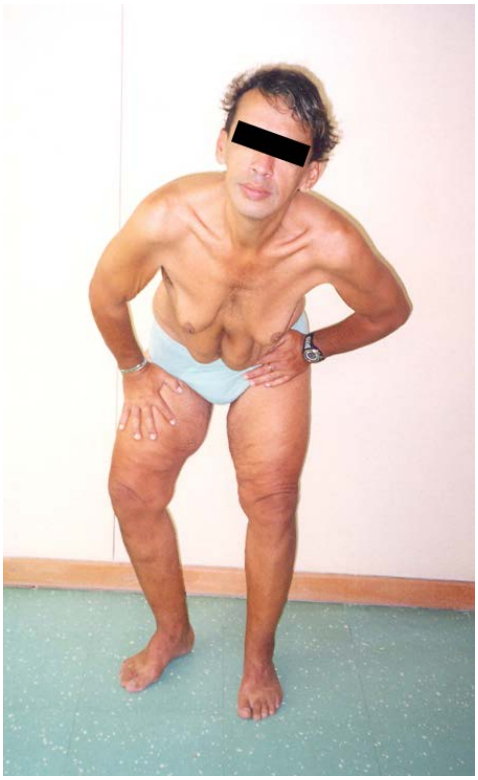

Figure 3. Post-surgery.

2. Material and Method

The patients mentioned in this manuscript are often abandoned in countryside cities, receive little attention and are neglected even by physicians due to lack of conditions to provide adequate care to alleviate the tensions and provide a better quality of social life [4]. We consider these as public health cases. We selected some patients to show our intention to achieve satisfactory results with zero death.

Metabolic surgeries are indicated in the case of two more harmful pathologies, namely, breast gigantism or abdominal gigantism. These are rare cases, because morbid obesity usually causes deformation of the whole body. Hypertension and diabetes are common in all cases. All patients sign the Informed Consent for the surgeon and for the hospital where they will be operated. They are aware of the possible surgical risks. They also authorize the use of pre- and post-operative images for scientific articles.

Breast gigantism: We have published extreme cases in which breasts prevented women from working to provide for their own livelihoods with professions such as ironers, cleaners, washers, seamstresses and others (**Figure 4** and **Figure 5**). Furthermore, obesity is a disease that causes hypertension and diabetes. It affects in all cases the ability to walk and the quality of life of these patients. Pre-operative care is meticulous; the patient goes through several workshop sessions (meetings) for clarifications and is informed about post-operative care measures.

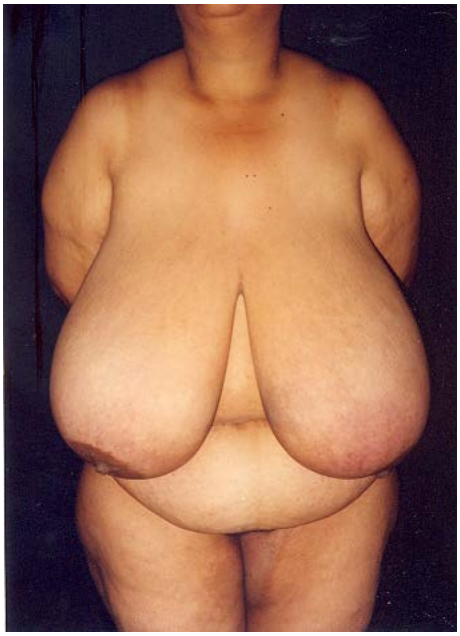

Figure 4. Giant breasts.

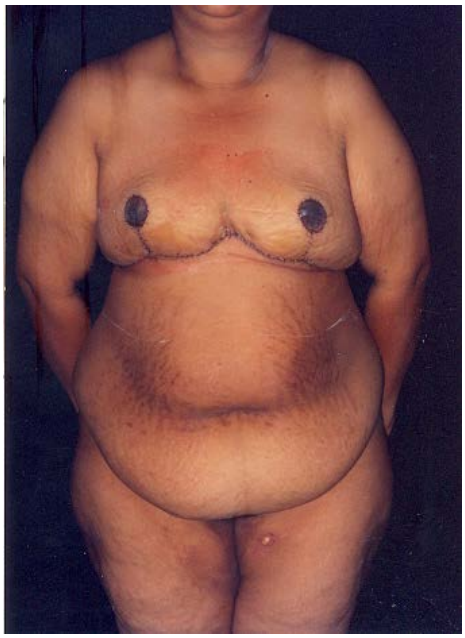

Figure 5. Post-operatory of reduction mammoplasty.

Abdominal gigantism: Distinction of sex becomes difficult in extreme cases (**Figure 6**), except for the beard when present. Surgical incisions in body relief surgeries are difficult and delicate because of the weight that medical assistants will have to handle. Hernias and eviscerations are frequently found during surgery (**Figure 7**). The postoperative is delicate and strict to avoid bruising and surgical wound dehiscence due to weight and change of position. The nature of the material is human and all methods are of gigantic and terrifying amputations.

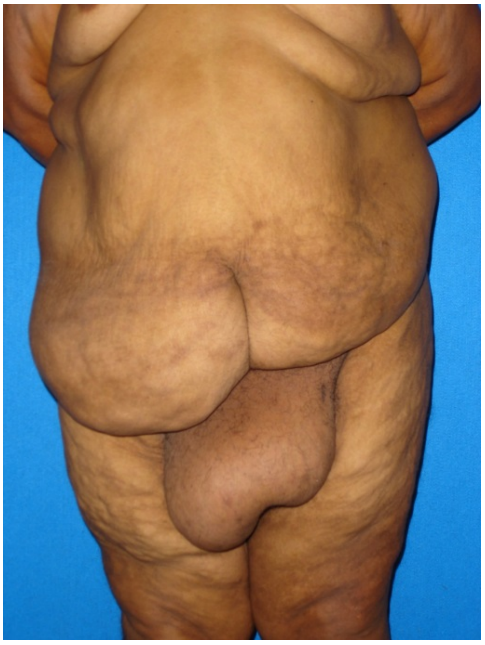

Figure 6. Male abdomen.

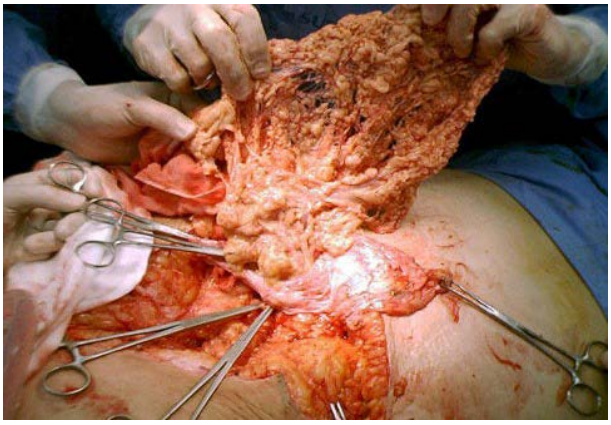

Figure 7. Hernia and evisceration.

In all cases of obesity surgeries, complications may happen in the post-operative period, including surgical site infection, seromas, surgical wound dehiscence, bleeding, deep thrombosis, and respiratory [5] or embolic infections. Cases reach a rate of up to 10%. According to Sergerman (1997), seromas can sometimes be treated at home or on an outpatient basis. Fistulas have decreased considerably in the last 30 years of history of bariatric surgery [6].

After Roux-en-Y gastric bypass, more control over morbid obesity was possible [7]. The hospital mortality rate was 5.5 per 1000 patients in the Unified Health System (SUS) in recent years [8] [9]. This rate varies from hospital to hospital and between different countries. The nutritional problem after bariatric surgery [10] deserves attention, including B12 deficiency. Nutritional issues are also present after body relief surgeries. In the United States, obesity has become synonymous with heart disease and causes of death [11].

3. Discussion

Many medical experts have sent these patients to specialized hospitals, which are non-existent in Brazil yet, but we are engaged in their implementation by means of the submission of projects to competent agencies. Cases like these are not so rare, and they imply a loss of self-esteem and will to live, as these patients think that they will not find doctors willing to assume the almost impossible task of helping them return to society healthy and enter the job market. In almost all cases, obesity is treated with general surgery, and there are no specific services aimed at obese people.

Body relief cases are usually addressed in plastic surgery services. We have not yet been supplied with suitable surgical centers with wide surgical tables, stretchers with larger dimensions, and increased circulation space for the work of the professionals involved in these cases. We created a masonry toilet of lower cost and stronger material and wider doors in order to facilitate the transport of obese patients.

Each bariatric surgeon will choose the most appropriate technique for each patient. We advise all obese people to participate in the Obesity Workshops, which are meetings where comprehensive instructions relevant to the pre- and postoperative procedures are provided, useful for all cases, including feeding and changes in behavior and habits.

4. Conclusion

We need to publish works and open the path for other physicians to deal with this situation. We need to have the boldness to raise awareness among national leaders about the urgent need to create specialized hospitals to assist part of the morbidly obese population, as these people are on the fringes of society, deprived of conditions or will to live. We go to the point of the exaggeration of suggesting the creation of new entities, such as a Post-Weight Loss Plastic Surgery Association, in order to provide more specific care for obese people, allowing the creation of more services for the preparation of medical residents.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Resende, J.H.C. (2008) Treaty of Plastic Surgery in Obesity. Ed. Rubio, Brazil.

[2] Benotti, P.N. and Force, R.A. (1995) The Role of Gastric Surgery in the Multidisciplinar Management of Severe Obesity. *American Journal of Surgery*, **169**, 361-367. https://doi.org/10.1016/S0002-9610(99)80177-9

[3] Brazilian Society of Bariatric and Metabolic Surgery. http://bvsms.saude.gov.br/bvs/dicas/124cir_bariatrica.html https://www.sbcbm.org.br/tecnicas-cirurgicas-bariatrica/

[4] Resende, J.H.C., de Moura, Á.I., Mariano, A.C.A., Silva, H.K., Silva, H.L., Campos,

I.A., de Lima, L.C.F., Gabriel, M.B. and Silverio, W.O. (2019) Gigantomastia in Female Workers: "Public Health Cases". *Modern Plastic Surgery*, **9**, 1-7. https://doi.org/10.1016/S0002-9610(99)80177-9

[5] Brazil. Ministry of Health (2000) Ordinance GM/MS nº 196, of February 29, 2000. Brasília-DF.

[6] Sugerman, H.J. (1997) Morbid Obesity. In: Zinner, M.J., Ed., *Maingot's Abdominal Operation*, 10th Edition, Appleton & Lange, Connecticut, 1057-1077.

[7] NIH Conference (1993) Gastrointestinal Surgery for Severe Obesity. Consensus Development Panel. *Annals of Internal Medicine*, **119**, 646-649.

[8] Christou, N.V. and Efthimiou, E. (2009) Bariatric Survey Waiting Time in Canada. *Canadian Journal of Surgery*, **52**, 229-234.

[9] Summer, A.E., Chin, M.M., Abrahm, J.L., *et al*. (1996) Ekevated Methylmalonic Acid and Total Homocysteine Levels Show High Prevalence of Vitamin B12 Deficiency after Gastric Surgery. *Annals of Internal Medicine*, **124**, 469-476. https://doi.org/10.7326/0003-4819-124-5-199603010-00002

[10] Bordalo, L.A., Mourão, D.M. and Bressan, J. (2011) Nutritional Deficiencies after Bariatric Surgery—Why Do They Occur? Review Article. *Acta Médica Portuguesa*, **24**, 1021-1028.

[11] Benalcazar, D.A. and Cascella, M. (2019) Obesity Surgery Pre-Op Assessment and Preparation. Department of Surgery, Columbia University, New York.

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Botulinum Toxin Type A and Its Possible Mechanisms on Wound Healing

Khushboo Gurung[1], Qian Tan[1,2*], Ye Xu[2], Bingchen Duan[3]

[1]Department of Burns, Plastic and Reconstructive Surgery, Nanjing Drum Tower Hospital, Affiliated to Medical School of Southeast University, Nanjing, China
[2]Department of Burns, Plastic and Reconstructive Surgery, Nanjing Drum Tower Hospital, Affiliated Hospital of Nanjing University, Nanjing, China
[3]Nanjing Drum Tower Hospital, Clinical College of Nanjing Medical University, Nanjing, China
Email: *smmutanqian@sina.com

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Abstract

Botulinum toxin type-A (BTX-A), a subtype from known seven types of botulinum neurotoxin (serotype A-G), is produced by a gram-positive bacterium, *Clostridium botulinum*. The toxin is now widely and efficiently used in treating a plethora of diverse symptoms and conditions. Recent evidence in the literature also shows that BTX-A exhibits a wide range of effects on non-neuronal cells. Its potential has markedly expanded to clinical applications other than the treatment of neurological and muscular conditions that are characterized by neuronal hyperactivity. A number of studies have shown BTX-A to improve the quality of scar outcome and prevent the formation of keloids and HTS. Although the mechanism of action of BTX-A on wound healing is still not clearly understood, lately there has been extensive research to grasp the underlying mechanisms of this multifunctional toxin. BTX-A seems to affect wound healing by a number of mechanisms that include action on tensile forces, inhibition of fibroblasts differentiation, downregulation of TGF-β1 and collagen expression. This review will explore the responses of Botulinum toxin type-A on wound healing and preventing pathological scars like HTS and keloids, and comprehend the overall effect BTX-A has on wound healing.

Keywords

Wound Healing, Botulinum Toxin Type-A, Hypertrophic Scar, Keloid, Fibroblast, Myofibroblasts, Transforming Growth Factor β-1, Collagen, Metalloproteinases, Connective Tissue Growth Factor

1. Introduction

Scars, especially hypertrophic or keloids have been of particular interest in plas-

tic and reconstructive surgery due to their recurrences and the therapeutic dilemma in treating them successfully. Disfiguring scars can be very distressful to patients regarding their appearance, particularly when located in the noticeable areas like head and neck; and can have an economic burden and psycho-sociological impact on the patients [1] [2]. Wound healing in itself is a very complex process involving various components and synchronous events that interplay as a consequence of chemical signaling between the various cells involved. Slight imbalance in any of these fragile processes can result in extensive fibrosis and give rise to abnormal scarring like HTS and keloids. So, many of the treatment modalities available these days have focused on this principle to control and prevent extensive hyper-proliferation of scar tissues. The benefits of early intervention on the outcome of various types of scars have been documented in the literature, yet no consensus on prevention or optimal treatment regime for scars has been manifested till now [3]. Other than intralesional steroid injection, application of pressure garments, laser and radiation therapy, scar excision, or various other methods, there is a lesser known treatment of injecting botulinum toxin type-A intralesionally that has been successfully reported over the years in the literature. The first complete description of clinical botulism ("sausage poisoning") published by Justinus Kerner was in the year 1820 [4] [5], and since then the application of botulinum toxin has been very wide and versatile. Hagenah *et al.* used BTX-A as a research tool to study the spinal cord physiology and published reports in 1977 [6]. Perception of botulinum toxin began to slowly change over the years, and in the early 1980s it took a turn when its therapeutic potential was finally realized. Over the years, it has been commonly used in medicine to treat chronic myofascial pain [7], headache [8], hyperhidrosis [9], urinary incontinence [10], various muscle spasms and diseases characterized by overactive muscle, or more recently in cosmetic applications [11] [12]. Several commercial toxins are available in the market under the brand names such as Botox and Dysport, among many others [13] [14].

1.1. BTX-A: Structure, and Mode of Action

Botulinum toxin-A released in its inactive form is a complex mixture of single polypeptide chain consisting of botulinum neurotoxin-A with a nontoxic non-hemagglutinin protein (NTNH) and other various associated non-toxic proteins (HA-hemagglutinin protein). When cleaved by its own proteases, it is converted into an active form consisting of a light chain (L; 50-kDa), and a heavy chain (H; 100-kDa), with 3 domains weakly held together by a peptide belt, a disulfide bond, and surface charges [15] [16]. Botulinum neurotoxin causes flaccid paralysis by binding pre-synaptically to high-affinity receptors on the cholinergic nerve terminals and thus inhibiting the release of acetylcholine from presynaptic neurons at the neuromuscular junction (NMJ) resulting in chemical denervation at the motor end plate [17].

The mechanism of nerve terminal intoxication by the botulinum neurotoxin is divided into five major steps, 1) binding to neuronal cell membrane (dual-receptor

complex), 2) botulinum neurotoxin-receptor complex internalization within an endocytic compartment, 3) low pH driven translocation of L chain across the vesicle membrane, 4) Thioredoxin (Trx) catalyzes the reduction of a disulphide bond releasing L-chain into the cytosol, and 5) cleavage of soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) substrate, and ensuing inhibition of membrane fusion and acetylcholine (ACh) release, thus leading to neuroparalysis as shown in **Figure 1** [18] [19]. Proximal axonal sprouting and muscle re-innervation by the formations of new NMJ leads to toxin recovery [20].

1.2. The H Chain

The H chain has two domains, HN—a translocation domain, and HC—a receptor-binding domain. The HC domain can be further divided into two distinct subdomains: 1) a carboxyl-terminal β-trefoil (HCC), and 2) an amino-terminal lectin-like jelly roll (HCN) [21]. The H chain provides specificity and binds the toxin to presynaptic receptors, and promotes L-chain translocation across the endosomal membrane. The HN domain has a central role in the translocation of the L chain across the membrane of the endocytic vesicle into the neuronal cytosol [16]. The HCC (binding domain), mediates neuro-specific binding of the toxin to a polysialoganglioside and also binds to a protein receptor, together they form a dual-receptor complex. This results in an accelerated and active interaction of the toxin with peripheral cholinergic nerve endings [22] [23] [24]. Although specific function of lectin-like HCN domain is yet unknown, evidence suggests it may promote attachment of BTX-A to the pre-synaptic membrane by interacting with certain anionic lipids [25].

1.3. The L Chain

The L chain is a metalloproteinase, possessing a zinc endopeptidase from the M27 family of peptidases, and is an active part of the toxin. It enters peripheral cholinergic nerve terminals where it cleaves the synaptosomal-associated protein SNAP-25 (SNARE protein family): a presynaptic membrane protein required for fusion of neurotransmitter containing vesicles [19] [26]. It impedes vesicle-plasma

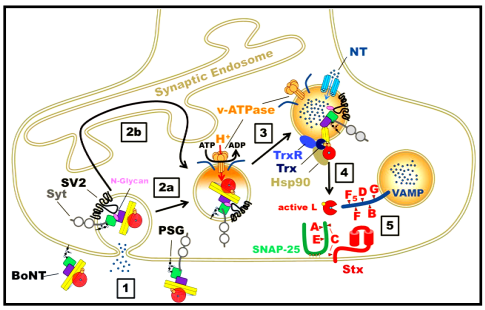

Figure 1. Mechanism of intoxication by various *Botulinum neurotoxins*, A-G. (PSG, polysialoganglioside; Syt, synaptotagmin; SV, synaptic vesicle; Stx, syntaxin; Trx, thioredoxin; TrxR, thioredoxinreductase; VAMP, vesicle-associated membrane protein).

membrane fusion resulting in persistent but reversible inhibition of acetylcholine release from axon endings producing flaccid paralysis [17] [27]. The seven toxin types (A-G) have different tertiary structures and sequence differences, and different toxin types target different SNARE family members [28]. This specificity of action has made them effective therapeutic agents for various syndromes caused by overactive cholinergic nerve terminals. The scope of clinical applications of BTXA is rapidly growing and is now being substantially used to treat a broad range of clinical conditions [17].

2. Wound Healing

To understand the mechanisms of BTX-A on the outcome of scars, comprehending the process of wound healing is very crucial. When the skin is broken, an intricately regulated process involving multiple overlapping sequences of biochemical events are set into motion to repair the damage. The phases of wound healing are: 1) hemostasis, 2) inflammatory response and cellular migration, 3) proliferation (neo-angiogenesis, formation of granulation tissue and ECM, re-epithelialization), and 4) tissue remodeling. Upon wounding, initial vasoconstriction along with activation of the clotting cascade achieves hemostasis. Platelets that come in contact with the exposed sub-endothelium, tissue factor and collagen lead to further platelet aggregation and degranulation [29]. Platelets release various chemokines, growth factors (GFs like TGF, PDGF, etc.), and other pro-inflammatory mediators, which induce migration of a variety of cells into the wound site [30]. In the inflammatory phase, the actions on cell receptor by these cytokines, chemokines, GFs, proteases, pH gradients, pO_2 gradients modulate intracellular signaling cascade that results in cell proliferation, migration, and differentiation [27].

Activated by various GFs like TGF-β family (TGF-β1, β2, and β3), IL family and angiogenesis factors (VEGF), the population of fibroblasts, keratinocytes, endothelial cells, and connective tissue greatly increases in the proliferative phase. Fibroblasts lay down ECM proteins including proteoglycans, fibronectins, and hyaluronic acid at the wound site and produce collagen and fibronectin, and eventually replace the temporary fibrin clot [31]. Thus formed granulation tissue is heavily vascularized by local angiogenesis [31] [32]. Different types of cells that are involved in the process of inflammation, neo-angiogenesis, and connective tissue synthesis adhere to, proliferate and differentiate on the collagen matrix produced and laid down by the fibroblasts. Whereas, myofibroblasts (transformed fibroblasts) connect to the surrounding protein collagen and fibronectin, assisting in wound contraction and promoting angiogenesis through matrix metalloproteinases (MMP) [33]. Remaining epithelial appendages near the wound and epithelial cells from around the wound border continue to repair the epidermis [34]. This phase continues for days and weeks [35].

The final step in wound healing is the remodeling phase and can take up to one or more years [29]. This stage in the wound healing needs a precise balance between the synthesis and degradation of ECM. Fibroblasts regulate both the

turnover and gradual breakdown of ECM by MMPs, and replacement of immature type III collagen by mature type I collagen [29] [30] [32]. As the scar matures over time, vascularity decreases and scar color changes, but only partially regains its original tensile strength. Although repair of the functional barrier is highly efficient, it does not always operate properly, and any disruption in the aforementioned processes will lead to uncontrolled wound healing resulting in two major pathological states: ulcerative defect or excessive scar formation like a hypertrophic scar or keloid [33] [35].

Hypertrophic scars (HTS) and keloids both are raised, thickened and firm scars formed due to overexpression of collagen during wound healing. Even though both can be pruritic, painful, it is also restricting and disfiguring, and there is a clear distinction between the two [36]. Unlike HTS, keloids are not contained within borders of the original injury, tend to overgrow into large benign tumors, can recur after excision, and are genetically predisposed. Often times HTS is self-limiting and can fade with time. However, studies have suggested hypertrophic scarring to be prevalent in darker skin populations and areas of skin subjected to tension [30] [36].

3. Effects and Potential Mechanisms of BTX-A on Wound Healing

3.1. Direct Inhibition of Active Fibroblasts and Increased Apoptosis

Over the years, there have been several in vitro as well as *in vivo* studies that have concluded that BTX-A is not only a neurotoxin but tends to have a molecular effect on a cellular level, particularly the inhibition of fibroblasts. Shown in Table 1 is a summary of studies which have indicated that BTX-A not only directly inhibits the proliferation but also affects the cell cycle distribution of fibroblasts derived from keloid, HTS, and scar contracture tissues when compared with normal fibroblasts [37]-[44]. Zhibo and colleagues conducted several studies to show the effect of BTX-A on fibroblasts derived from HTS and keloids. In one study, fibroblasts treated with BTX-A at the dose of 1 U/10^6 cells and 2.5 U/10^6 cells showed 58% and 61% in G0 to G1 phase, 8% and 9% in G2 to M phase, and 34% and 30% in S phase, respectively, showing a greater population of fibroblast arrested in G0 to G1 phase [40]. BTX-A effectively inhibited the proliferation of fibroblasts derived from HTS and also decreased CTGF protein thereby preventing excess collagen deposition. In another experiment, 64% of BTX-A treated fibroblast were in G0 to G1 phase compared to only 36% of control fibroblasts; a greater number of the control fibroblasts were in the proliferative phase (21% G2 to M; 43% in S) [45]. In yet another study by Zhibo *et al*., keloids treated with intralesional BTX-A (70 to 140 U) per session at a 3-month interval for a maximum of 9 months showed striking outcomes. With no therapy failures and very high patient satisfaction 25% of subjects reported excellent, 41.6% good, and 33% fair [43]. Another similar study with 19 patients treated

Table 1. Effects of botulinum toxin type-A. Summary of studies included.

First Author (year)	Study type/Details	Type of Scar/Cell	Toxin Application	Results
Xiao Z *et al.* (2009) [44]	Prospective uncontrolled trial. 6 months follow-up	Active HTS from 19 patients	Intralesional BTX-A injection of 2.5 U per cubic cm of lesion, once monthly for 3 months	All patients showed acceptable improvement and high rate of therapeutic satisfaction. Scores for erythema, itching sensation, and pliability post BTX-A injections were significantly lower than prior to BTX-A injections ($p < 0.01$).
Xiao Z *et al.* (2011) [40]	*In-vitro*/cell culture study	HTS derived FBs from 8 different patients	3 groups; cells treated with BTX-A in concentrations of 1 U/10^6 cells vs. 2.5 U/10^6 cells vs. control	Proliferation of FBs treated with BTX-A was slower than of FBs without BTX-A ($p < 0.01$). Compared with FBs without BTX-A, BTX-A at 1 U/10^6 cells decreased expression of CTGF by 49.2% ± 12.5% ($p < 0.01$), and BTX-A at 2.5 U/10^6 cells, decreased CTGF expression by 56.9% ($p < 0.01$).
Zhibo X *et al.* (2008) [45]	*In-vitro*/cell culture study	FBs cultured from HTS of 8 different patients	FBs in 1 U/10^6 BTX-A vs. control FBs	Significant differences in cell cycle distribution between experimental (64% in G0 - G1, 6.4% in G2-M, 29% in S phase), compared to control FBs (36% in G0 - G1; majority in proliferative phase; 21% in G2-M, 43% in S), ($p < 0.01$).
Jeong HS *et al.* (2015) [37]	*In-vitro*/cell culture study	HTS derived FBs vs. normal mature scar derived FBs	FBs treated with BTX-A 4 U/ml vs. control	FB proliferation in both normal mature scar and hypertrophic scar tissue decreased significantly after treatment with 4 U/ml BTX-A ($p < 0.001$). α-smooth muscle actin mRNA and proteins also decreased in BTX-A treated group compared to control (TGF-β1 only) of FBs derived from HTS, but not FBs derived from normal mature scars. FBs to myofibroblasts differentiation decreased in FBs of HTS after BTX-A treatment.
Xiao Z Qu G. (2012) [39]	*In-vivo* experiment (animal model)	HTS of 8 different rabbits ears.	Rt. ear injected with BTX-A (0.5 U per cubic cm, once a month for 3 months), Lt. ear as control	Thicknesses of HTS in BTXA group were lower than in control groups ($P < 0.01$). Collagen fibers were thicker and arrangement of fibers was disordered in control group than in BTXA group.
Xiao Z *et al.* (2010) [42]	*In-vitro*/cell culture study	HTS tissue obtained from 8 different patients	FBs treated with BTX-A concentration of 1 U/10^6 cells, 2.5 U/10^6 cells, and control	TGF-β1 concentration per cell in FBs without BTX-A was higher than in FBs with BTX-A ($p < 0.01$). Significant difference noted in TGF-β1 production per cell between FBs treated with 1 U/10^6 cells of BTX-A and FBs treated with 2.5 U/10^6 cells of BTX-A ($p < 0.01$).
Hao R *et al.* (2018) [46]	*In-vitro* study	Human Keloid FBs vs. Normal FBs	FBs treated with different concentrations of BTX-A (0.01, 0.1, 1 and 10 U/L)	Viability of Keloid FBs decreased with increasing BTXA dose. BTXA inhibited proliferation, and S phase of Keloid FBs. MMP-1, -2 RNA and protein showed high expression, but TGF-β1 and MMP-9 showed low expression than control.
Chen M *et al.* (2016) [47]	*In-vitro*/cell culture study	Scar contracture tissue from 10 patients	BTX-A concentrations of 1 U/10^6 cells, 2.5 U/10^6 cells, and control	FBs without BTX-A treatment had higher proliferation than groups with BTX-A; proliferation of FBs significantly inhibited by BTX-A ($p < 0.05$). BTX-A also inhibited protein of α-SMA and myosin II in FBs treated with BTX-A compared to FBs without BTXA ($p < 0.05$).
Liu DQ *et al.* (2017) [72]	*In-vivo* experiment (animal model)	HTS of 18 different rabbit ears	4 groups: 12 ears as BTX-A (0.5, 1.0, 1.5, 2.0 IU), 12 ears as triamcinolone acetonide (TAC) group, 12 ears phosphate-buffered saline (PBS), and healthy skin as control	Mean hypertrophic index of HTS with BTX-A (2.0 IU) were lower than that of control ($p < 0.05$). BTX-A and the TAC group showed significantly less expression of collagen fibrils compared to PBS. BTXA (2.0 IU) and TAC significantly reduced FBs compared to control group.
Lee BJ *et al.* 2009 [73]	Prospective randomized experimental study	Surgical skin wounding on the dorsum of rat	10 U, 0.5 mL BTXA injected in one wound and normal saline injected into adjacent wound as control	Significant differences in wound size at 3rd and 4th week between BTXA and control ($p < 0.05$). Less inflammatory cells in BTXA group than control at 2nd week ($p < 0.05$). BTXA group showed less FBs and fibrosis than control at 4th week ($p < 0.05$). BTX-A group had strong collagen density than control at 8th week ($p < 0.05$). At 4th week, BTX-A group had lower TGF-β1 expression than control ($p < 0.05$).

with BTX-A at monthly intervals for 3 months resulted in remarkable improvement in symptoms of erythema, itching, pliability, and high patient satisfaction [44].

More recently, Hao *et al*., compared the viability of keloid fibroblast to normal fibroblasts by treating both with increasing dose of BTX-A. At lowered BTX-A concentrations, no significant changes were noted but when the dose was increased to 1 U/L, keloid fibroblast showed fragmentation, increased S phase, increased cell apoptosis and a significant decrease in the number of adherent cells [46]. Other several studies have also reported similar inhibitory effects of BTX-A on keloid or scar-derived fibroblasts. The number of viable fibroblasts was significantly decreased when the fibroblasts derived from both HTS and normal mature scar tissue was treated with BTX-A (0 or 4 units/ml) ($p < 0.001$), suggesting that BTX-A inhibited the proliferation of fibroblasts [37].

In another study, control fibroblasts from normal tissue showed very high proliferation on the 7th day, whereas fibroblasts from scar contracture tissue treated with BTX-A 2.5 U/10^6 cells showed nucleus pyknosis and significant cellular apoptosis ($p < 0.05$) [47]. BTX-A not only seems to arrest the proliferation of fibroblasts but also escalates the apoptotic activity, leading to an overall decline in the fibroblast population in the granulation tissue hence controlling the extent of fibrosis.

3.2. Downregulation of TGF-β1 Expression/SMAD Signaling Pathway

Dysregulation of cytokine TGF-β1 seems to be the central driving force in the pathogenesis of HTS and keloids. TGF-β1 is a multifunctional growth factor expressed by most cells in wound healing and play a prominent part in signaling cellular proliferation, differentiation, apoptosis and adhesion, stimulation and deposition of ECM, including collagen type I, collagen type III, and fibronectin [42] [48] [49]. Over-expressions of TGF-β1 and -β2 along with flawed SMAD activity, and decreased expression of TGF-β3 increase collagen production and ECM deposition leading to the pathogenesis of abnormal scars [50] [51] [52] [53] [54]. TGF-β1 and -β2 activates fibroblasts, while TGF-β3 is a receptor antagonist and leads to diminished fibroblast activity. In TGFβ1/SMAD signaling pathway, TGF-β1 binds to two different trans-membrane receptor serine/threonine kinases and activates the cytoplasmic SMAD proteins. Phosphorylation of receptor-activated SMAD2 and SMAD3 by TGF-β receptor type I results in the formation of a complex with Co-SMAD to produce SMAD4 [55]. Through this mechanism TGF-β1 activates collagen gene transcription and differentiates fibroblasts into myofibroblasts. Aberration in these processes culminates to abnormal scar pathogenesis.

In different studies, BTX-A has shown to decrease the levels of TGF-β1 induced phosphorylation of SMAD2 activity in fibroblasts derived from scar contractures [56] [57]. Since TGF-β1 is also secreted by fibroblasts, and BTX-A's ability to directly inhibit the growth and enhance apoptosis of fibroblasts further

decreases secretion of TGF-β1 from these cells and in turn reduces the final fibroblasts turnover. Zhibo *et al.* found that with BTX-A treatment inhibition of fibroblasts proliferation derived from HTS also coincided with down-regulation of TGF-β1 protein expression, highlighting the molecular mechanism of BTX-A in HTS [42]. In *in-vitro* studies, exogenous addition of neutralizing antibodies to TGF-β1 and TGF-β2 reduced neovascularization, fibronectin, collagen type III and type I deposition in the early wound healing [54] It also improved the structure of the neo-dermis resembling more closely to that of the normal dermis. Likewise, exogenous administration of TGF-β3 peptide had similar effects, whereas wounds treated with TGF-β1 or TGF-β2 had more extracellular matrix deposition [54]. TGF-β1 has been regarded as a dominant cytokine closely linked to the regulation of scar pathogenesis, and the action of BTX-A is effective and significant in the inhibition of HTS and keloid formation through the interference of TGF-β/SMAD signaling pathway.

3.3. Inhibition of Fibroblast to Myofibroblast Differentiation

In the late proliferative phase, via TGF-β1, fibroblasts differentiate to a specialized proto-myofibroblasts, which in response to the cellular changes and mechanical tension forms myofibroblasts. These contractile cells show specific attributes of both fibroblasts and smooth muscle cells. Induced by TGF-β1, myofibroblast along with stress fibers containing α-smooth muscle actin (α-SMA) enhances increased proliferation, migration, deposition of ECM, and further production of cytokines. It also facilitates wound contraction and thus plays a primary role in the maturation of granulation tissue [58] [59] [60] [61]. Myosin II has a role in the cell deformation, adhesion, and along with actin can regulate the cell contraction process [62] [63] [64]. The contractility of fibroblasts corresponds to the expression of α-SMA, and by lessening the expression of α-SMA and myosin II, the contraction of scars can be greatly reduced [65] [66]. TGF-β is a powerful mediator of myofibroblast differentiation that directly boots myofibroblast maturity by the inducement of α-SMA expression, and also function as a stimulant of EMT CTGF, which acts downstream of TGF-β to heighten TGF-β-mediated response [32] [54] [65] [67].

Several animal models and cell culture studies have exhibited that inhibition of TGF-β1 signaling at all levels of the pathway effectively prevents myofibroblast formation and development of fibrosis. These studies show that BTX-A inhibits TGF-β1, and by blocking this pathway BTX-A successfully inhibits the differentiation of myofibroblasts and their expression of α-SMA and myosin II proteins [37] [47]. In a study conducted by Jeong *et al*., BTX-A treated HTS fibroblasts showed a significant decrease in α-SMA transcription, α-SMA protein levels, and α-SMA staining [37]. Another similar study showed a significant decrease in the expression of α-SMA mRNA levels in fibroblasts derived from HTS when treated with BTX-A [47]. Since α-SMA is a marker for myofibroblast, the decreased level of α-SMA in all these studies clearly show the inhibitory effect of BTX-A on TGF-β1 signaling and subsequent prevention of myofibroblast diffe-

rentiation by downregulating TGF-β1 expression. These can partly explain the molecular mechanism and effect of BTX-A on fibroblasts thus curtailing abnormal scarring.

3.4. BTX-A on the Expression of Collagen, MMP-1, MMP-2, and MMP-9

Collagen is a long fibrous protein displaying outstanding endurance and is one of the main components of ECM. Fibroblasts in the proliferative phase synthesize collagen and together with elastin and soft keratin, they provide tensile strength and elasticity to tissues. With regard to normal fibroblasts, there is a 3-fold increase of collagen in HTS and a 20-fold increase in keloids, leading to a larger and abnormal scar [68]. Histologically, HTS has more of collagen type III in a wavy, regular pattern arranged parallel to the skin surface, whereas keloids have a haphazard pattern, or nodular, whorls of collagen type I and type III [69].

TGF-β1 has demonstrated to up-regulate the expression of different types of collagen in cultured fibroblasts, and excessive collagen is a key feature of HTS and keloid. In one recent study, keloid fibroblasts treated with BTX-A (0.5 unit/10^5 cells) significantly decreased collagen type III mRNA expression ($p < 0.05$) compared to no treatment control [70]. Collagen type III expression also decreased significantly in BTX-A of 0.5 unit/10^5 cells with TGF-β1 compared to TGF-β1 alone [70]. In another cell culture study, collagen type $I_{\alpha 1}$ was significantly decreased ($p < 0.05$) by BTX-A but collagen type III expression was not affected in the same study [57]. In other animal model studies, characteristics of collagen in HTS were compared with controls to assess the effects of BTX-A on hyper-proliferative scars [39] [71] [72] [73]. Scar thickness and deposition of collagen was examined histologically. HTS tissue administered with BTX-A showed a decrease in the number of collagen fibrils with more organized fibers, absent of granulomatous or inflammatory infiltrates. The control had thicker collagen fibers with a haphazard arrangement. The BTX-A treated group also showed diminished expression of TGF-β1 in one of the animal model studies [73].

MMPs are matrix metalloproteinases that are regulated by TGF-β1 and have an important role in the regulation of synthesis and breakdown of ECM. MMP-1 (collagenase) and MMP-2 (gelatinase-A) degrade ECM during tissue remodeling and reduction in the synthesis of these molecules may explain the lack of scar regression seen in abnormal scarring [74]. In one study, when keloid fibroblasts were treated with BTX-A, it resulted in a substantial increase in the expressions of MMP-1 and MMP-2 genes in comparison to normal fibroblasts, but the expressions of TGF-β1 gene and MMP-9 declined with increasing toxin dose [46]. Other studies showed a significant increase in MMP-1 mRNA expression and MMP-2 activity in keloid fibroblasts compared with the control group ($p < 0.05$) after treatment with BTX-A even without the addition of exogenous TGF-β [70]. In another experimental study, fibroblasts treated with BTX-A with or without

the combination of TGF-β1 significantly increased MMP-2 and matrix MMP-9 expression when compared to control group ($p < 0.05$), but MMP-2 decreased significantly after treatment with only TGF-β1 [57]. These studies suggest that BTX-A inhibits TGF-β1 and as a result increases the expression of MMP-1 and MMP-2 which then prevents excess collagen and ECM deposition, resulting in less fibrosis. BTX-A thus downregulates collagen expression and upregulates MMPs to reduce overtly synthesized ECM.

3.5. BTX-A Inhibits Connective Tissue Growth Factor (CTGF) Expression

Connective tissue growth factor (CTGF) is a pro-fibrotic and a downstream regulator of some of the responses of TGF-β1 functions. Induction of this complex molecule by TGF-β1 produce prolonged signaling of collagen mRNA expression, resulting in excessive collagen deposition, cellular adhesion, and growth leading to a state of sustained fibrosis [75] [76] [77] [78]. Therefore, blocking CTGF can prevent the synergistic effect of CTGF and TGF-β1 on hyper-proliferative scars. CTGF is excessively expressed in dermal fibrotic lesions like keloids, HTS, scleroderma, fibro-sarcomas, and as well as in lung, gingival, and liver fibrosis [79]-[84]. The elevated level of CTGF expression observed in fibrotic lesions is one of the best molecular markers of the fibrotic phenotype.

The effects of BTX-A on CTGF in hypertrophic scar still are largely unknown. However, a study exhibited that BTX-A adequately suppressed the growth of fibroblasts derived from HTS in 8 different patients, which in turn bring about a significant decrease in CTGF expression [40]. Compared to fibroblasts without BTX-A treatment, HTS derived fibroblasts when treated with BTX-A at 1.0 U/10^6 cells reduced CTGF expression by 49.2% ± 12.5% ($p < 0.01$), and with BTX-A at 2.5 U/10^6 cells reduced CTGF expression by 56.9% ($p < 0.01$) [40]. These results displayed that BTX-A had significant effects on the down-regulation of CTGF expression. So, the capacity of BTX-A to reduce CTGF expression and inhibit fibroblast proliferation may be associated with clinical improvement in the hypertrophic scar.

3.6. Relieves Wound Tension

Skin-tension is a known cause that acts on the wound borders during the healing process and prolongs the inflammatory reactions that aggravate fibrosis. The mechanical tension of skin closure is converted chemically into complex signaling that releases cytokines, particularly TGF-β1, that directly influence the quality of a scar [85] [86]. The mechanical stress detected by the mechanoreceptors in cells stimulates myofibroblast contraction that activates latent TGF-β1 in the ECM [87] [88]. Thus, this mechano-chemical stimulation accelerates the induction of cell proliferation, collagen synthesis and upregulation of expressions of TGF-β1, integrin-β1, and cytoskeleton p130Cas [87] [89] [90]. It is known that wound edges perpendicular to the relaxed skin tension lines (RSTL) pull the wound muscles and tissues with an opposing force that impedes the normal

wound healing process, and leads to sustained inflammation and fibrosis leading to the formation of HTS and keloids.

BTX-A, when injected locally, causes temporary paralysis of the muscles and tissues in the wound edges and reduces the dynamic muscle tension to enhance the healing process by blocking the mechanotransduction [88] [90] [91] [92]. In the year 2000, Gassner *et al*., used BTX-A on primates to show the effect it had on wound tension [93]. Gassner *et al*., in another study showed that BTX-A immobilized wound edges when injected locally in facial wounds resulting in a better cosmetic outcome compared to the controls [94]. Gassner conducted several studies and showed promising results of the effect of chemo-immobilization on wounds by injecting BTX-A locally [94] [95] [96]. Other studies have also effectively concluded that BTX-A injection can relax the wound edges in the anatomic locations prone to tensile stress by temporary paralysis of the muscles resulting in a far better outcome than in the controls [97] [98].

4. Conclusion

In this manuscript, the authors have studied responses of botulinum toxin type-A on wound healing and preventing pathological scars like HTS and keloids, and comprehended the overall effect BTX-A has on wound healing. The mechanisms of BTX-A on the prevention of HTS and keloids are still not understood very well but the staggering amount of results of *in vivo* and *in vitro* experiments has indeed identified a number of effects of BTX-A on non-neuronal cells in the skin. This multifunctional toxin seems to have a direct action on dermal fibroblasts, TGF-β1 expression and wound tension, and if effectively applied in the very early stages of wound healing can regress abnormal scarring and prevent HTS and keloids. However, there is much to be investigated regarding the mechanism of actions of BTX-A on wound healing and doors are just opening for further studies.

Conflicts of Interest

None declared.

References

[1] Ziade, M., *et al.* (2013) Use of Botulinum Toxin Type A to Improve Treatment of Facial Wounds: A Prospective Randomised Study. *Journal of Plastic, Reconstructive & Aesthetic Surgery*, **66**, 209-214. https://doi.org/10.1016/j.bjps.2012.09.012

[2] Zhang, D.Z., *et al.* (2016) Botulinum Toxin Type A and the Prevention of Hypertrophic Scars on the Maxillofacial Area and Neck: A Meta-Analysis of Randomized Controlled Trials. *PLoS ONE*, **11**, e0151627. https://doi.org/10.1371/journal.pone.0151627

[3] Leventhal, D., Furr, M. and Reiter, D. (2006) Treatment of Keloids and Hypertrophic Scars: A Meta-Analysis and Review of the Literature. *Archives of Facial Plastic Surgery*, **8**, 362-368. https://doi.org/10.1001/archfaci.8.6.362

[4] Kerner, J.A.C. (1820) Neue Beobachtungen über die in Württemberg so häufig vor-

fallenden tödlichen Vergiftungen durch den Genuss geräucherter Würste.

[5] Kerner, J.A.C. (1822) Das Fettgift, oder die Fettsa\0308ure und ihre Wirkungen auf den thierischen Organismus, etc.

[6] Hagenah, R., Benecke, R. and Wiegand, H. (1977) Effects of Type a Botulinum Toxin on the Cholinergic Transmission at Spinal Renshaw Cells and on the Inhibitory Action at Ia Inhibitory Interneurones. *Naunyn-Schmiedeberg's Archives of Pharmacology*, **299**, 267-272. https://doi.org/10.1007/BF00500319

[7] Nix, W.A. (2007) Botulinum Toxin in Chronic Myofascial Pain. *Schmerz*, **21**, 467-468. https://doi.org/10.1007/s00482-007-0567-y

[8] Ashkenazi, A. and Silberstein, S. (2008) Botulinum Toxin Type A for the Treatment of Headache: Why We Say Yes. *Archives of Neurology*, **65**, 146-149. https://doi.org/10.1001/archneurol.2007.20

[9] Wheeler, T. (2012) Sweat and Tears: Treating the Patient with Primary Hyperhidrosis. *British Journal of Nursing*, **21**, 408, 410-412. https://doi.org/10.12968/bjon.2012.21.7.408

[10] Santos-Silva, A., da Silva, C.M. and Cruz, F. (2013) Botulinum Toxin Treatment for Bladder Dysfunction. *International Journal of Urology*, **20**, 956-962. https://doi.org/10.1111/iju.12188

[11] Beer, K. and Waibel, J. (2007) Botulinum Toxin Type A Enhances the Outcome of Fractional Resurfacing of the Cheek. *Journal of Drugs in Dermatology*, **6**, 1151-1152.

[12] Lee, H., Saladi, R.N. and Fox, J.L. (2008) Cohort Study on Patient Response to Botulinum Toxin Cosmetic Therapy. *Journal of Cosmetic Dermatology*, **7**, 39-42. https://doi.org/10.1111/j.1473-2165.2008.00359.x

[13] Egan, M. (2014) Botox Maker Bought for $66 Billion in Biggest Deal of 2014. http://money.cnn.com/2014/11/17/investing/allergan-actavis-merger-boom

[14] Allergan (2015) Actavis plc Is Now Allergan plc.

[15] Felber, E.S. (2006) Botulinum Toxin in Primary Care Medicine. *The Journal of the American Osteopathic Association*, **106**, 609-614.

[16] Li, B., *et al*. (2010) Small Molecule Inhibitors as Countermeasures for Botulinum Neurotoxin Intoxication. *Molecules*, **16**, 202-220. https://doi.org/10.3390/molecules16010202

[17] Montecucco, C. and Molgo, J. (2005) Botulinal Neurotoxins: Revival of an Old Killer. *Current Opinion in Pharmacology*, **5**, 274-279. https://doi.org/10.1016/j.coph.2004.12.006

[18] Pirazzini, M., *et al*. (2017) Botulinum Neurotoxins: Biology, Pharmacology, and Toxicology. *Pharmacological Reviews*, **69**, 200-235. https://doi.org/10.1124/pr.116.012658

[19] Lacy, D.B., *et al*. (1998) Crystal Structure of Botulinum Neurotoxin Type A and Implications for Toxicity. *Nature Structural Biology*, **5**, 898-902. https://doi.org/10.1038/2338

[20] de Paiva, A., *et al*. (1999) Functional Repair of Motor Endplates after Botulinum Neurotoxin Type A Poisoning: Biphasic Switch of Synaptic Activity between Nerve Sprouts and Their Parent Terminals. *Proceedings of the National Academy of Sciences of the United States of America*, **96**, 3200-3205. https://doi.org/10.1073/pnas.96.6.3200

[21] Davies, J.R., *et al*. (2018) High Resolution Crystal Structures of *Clostridium botulinum* Neurotoxin A3 and A4 Binding Domains. *Journal of Structural Biology*, **202**, 113-117. https://doi.org/10.1016/j.jsb.2017.12.010

[22] Rossetto, O., Pirazzini, M. and Montecucco, C. (2014) Botulinum Neurotoxins: Genetic, Structural and Mechanistic Insights. *Nature Reviews Microbiology*, **12**, 535-349. https://doi.org/10.1038/nrmicro3295

[23] Montecucco, C. (1986) How Do Tetanus and Botulinum Toxins Bind to Neuronal Membranes? *Trends in Biochemical Sciences*, **11**, 314-317. https://doi.org/10.1016/0968-0004(86)90282-3

[24] Brunger, A.T. and Rummel, A. (2009) Receptor and Substrate Interactions of Clostridial Neurotoxins. *Toxicon*, **54**, 550-560. https://doi.org/10.1016/j.toxicon.2008.12.027

[25] Zhang, Y., *et al*. (2012) Simultaneous and Sensitive Detection of Six Serotypes of Botulinum Neurotoxin Using Enzyme-Linked Immunosorbent Assay-Based Protein Antibody Microarrays. *Analytical Biochemistry*, **430**, 185-192. https://doi.org/10.1016/j.ab.2012.08.021

[26] Pellett, S., *et al*. (2018) The Light Chain Defines the Duration of Action of Botulinum Toxin Serotype A Subtypes. *mBio*, **9**, pii: e00089-18. https://doi.org/10.1128/mBio.00089-18

[27] Pirazzini, M., *et al*. (2015) The Thioredoxin Reductase—Thioredoxin Redox System Cleaves the Interchain Disulphide Bond of Botulinum Neurotoxins on the Cytosolic Surface of Synaptic Vesicles. *Toxicon*, **107**, 32-36. https://doi.org/10.1016/j.toxicon.2015.06.019

[28] Arnon, S.S., *et al*. (2001) Botulinum Toxin as a Biological Weapon: Medical and Public Health Management. *JAMA*, **285**, 1059-1070. https://doi.org/10.1001/jama.285.8.1059

[29] Flanagan, M. (2013) Wound Healing and Skin Integrity: Principles and Practice. John Wiley and Sons Ltd., Hoboken.

[30] Gauglitz, G.G., *et al*. (2011) Hypertrophic Scarring and Keloids: Pathomechanisms and Current and Emerging Treatment Strategies. *Molecular Medicine*, **17**, 113-125. https://doi.org/10.2119/molmed.2009.00153

[31] Roberts, A.B. and Sporn, M.B. (1993) Physiological Actions and Clinical Applications of Transforming Growth Factor-Beta (TGF-Beta). *Growth Factors*, **8**, 1-9. https://doi.org/10.3109/08977199309029129

[32] Biernacka, A., Dobaczewski, M. and Frangogiannis, N.G. (2011) TGF-Beta Signaling in Fibrosis. *Growth Factors*, **29**, 196-202. https://doi.org/10.3109/08977194.2011.595714

[33] Mayrand, D., *et al*. (2012) Angiogenic Properties of Myofibroblasts Isolated from Normal Human Skin Wounds. *Angiogenesis*, **15**, 199-212. https://doi.org/10.1007/s10456-012-9253-5

[34] Pastar, I., *et al*. (2014) Epithelialization in Wound Healing: A Comprehensive Review. *Advances in Wound Care* (*New Rochelle*), **3**, 445-464. https://doi.org/10.1089/wound.2013.0473

[35] Su, W.H., *et al*. (2010) Nonsteroidal Anti-Inflammatory Drugs for Wounds: Pain Relief or Excessive Scar Formation? *Mediators of Inflammation*, **2010**, Article ID: 413238. https://doi.org/10.1155/2010/413238

[36] Butzelaar, L., *et al*. (2016) Currently Known Risk Factors for Hypertrophic Skin Scarring: A Review. *Journal of Plastic, Reconstructive & Aesthetic Surgery*, **69**, 163-169. https://doi.org/10.1016/j.bjps.2015.11.015

[37] Jeong, H.S., *et al*. (2015) Effect of Botulinum Toxin Type A on Differentiation of Fibroblasts Derived from Scar Tissue. *Plastic and Reconstructive Surgery*, **136**,

171e-178e. https://doi.org/10.1097/PRS.0000000000001438

[38] Oh, S.H., *et al*. (2012) The Potential Effect of Botulinum Toxin Type A on Human Dermal Fibroblasts: An *in Vitro* Study. *Dermatologic Surgery*, **38**, 1689-1694. https://doi.org/10.1111/j.1524-4725.2012.02504.x

[39] Xiao, Z. and Qu, G. (2012) Effects of Botulinum Toxin Type a on Collagen Deposition in Hypertrophic Scars. *Molecules*, **17**, 2169-2177. https://doi.org/10.3390/molecules17022169

[40] Xiao, Z., *et al*. (2011) Botulinum Toxin Type a Inhibits Connective Tissue Growth Factor Expression in Fibroblasts Derived from Hypertrophic Scar. *Aesthetic Plastic Surgery*, **35**, 802-807. https://doi.org/10.1007/s00266-011-9690-3

[41] Wang, L., Tai, N.Z. and Fan, Z.H. (2009) Effect of Botulinum Toxin Type A Injection on Hypertrophic Scar in Rabbit Ear Model. *Chinese Journal of Plastic Surgery*, **25**, 284-287.

[42] Xiao, Z., *et al*. (2010) Effect of Botulinum Toxin Type A on Transforming Growth Factor Beta1 in Fibroblasts Derived from Hypertrophic Scar: A Preliminary Report. *Aesthetic Plastic Surgery*, **34**, 424-427. https://doi.org/10.1007/s00266-009-9423-z

[43] Zhibo, X. and Miaobo, Z. (2009) Intralesional Botulinum Toxin Type A Injection as a New Treatment Measure for Keloids. *Plastic and Reconstructive Surgery*, **124**, 275e-277e. https://doi.org/10.1097/PRS.0b013e3181b98ee7

[44] Xiao, Z., Zhang, F. and Cui, Z. (2009) Treatment of Hypertrophic Scars with Intralesional Botulinum Toxin Type A Injections: A Preliminary Report. *Aesthetic Plastic Surgery*, **33**, 409-412. https://doi.org/10.1007/s00266-009-9334-z

[45] Zhibo, X. and Miaobo, Z. (2008) Botulinum Toxin Type A Affects Cell Cycle Distribution of Fibroblasts Derived from Hypertrophic Scar. *Journal of Plastic, Reconstructive & Aesthetic Surgery*, **61**, 1128-1129. https://doi.org/10.1016/j.bjps.2008.05.003

[46] Hao, R., *et al*. (2018) Efficacy and Possible Mechanisms of Botulinum Toxin Type A on Hypertrophic Scarring. *Journal of Cosmetic Dermatology*, **17**, 340-346. https://doi.org/10.1111/jocd.12534

[47] Chen, M., *et al*. (2016) Botulinum Toxin Type A Inhibits alpha-Smooth Muscle Actin and Myosin II Expression in Fibroblasts Derived From Scar Contracture. *Annals of Plastic Surgery*, **77**, e46-e49. https://doi.org/10.1097/SAP.0000000000000268

[48] Raghow, R., *et al*. (1987) Transforming Growth Factor-Beta Increases Steady State Levels of Type I Procollagen and Fibronectin Messenger RNAs Posttranscriptionally in Cultured Human Dermal Fibroblasts. *Journal of Clinical Investigation*, **79**, 1285-1288. https://doi.org/10.1172/JCI112950

[49] Chen, M.A. and Davidson, T.M. (2005) Scar Management: Prevention and Treatment Strategies. *Current Opinion in Otolaryngology & Head and Neck Surgery*, **13**, 242-247. https://doi.org/10.1097/01.moo.0000170525.74264.f8

[50] Fujiwara, M., Muragaki, Y. and Ooshima, A. (2005) Keloid-Derived Fibroblasts Show Increased Secretion of Factors Involved in Collagen Turnover and Depend on Matrix Metalloproteinase for Migration. *British Journal of Dermatology*, **153**, 295-300. https://doi.org/10.1111/j.1365-2133.2005.06698.x

[51] Tanriverdi-Akhisaroglu, S., Menderes, A. and Oktay, G. (2009) Matrix Metalloproteinase-2 and -9 Activities in Human Keloids, Hypertrophic and Atrophic Scars: A Pilot Study. *Cell Biochemistry and Function*, **27**, 81-87. https://doi.org/10.1002/cbf.1537

[52] Babalola, O., *et al*. (2013) The Role of microRNAs in Skin Fibrosis. *Archives of*

Dermatological Research, **305**, 763-776. https://doi.org/10.1007/s00403-013-1410-1

[53] Wolfram, D., *et al*. (2009) Hypertrophic Scars and Keloids—A Review of Their Pathophysiology, Risk Factors, and Therapeutic Management. *Dermatologic Surgery*, **35**, 171-181. https://doi.org/10.1111/j.1524-4725.2008.34406.x

[54] Shah, M., Foreman, D.M. and Ferguson, M.W. (1995) Neutralisation of TGF-beta 1 and TGF-beta 2 or Exogenous Addition of TGF-beta 3 to Cutaneous Rat Wounds Reduces Scarring. *Journal of Cell Science*, **108**, 985-1002.

[55] Shi, Y. and Massague, J. (2003) Mechanisms of TGF-beta Signaling from Cell Membrane to the Nucleus. *Cell*, **113**, 685-700. https://doi.org/10.1016/S0092-8674(03)00432-X

[56] Lee, S.D., *et al*. (2016) The Effect of Botulinum Neurotoxin Type A on Capsule Formation around Silicone Implants: The *in Vivo* and *in Vitro* Study. *International Wound Journal*, **13**, 65-71. https://doi.org/10.1111/iwj.12228

[57] Kim, S., *et al*. (2016) Effect of Botulinum Toxin Type A on TGF-beta/Smad Pathway Signaling: Implications for Silicone-Induced Capsule Formation. *Plastic and Reconstructive Surgery*, **138**, 821e-829e. https://doi.org/10.1097/PRS.0000000000002625

[58] Gabbiani, G., Ryan, G.B. and Majne, G. (1971) Presence of Modified Fibroblasts in Granulation Tissue and Their Possible Role in Wound Contraction. *Experientia*, **27**, 549-550. https://doi.org/10.1007/BF02147594

[59] Gabbiani, G. (2003) The Myofibroblast in Wound Healing and Fibrocontractive Diseases. *The Journal of Pathology*, **200**, 500-503. https://doi.org/10.1002/path.1427

[60] Hinz, B., *et al*. (2007) The Myofibroblast: One Function, Multiple Origins. *The American Journal of Pathology*, **170**, 1807-1816. https://doi.org/10.2353/ajpath.2007.070112

[61] Darby, I.A. and Hewitson, T.D. (2007) Fibroblast Differentiation in Wound Healing and Fibrosis. *International Review of Cytology*, **257**, 143-179. https://doi.org/10.1016/S0074-7696(07)57004-X

[62] Tanaka, J., *et al*. (1993) Morphological and Biochemical Analyses of Contractile Proteins (Actin, Myosin, Caldesmon and Tropomyosin) in Normal and Transformed Cells. *Journal of Cell Science*, **104**, 595-606.

[63] Bond, J.E., *et al*. (2011) Temporal Spatial Expression and Function of Non-Muscle Myosin II Isoforms IIA and IIB in Scar Remodeling. *Laboratory Investigation*, **91**, 499-508. https://doi.org/10.1038/labinvest.2010.181

[64] Vicente-Manzanares, M., *et al*. (2009) Non-Muscle Myosin II Takes Centre Stage in Cell Adhesion and Migration. *Nature Reviews Molecular Cell Biology*, **10**, 778-790. https://doi.org/10.1038/nrm2786

[65] Tomasek, J.J., *et al*. (2002) Myofibroblasts and Mechano-Regulation of Connective Tissue Remodelling. *Nature Reviews Molecular Cell Biology*, **3**, 349-363. https://doi.org/10.1038/nrm809

[66] Zaleskas, J.M., *et al*. (2001) Growth Factor Regulation of Smooth Muscle Actin Expression and Contraction of Human Articular Chondrocytes and Meniscal Cells in a Collagen-GAG Matrix. *Experimental Cell Research*, **270**, 21-31. https://doi.org/10.1006/excr.2001.5325

[67] Arno, A.I., *et al*. (2014) New Molecular Medicine-Based Scar Management Strategies. *Burns*, **40**, 539-551. https://doi.org/10.1016/j.burns.2013.11.010

[68] Berman, B., Maderal, A. and Raphael, B. (2017) Keloids and Hypertrophic Scars: Pathophysiology, Classification, and Treatment. *Dermatologic Surgery*, **43**, S3-S18. https://doi.org/10.1097/DSS.0000000000000819

[69] Jumper, N., Paus, R. and Bayat, A. (2015) Functional Histopathology of Keloid Disease. *Histology and Histopathology*, **30**, 1033-1057.

[70] Roh, T.S., *et al*. (2013) The Effects of Botulinum Toxin A on Collagen Synthesis, Expression of MMP (Matrix Metalloproteinases)-1,2,9 and TIMP (Tissue Inhibitors of Metalloproteinase)-1 in the Keloid Fibroblasts. *Archives of Aesthetic Plastic Surgery*, **19**, 114-119. https://doi.org/10.14730/aaps.2013.19.2.114

[71] Fanous, A., *et al*. (2019) Treatment of Keloid Scars with Botulinum Toxin Type A versus Triamcinolone in an Athymic Nude Mouse Model. *Plastic and Reconstructive Surgery*, **143**, 760-767. https://doi.org/10.1097/PRS.0000000000005323

[72] Liu, D.Q., Li, X.J. and Weng, X.J. (2017) Effect of BTXA on Inhibiting Hypertrophic Scar Formation in a Rabbit Ear Model. *Aesthetic Plastic Surgery*, **41**, 721-728. https://doi.org/10.1007/s00266-017-0803-5

[73] Lee, B.J., *et al*. (2009) Effect of Botulinum Toxin Type a on a Rat Surgical Wound Model. *Clinical and Experimental Otorhinolaryngology*, **2**, 20-27. https://doi.org/10.3342/ceo.2009.2.1.20

[74] Niessen, F.B., *et al*. (1999) On the Nature of Hypertrophic Scars and Keloids: A Review. *Plastic and Reconstructive Surgery*, **104**, 1435-1458. https://doi.org/10.1097/00006534-199910000-00031

[75] Grotendorst, G.R. (1997) Connective Tissue Growth Factor: A Mediator of TGF-β Action on Fibroblasts. *Cytokine & Growth Factor Reviews*, **8**, 171-179. https://doi.org/10.1016/S1359-6101(97)00010-5

[76] Duncan, M.R., *et al*. (1999) Connective Tissue Growth Factor Mediates Transforming Growth Factor Beta-Induced Collagen Synthesis: Down-Regulation by cAMP. *The FASEB Journal*, **13**, 1774-1786. https://doi.org/10.1096/fasebj.13.13.1774

[77] Weston, B.S., Wahab, N.A. and Mason, R.M. (2003) CTGF Mediates TGF-Beta-Induced Fibronectin Matrix Deposition by Upregulating Active Alpha5beta1 Integrin in Human Mesangial Cells. *Journal of the American Society of Nephrology*, **14**, 601-610. https://doi.org/10.1097/01.ASN.0000051600.53134.B9

[78] Abreu, J.G., *et al*. (2002) Connective-Tissue Growth Factor (CTGF) Modulates Cell Signalling by BMP and TGF-Beta. *Nature Cell Biology*, **4**, 599-604. https://doi.org/10.1038/ncb826

[79] Igarashi, A., *et al*. (1996) Connective Tissue Growth Factor Gene Expression in Tissue Sections from Localized Scleroderma, Keloid, and Other Fibrotic Skin Disorders. *Journal of Investigative Dermatology*, **106**, 729-733. https://doi.org/10.1111/1523-1747.ep12345771

[80] Lasky, J.A., *et al*. (1998) Connective Tissue Growth Factor mRNA Expression Is Upregulated in Bleomycin-Induced Lung Fibrosis. *American Journal of Physiology*, **275**, L365-L371. https://doi.org/10.1152/ajplung.1998.275.2.L365

[81] Paradis, V., *et al*. (1999) Expression of Connective Tissue Growth Factor in Experimental Rat and Human Liver Fibrosis. *Hepatology*, **30**, 968-976. https://doi.org/10.1002/hep.510300425

[82] Chen, S.J., *et al*. (1999) Stimulation of Type I Collagen Transcription in Human Skin Fibroblasts by TGF-Beta: Involvement of Smad 3. *Journal of Investigative Dermatology*, **112**, 49-57. https://doi.org/10.1046/j.1523-1747.1999.00477.x

[83] Uzel, M.I., *et al*. (2001) Connective Tissue Growth Factor in Drug-Induced Gingival Overgrowth. *Journal of Periodontology*, **72**, 921-931. https://doi.org/10.1902/jop.2001.72.7.921

[84] Leask, A., Holmes, A. and Abraham, D.J. (2002) Connective Tissue Growth Factor:

A New and Important Player in the Pathogenesis of Fibrosis. *Current Rheumatology Reports*, **4**, 136-142. https://doi.org/10.1007/s11926-002-0009-x

[85] Mustoe, T.A., *et al*. (2002) International Clinical Recommendations on Scar Management. *Plastic and Reconstructive Surgery*, **110**, 560-571. https://doi.org/10.1097/00006534-200208000-00031

[86] Derderian, C.A., *et al*. (2005) Mechanical Strain Alters Gene Expression in an *in Vitro* Model of Hypertrophic Scarring. *Annals of Plastic Surgery*, **55**, 69-75. https://doi.org/10.1097/01.sap.0000168160.86221.e9

[87] Ogawa, R. (2008) Keloid and Hypertrophic Scarring May Result from a Mechanoreceptor or Mechanosensitive Nociceptor Disorder. *Medical Hypotheses*, **71**, 493-500. https://doi.org/10.1016/j.mehy.2008.05.020

[88] Wipff, P.J., *et al*. (2007) Myofibroblast Contraction Activates Latent TGF-beta1 from the Extracellular Matrix. *Journal of Cell Biology*, **179**, 1311-1323. https://doi.org/10.1083/jcb.200704042

[89] Kuang, R., *et al*. (2015) Influence of Mechanical Stimulation on Human Dermal Fibroblasts Derived from Different Body Sites. *International Journal of Clinical and Experimental Medicine*, **8**, 7641-7647.

[90] Shu, M.G., Han, Y., Yang, L., Zhang, L.X., Xia, W.S., Liu, D. and Guo, S.Z. (2008) The Effect of Mechanical Stress on the Expression of Growth Factor in Human Skin Fibroblasts. *Chinese Journal of Aesthetic Medicine*, **17**, 689-691.

[91] Carruthers, A. and Carruthers, J. (2001) Botulinum Toxin Type A: History and Current Cosmetic Use in the Upper Face. *Seminars in Cutaneous Medicine and Surgery*, **20**, 71-84. https://doi.org/10.1053/sder.2001.25138

[92] Silberstein, E., *et al*. (2018) Effect of Botulinum Toxin A on Muscle Healing and Its Implications in Aesthetic and Reconstructive Surgery. *Aesthetic Surgery Journal*, **38**, 557-561. https://doi.org/10.1093/asj/sjx207

[93] Gassner, H.G., Sherris, D.A. and Otley, C.C. (2000) Treatment of Facial Wounds with Botulinum Toxin A Improves Cosmetic Outcome in Primates. *Plastic and Reconstructive Surgery*, **105**, 1948-1953. https://doi.org/10.1097/00006534-200005000-00005

[94] Gassner, H.G. and Sherris, D.A. (2003) Chemoimmobilization: Improving Predictability in the Treatment of Facial Scars. *Plastic and Reconstructive Surgery*, **112**, 1464-1466. https://doi.org/10.1097/01.PRS.0000081073.94689.DB

[95] Gassner, H.G., *et al*. (2006) Botulinum Toxin to Improve Facial Wound Healing: A Prospective, Blinded, Placebo-Controlled Study. *Mayo Clinic Proceedings*, **81**, 1023-1028. https://doi.org/10.4065/81.8.1023

[96] Gassner, H.G., Sherris, D.A. and Friedman, O. (2009) Botulinum Toxin-Induced Immobilization of Lower Facial Wounds. *Archives of Facial Plastic Surgery*, **11**, 140-142. https://doi.org/10.1001/archfacial.2009.3

[97] Tollefson, T.T., *et al*. (2006) Botulinum Toxin to Improve Results in Cleft Lip Repair. *Archives of Facial Plastic Surgery*, **8**, 221-222. https://doi.org/10.1001/archfaci.8.3.221

[98] Choi, J.C., Lucarelli, M.J. and Shore, J.W. (1997) Use of Botulinum A Toxin in Patients at Risk of Wound Complications Following Eyelid Reconstruction. *Ophthalmic Plastic and Reconstructive Surgery*, **13**, 259-264. https://doi.org/10.1097/00002341-199712000-00006

Abbreviations

BTX-A: Botulinum Toxin Type-A; NMJ: Neuromuscular Junction; ECM: Extracellular Matrix; HTS: Hypertrophic Scar; TGF β-1: Transforming Growth Factor β-1; MMP: Matrix Metalloproteinase; CTGF: Connective Tissue Growth Factor; EMT: Epithelial-Mesenchymal Transition

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The Combination of Moberg Flap with V-Y Advancement and Reverse Adipofascial Cross Finger Flap for Coverage of Degloving Injury of the Thumb-Case Report

Medhat Emil*, Dalia Habib, Omar Al Ameri, Dalal Al Mansoori

Department of Plastic and Reconstructive Surgery, Zayed Military Hospital, Abu Dhabi, UAE
Email: *medhatemil1@hotmail.com

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Abstract

We report a case of 22 years old male patient who is a worker in a factory and sustained degloving injury of his left thumb in a machine while working. There was loss of the pulp of the thumb extending circumferentially to the dorsal aspect with loss of the skin of the terminal phalanx and part of the proximal phalanx. The nail and germinal matrix were lost with exposure of the bone and extensor pollicis longus tendon insertion. The thumb was totally covered with a combination of two flaps: Moberg flap with V-Y advancement was used to cover most of the volar surface of the thumb and reverse adipofascial cross finger flap from the adjacent index finger was used to cover the dorsal surface and the tip of the thumb. The reverse adipofascial cross finger flap was covered with split thickness skin graft. Three weeks later this flap was divided and the thumb was mobilized freely. The patient had a full range of movement of the thumb and index finger with few settings of physiotherapy postoperatively. We recommend combining both of these flaps to reconstruct degloving injury of the thumb as they provide near adjacent tissue of similar texture, preserve sensation at the volar aspect of the thumb and also avoid the complications of the distant flaps.

Keywords

Moberg Flap, Reverse Adipofascial Cross Finger Flap, Degloving Injury Thumb

1. Introduction

The thumb is responsible for about 40% of the function of the hand. Preserving

every millimeter of it can make a difference in the patient's daily activities. Many techniques are described to reconstruct degloving injuries of the thumb. This article will discuss the procedure we used to reconstruct a degloving injury of the thumb using a combination of two flaps; one from the volar aspect of the thumb itself and another one from the adjacent index finger to preserve its length with durable flaps and preserve sensation at its volar aspect.

2. Case Report

A twenty two years old worker in a factory presented to the Emergency Department with circumferential degloving injury of his left thumb while working on a machine. There was loss of skin, subcutaneous fat, the whole nail and the nailbed and the tip of the head of the bone of the terminal phalanx (Figure 1). The bone of the terminal phalanx was totally exposed with exposure of the extensor tendon on the interphalangeal joint dorsally. The interphalangeal joint was covered with skin volarly with no exposure of the flexor pollicis longus tendon insertion. There was no skin at the dorsal aspect of the interphalangeal joint and the distal part of the proximal phalanx dorsally. The patient did not bring the degloved part with him as it was crushed in the machine.

3. Operative Procedure

The patient was taken to the operation theatre on the same day and under the effect of general anaesthesia and with the use of arm tourniquet, wound debridement was done. The volar skin was advanced distally using Moberg flap to cover most of the terminal phalanx (Figure 2). The flap is an axial one with both the digital arteries on the volar surface of the thumb included in it. To

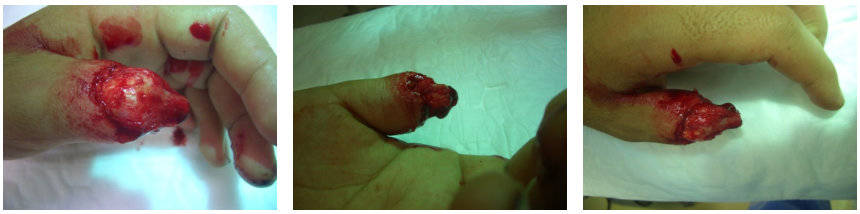

Figure 1. Circumferential degloving injury left thumb with loss of skin, subcutaneous fat, the whole nail and the nailbed and the tip of the head of the bone of the terminal phalanx. There is exposure of the bone of the terminal phalanx and the extensor tendon on the interphalangeal joint dorsally.

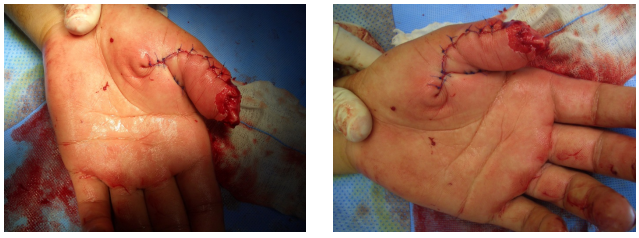

Figure 2. Moberg flap with V-Y advancement incorporation used to cover most of the terminal phalanx volarly.

prevent flexion of the thumb at the interphalangeal joint, a "V" incision was made at the base of the thumb and was sutured in a "Y" shape fashion after advancing the flap distally as much as possible. Suturing done using 4/0 Prolene sutures. The flap could not reach the tip of the amputation stump and this part along with the dorsum of the thumb was covered with reverse adipofascial flap from the adjacent index finger.

The reverse adipofascial flap was designed on the dorsal aspect of the proximal phalanx of the left index finger. The skin (epidermis and dermis) was raised, like a page of a book with the base being on the contralateral side of the recipient digit (**Figure 3**). The adipofascial flap formed of all of the tissue between the dermis and paratenon, including the dorsal veins, fat and fascia. The flap was raised like a page of a book, with the base on the ipsilateral side of the thumb. Dissection of the flap was done with care to keep the paratenon of the extensor tendon intact. The adipofascial flap was inset to the defect and sutured to the radial edge and distal part of the Moberg flap using 4/0 Vicryl sutures. Split thickness skin graft was harvested from the medial aspect of the arm and applied on the flap after making few holes in the graft (**Figure 4**). The previously elevated skin of the proximal phalanx of the index finger was repositioned back to its original place to cover the paratenon of the extensor tendon and sutured to the surrounding skin proximally and distally with 5/0 Prolene sutures. It was sutured to the adjacent skin graft, but not to the underlying adipofascial flap, using 4/0 Vicryl sutures. A small tube drain was kept under the skin of the proximal phalanx of the index finger to prevent haematoma formation. Two 2/0 Prolene sutures were used to keep the proximal part of the thumb and the index finger close together to avoid unexpected abduction of the thumb with resultant flap disruption during subsequent change of the dressings. Volar slab was applied in the functional position. The small tube drain was removed 2 days postoperatively. There was no haematoma formation and there was no infection. Both the flaps were viable and the graft take was 100%. The reverse adipofascial flap was divided 3 weeks postoperatively and the patient started physiotherapy from the 2nd day of the division (**Figure 5**). Physiotherapy and follow up continued for about 2 months and there was no stiffness of the thumb or the index finger.

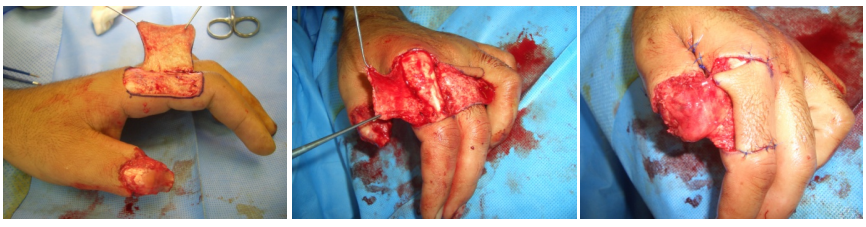

Figure 3. The reverse adipofascial flap: The skin (epidermis and dermis) was raised, like a page of a book with the base being on the contralateral side of the recipient digit. The adipofascial flap formed of all of the tissue between the dermis and paratenon raised like a page of a book, with the base on the ipsilateral side of the thumb. The paratenon of the extensor tendon is kept intact. The adipofascial flap was inset to the defect.

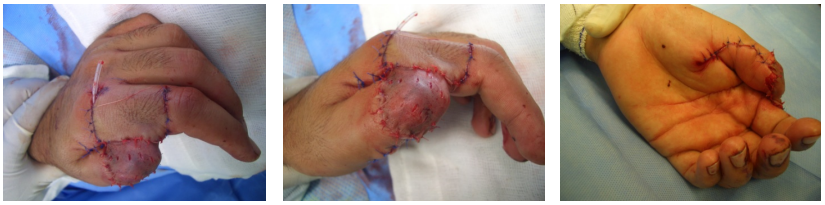

Figure 4. The adipofascial flap sutured to the radial edge and distal part of the Moberg flap and covered with split thickness skin graft. The previously elevated skin of the proximal phalanx of the index finger was repositioned back to its original place.

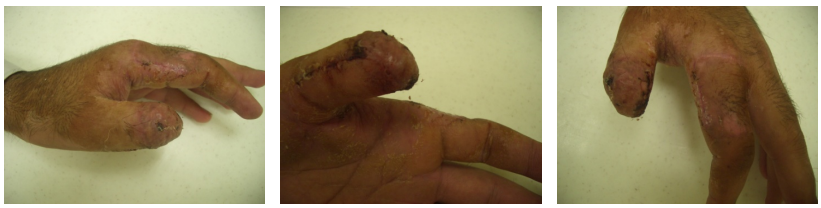

Figure 5. Three weeks later the reverse adipofascial flap was divided with good abduction of the thumb.

4. Discussion

The Moberg flap was first described in 1964 to cover defects at the pulp of the thumb by advancing the volar skin distally. It was used also with success in other fingers [1].

The Moberg flap involves making two bilateral incisions dorsal to the neurovascular bundles. The flap is raised over the paratenon including both the neurovascular bundles and advanced distally to cover the raw area at the tip of the thumb with the interphalangeal joint kept in flexion position. This can result in inability to extend the interphalangeal joint of the thumb fully post operatively [2]. To avoid this, different techniques were prescribed to provide more advancement of the flap without the need to keep the interphalangeal joint in flexion position. One of these techniques was prescribed by Jindal *et al.* in the form of "Z" plasty modification at the base of the flap [3]. Another technique to avoid flexion of the interphalangeal joint is to incorporate "V" at the base of the flap and the proximal defect is closed in a "V-Y" fashion [4]. In our case, we used the "V-Y" advancement technique to move the flap distally without flexing the interphalangeal joint of the thumb.

In a study done by Baumeister *et al.* on 36 patients, eighty three percent of the cases had the defects covered with the flap without additional iatrogenic shortening of the thumb [5]. In our case, the flap was advanced distally as much as possible but could not cover the whole distal phalanx volarly till the tip of the thumb. The bone was not shortened, as each millimeter of the thumb counts, but its tip was covered with the distal part of a reverse cross finger adipofascial flap from the dorsal aspect of the adjacent index finger. This flap was used also to cover the rest of the raw area at the dorsal aspect of the thumb.

Anatomic studies showed that this flap is based on constant dorsal branches of the palmar digital arteries in the proximal phalanx, which anastomose with the

vascular system of the dorsal skin. The flap is drained by small vena concomitants that follow the arterial branches [6].

We elevated the flap based on this understanding of the blood supply after dissecting it from the overlying skin and the underlying paratenon of the extensor tendon of the index finger. The flap was turned over on its attached base at the radial side of the index finger to cover the raw area of the thumb and the skin over the donor site was repositioned over the paratenon. Split thickness skin graft was applied on the flap on the thumb only. We preferred this technique over other techniques as de-epithelialization of the skin of the donor finger and including it with the flap as one mass with turning over to cover the raw area [7] [8]. We feel that such procedures can predispose to implantation dermoid cyst formation if no adequate de-epithelialization of the flap done. Also applying skin graft on both the donor and recipient digits may be unnecessary if only skin graft can be applied on the recipient digit in the procedure we used avoiding disfigurement of the donor digit and maintaining its normal hairy skin shape.

Adipofascial flaps can also be used as turnover flaps from the same digit to cover defects of the digit distally. This homodigital flap can be used with success to cover the dorsal aspects of the middle [9] and terminal [10] phalanges of the fingers and also to cover raw areas at the dorsal aspect of the thumb [11]. It is a one stage procedure and does not involve other fingers than the one injured and when compared with the de-epithelialized cross finger flap it showed better results [12]. In our case we could not use this flap as the injury was circumferential and not involving the dorsal aspect only. Combining such a turnover flap with the Moberg flap can jeopardize the venous return of the thumb as the flaps are raised from both the volar and dorsal aspects of the thumb. Providing another flap from an adjacent finger can eliminate this risk.

5. Conclusions

The Moberg flap preserves the touch sensation at the volar aspect of the thumb replacing its pulp with the same type of tissue. Incorporation of V-Y advancement fashion at its base avoids flexion deformity of the thumb. The reverse adipofascial cross finger flap is a simple and rapid procedure. It is an excellent option because of its thinness, good pliability and minimal donor site deformity.

Combining both flaps together can avoid the need for distant flaps leaving the shoulder and hand entirely free and avoiding bulky disfigured insensate flaps.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

Ethical Approval

The procedures performed in this study involving human participant were in

accordance with the international ethical standards.

Informed Consent

Additional informed consent was obtained from the participant for whom identifying information is included in this article.

References

[1] Macht, S.D. and Watson, H.K. (1980) The Moberg Volar Advancement Flap for Digital Reconstruction. *Journal of Hand Surgery*, **5**, 372-376. https://doi.org/10.1016/S0363-5023(80)80179-1

[2] Ricardo, M. and Eilen, M. (2016) Moberg Advancement Flap in the Thumb. Orthopedics and Rheumatology Open Access Journal, **3**, 555625. https://doi.org/10.19080/OROAJ.2016.03.555625

[3] Jindal, R., Schultz, B.E., Ruane, E.J. and Spiess, A.M. (2016) Cadaveric Study of a Z-Plasty Modification to the Moberg Flap for Increased Advancement and Decreased Morbidity. *Plastic and Reconstructive Surgery*, **137**, 897-904. https://doi.org/10.1097/01.prs.0000479988.80490.b8

[4] Page, F., Langley, C. and Lamyman, M. (2016) Management of Fingertip Injuries. The British Editorial Society of Bone & Joint Surgery. Oxford University Hospitals NHS Foundation Trust Headley Way, Oxford.

[5] Baumeister, S., Menke, H., Wittemann, M. and Germann, G. (2002) Functional Outcome after the Moberg Advancement Flap in the Thumb. *The Journal of Hand Surgery*, **27**, 105-114. https://doi.org/10.1053/jhsu.2002.30921

[6] Braga-Silva, J. (2005) Anatomic Basis of Dorsal Finger Skin Cover. *Techniques in Hand and Upper Extremity Surgery*, **9**, 000-000. https://doi.org/10.1097/01.bth.0000173726.99670.f0

[7] Cappel, D.A. and Burech, J. (1985) The Cross-Finger Flap: An Established Reconstructive Procedure. *Hand Clinics*, **1**, 677-683.

[8] Gürbüz, K., Uzun, E. and Ozan, F. (2016) Covering the Dorsal Finger Defect with Reverse Cross Finger Flap. Dorsal Defect and Cross Finger Flap. *Journal of Clinical and Analytical Medicine*, **7**, 262-264. https://doi.org/10.4328/JCAM.4133

[9] Naji, S.H. (2011) Dorsal Adipofascial Turnover Flap for Dorsal Defects of Finger Injury. *Medical Journal of Babylon*, **8**, 67-72.

[10] Ozdemir, R., Kilinç, H., Sensöz, O., Unlü, R.E. and Baran, C.N. (2001) Innervated Dorsal Adipofascial Turnover Flap for Fingertip Amputations. *Annals of Plastic Surgery*, **46**, 9-14. https://doi.org/10.1097/00000637-200101000-00003

[11] Al-Qattan, M.M. (2001) The Adipofascial Turnover Flap for Coverage of the Exposed Distal Interphalangeal Joint of the Fingers and Interphalangeal Joint of the Thumb. *Journal of Hand Surgery*, **26**, 1116-1119. https://doi.org/10.1053/jhsu.2001.28758

[12] Al-Qattan, M.M. (2005) De-Epithelialized Cross-Finger Flaps versus Adipofascial Turnover Flaps for the Reconstruction of Small Complex Dorsal Digital Defects: A Comparative Analysis. *The Journal of Hand Surgery*, **30**, 549-557. https://doi.org/10.1016/j.jhsa.2005.02.004

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Reliability of the Estimation of the Take of Split Thickness Graft by the Observation Method

Kwesi O. Nsaful[1*], A. B. Paintsil[2], J. C. B. Dakubo[3], J. Nsaful[3], K. Appiah-Labi[4], E. Nartey[5]

[1]Plastic, Reconstructive Surgery and Burns Unit, 37 Military Hospital, Accra, Ghana
[2]National Reconstructive Plastic Surgery and Burns Unit, Korle Bu Teaching Hospital, Accra, Ghana
[3]The Surgical Department, Korle Bu Teaching Hospital, Accra, Ghana
[4]Department of Microbiology, Korle Bu Teaching Hospital, Accra, Ghana
[5]Data and Statistical Analysis Department, Korle Bu Teaching Hospital, Accra, Ghana
Email: *knsaful@yahoo.co.uk

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Abstract

Introduction: Split thickness skin grafts are frequently employed to provide biological cover for extensive wounds. The take of the skin graft is traditionally estimated by observation and recorded as a percentage. The intent of this study was to ascertain the reliability of the observation method in comparison with the Image J digital programme. **Materials and Methods:** The study was a longitudinal study conducted on the wards of the National Reconstructive Plastic Surgery and Burns Centre (NRPSBC) at the Korle Bu Teaching Hospital (KBTH) on patients who were admitted during the period of the study with wounds who received split skin grafts. Image J®, an image analysis program, was employed in the calculation of the take of the grafts. These were compared to values obtained by estimation by observation. **Results:** There was no statistically significant difference between the estimation of graft take, made by observation and using Image J® digital programme. **Conclusion:** The estimation of graft take by observation is an acceptable practice.

Keywords

Split Skin Graft, Take of Graft, Estimation by Observation, Image J

1. Introduction

Skin grafts are commonly used to close skin defects and have been used since the early 1500s [1]. The practice originated among the tile maker caste in India approximately 3000 years ago [2].

A Split-Thickness Skin Graft (STSG) is indicated in most wounds that cannot be closed primarily or when closure by secondary intention is contraindicated. It

is also indicated for a relatively large wound (>5 cm in diameter) that would take many weeks to heal secondarily [3]. Skin grafts are employed in a variety of conditions, such as traumatic wounds, defects after tumour resection, burn reconstruction, scar contracture release, congenital skin deficiencies, hair restoration, vitiligo, and nipple-areola reconstruction [2] [4] [5].

The take of the skin graft is traditionally estimated by observation and recorded as a percentage—a take of 100% occurring when all the recipient wound bed is covered by the skin graft. However, very little work is available to ascertain the reliability of the estimation by observation method. This work aims at ascertaining the reliability of the observation method.

Image J is a Java-based program developed at the National Institutes of Health and the Laboratory for Optical and Computational Instrumentation (LOCI, University of Wisconsin) [6]. Image J known in previous incarnations as NIH Image, is a scientific image analysis program [7] [8] [9].

Image J can display, edit, analyze, process, save, and print 8-bit color and grayscale, 16-bit integer, and 32-bit floating point images [10]. Image J can be used to calculate area and pixel value statistics of user-defined selections and intensity-threshold objects. It can measure distances and angles. It can create density histograms and line profile plots [10].

The Image J method was employed in the calculation of the take of the grafts. These were compared to values obtained by estimation by observation.

2. Methodology

2.1. Study Design

The study was a longitudinal analytical study. The study was conducted on the wards of the NRPSBC at the KBTH.

The study was conducted on patients with burns and other ulcers, which required split-thickness grafting, brought to the NRPSBC at the KBTH for management. Patients with burn wounds and acute ulcers admitted to the NRPSBC at the KBTH during the period of the study who required split skin grafting as part of their treatment were included in the study. Excluded from the study were;

1) Patients with chronic ulcers—A chronic ulcer is a wound that shows no tendency to heal after three months of appropriate treatment or is still not fully healed at 12 months [11].

2) Weight bearing plantar ulcers.

3) Patients with previously failed skin grafts.

The period of the study spanned May 2016 to Jan 2017.

The sample size for the study was calculated comparing two proportions *i.e.* the proportion of graft failure due to infection and proportion of graft failure in general.

A total minimum sample of 65 was obtained. Accounting for contingencies such as loss to follow-up and incomplete data, the sample size was increased by

10% (minimum sample of 72).

The sample estimate for the study was therefore 72 patients.

Data collected included:

- Patient demographics,
- Ulcer aetiology,
- The Percentage graft take by observation,
- The Percentage graft take using the Image J.

Graft failure was defined as loss of split skin graft that will require re-grafting of the wound bed.

2.2. Procedure

Skin grafts were performed by standard operating techniques. All operations were performed under either general or regional anaesthesia with prophylactic antibiotics (Intravenous Cefuroxime-Child 1 month - 18 years: 50 mg/kg, Adult: 1.5 gm). Split thickness skin grafts were harvested using a Graft knife or a Dermatome. To ensure as much as possible that the graft thickness was similar, in all cases:

1) During the use of Graft knife, the distance between the roller and the blade was kept constant with the wheel locked between calibrations 1 and 2.

2) During the use of the Dermatome, it was set at calibration 0.10 inches.

The grafts, when required, were meshed prior to application. The grafts were secured to the wound beds with sutures or staples. In addition, immobilization techniques including the use of bolster dressings and Plaster of Paris were used when skin grafts were applied onto mobile surfaces. This was done to prevent movement of the graft on the bed, which will interrupt revascularization.

All patients for this study were placed on routinely used intravenous antibiotics Cefuroxime (Child 1 month to 18 years: 20 mg/kg every 8 hours, Adult: 750 mg every 8 hours). A dose was given intraoperatively and regular doses given postoperatively for 5 days and then on oral Cefuroxime (Child 3 months-12 years: 30 mg/kg/day suspension PO in 2 divided doses, or Adult: 500 mg 12 hourly) from POD 6 to POD 14.

The "take" of the graft was estimated by observation by any of three Senior Residents assigned to this study. These Senior Residents were blinded all through this study to the Image J results. This was done at the change of dressing of the recipient site *i.e.* Post-operative days 5, 10 and 14. The graft "take" on these days was recorded as a percentage.

2.3. The Use of Image J® in Graft Take Measurements

To measure the area of the wound covered by the graft as well as the raw area(s) (*i.e.* the area not covered by the graft) a known measure on the patient is taken and used to set the scale for the measurement. In Patient 04 on POD 5, for example, the known measure taken was 3 cm (**Figure 1**).

This was used to set the scale as in **Figure 2**.

Wound bed is outlined. The Surface area of the marked out area is calculated using the set scale (Figure 3).

Image J® was used to measure the areas (Figure 4). Serial 6 (62.007 cm^2) is the total surface area of the wound bed. Serial 1 - 5 are the areas of the raw areas. Area Serial 1 - 5 is totaled (4.148 cm^2) and deducted from the total. The Result (57.859 cm^2) was calculated as a percentage of the total and that gives the percentage take of Patient 04 on POD 5 calculated using Image J® (93.31%) (Figure 5).

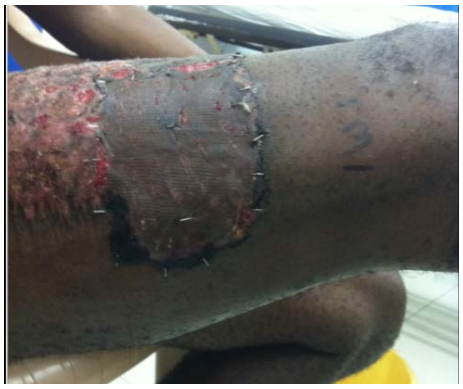

Figure 1. Image of patient 04 on POD 5 with 3 cm measured on the skin of the patient which was used as the known measure for setting of the scale.

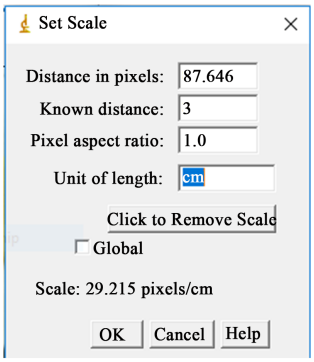

Figure 2. Setting of scale for measurement of patient 04 POD5.

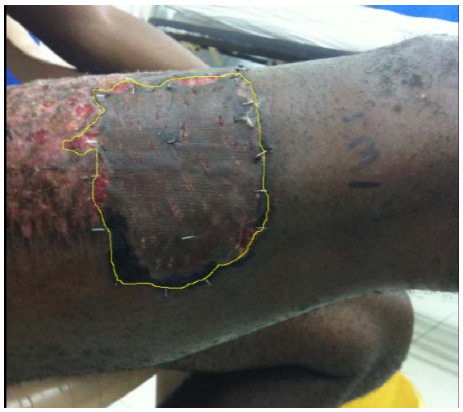

Figure 3. Image of 04 on POD 5 with an outline of the wound to be measured.

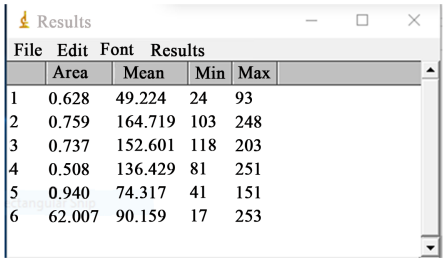

Figure 4. Wound area measurements obtained using Image J®.

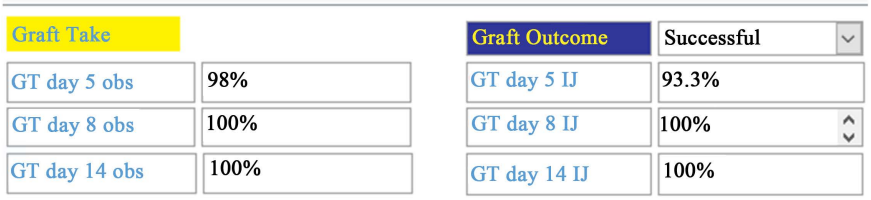

Figure 5. Recorded values of graft take obtained using observation and Image J® digital imaging for Patient 04.

3. Results

In total, 72 patients were included in the study. The median age of the patients was 30 years (range 3 months to 67 years). Patients aged 18 - 29 years had the highest population forming almost a third of the study population (30.6%). Men outnumbered women (54.2% vs 45.8). Thirty-one (53.5%) of the patients above 18 years were found to be obese or overweight.

Table 1 shows the wound aetiologies fell into one of six groups with the majority from trauma and burns *i.e.* 54 (75%). The BMI was calculated only for patients 18 years and above (*i.e.* 57).

Comparison between Estimation of Graft Take, Made by Observation and by Using Image J®

The graft take on Postoperative day 5, Postoperative day 8 and Postoperative Day 14 were recorded using both the Observation and Image J methods (Tables 2-4).

These were compared and statistically analysed. There was no statistically significant difference between the estimation of Graft take, made by observation and using Image J® digital programme. The only differences were seen with estimates of cellulitic wounds on Day 5 and Flap site wounds on day 14 (Table 5).

4. Discussion

Ulcers, including traumatic wounds, defects after resection of tumours, burn wounds, etc., impact negatively on the quality of life. Grafting, which aids faster wound healing, serves as a means to relief the patient of distress. Thus when a graft fails the impact on the patient is immense. Such a patient suffers psychological and financial difficulties, being saddled with the extra cost of another surgery and the extra cost of extended hospital stay.

Table 1. Demographic and clinical characteristics of study participants.

Characteristic		Proportion
		n, %
Age range (years) (N = 72)		3/12 - 67.0
Median age [Interquartile Range] (years) (N = 72)		30 [19 - 47.5]
Age group (N = 72)	<18 years	15 (20.8)
	18 - 29 years	22 (30.6)
	30 - 39 years	9 (12.5)
	40 - 49 years	9 (12.5)
	50 - 59 years	12 (16.7)
	>59 years	5 (6.9)
Gender (N = 72)	Male	39 (54.2)
	Female	33 (45.8)
BMI category (N = 58)	Normal	27 (46.5)
	Overweight/Obese	31 (53.5)
Ulcer aetiology (N = 72)	Trauma	28 (38.9)
	Burns	26 (36.1)
	Cellulitis	6 (8.3)
	Post ex tumour	5 (6.9)
	Flap site	3 (4.2)
	Fasciitis	2 (2.8)
	SSG donor site	2 (2.8)

Table 2. Graft take recorded by observation and Image J on postoperative day 5.

Patient Code	GT POD 5 Obs	GT POD 5 IJ
1	85	71
2	99	97.5
3	98	96.7
4	98	95.8
5	85	86.1
6	90	85
7	65	82.6
8	95	93.7
9	98	98.3
10	95	89.3
11	99	99.7
12	85	78
13	60	87.2
14	99	98
15	95	87.1
16	85	74
17	90	91.3

Continued

18	95	88.7
19	90	88.2
20	70	65.7
21	80	66.3
22	100	100
23	99	97.7
24	90	78.8
25	95	92.8
26	70	76
27	95	93
28	99	94.7
29	50	66.7
30	98	97.8
31	85	91.6
32	92	89.9
33	85	82
34	85	83
35	95	80.8
36	97	83
37	92	91.4
38	98	83.1
39	98	96.5
40	95	93
41	70	84.1
42	100	100
43	70	61.2
44	75	86.2
45	85	78.3
46	100	100
47	90	76
48	80	67
49	70	83
50	50	33.5
51	80	74.3
52	100	100
53	95	86.7
54	80	76.7
55	95	81.9
56	90	88.5
57	85	79.8
58	99	96.4

Continued

59	95	87.9
60	85	81.3
61	98	94.1
62	80	73.3
63	90	78.1
64	85	79.1
65	98	90.1
66	95	85
67	100	100
68	90	83.1
69	100	97.7
70	98	97.7
71	75	81.3
72	98	93.8

Table 3. Graft take recorded by observation and Image J on postoperative day 8.

Patient Code	GT POD 8Obs	GT POD 8 IJ
1	90	76
2	99	98.7
3	98	90.5
4	100	100
5	80	75.3
6	80	83.2
7	40	63.2
8	95	98.4
9	90	94
10	90	88.3
11	95	94.6
12	85	85.8
13	55	68.2
14	98	97.6
15	90	94.6
16	55	66.1
17	98	91.6
18	95	85
19	60	53.7
20	60	65.1
21	75	62
22	99	89.6
23	95	89.2
24	90	72.3
25	90	83
26	60	51.3

Continued

27	85	81.7
28	95	88.3
29	45	66.1
30	85	78.5
31	99	95.4
32	95	83.1
33	85	86.5
34	70	67.3
35	95	71
36	75	67.9
37	80	89.9
38	80	84.6
39	100	100
40	100	100
41	60	81.3
42	98	96.4
43	60	43.9
44	85	88
45	80	79.1
46	98	98.5
47	85	78.5
48	75	69.1
49	60	81.6
50	30	30
51	90	78
52	100	100
53	80	71.8
54	75	64.5
55	70	74
56	90	82.2
57	98	94.1
58	65	57
59	55	62.5
60	80	82.5
61	95	86.1
62	85	79.1
63	92	86.3
64	75	71.5
65	95	89.3
66	90	82.5
67	98	91.2
68	85	79.1
69	95	87.3
70	98	93
71	80	68.3
72	98	95.6

Table 4. Graft take recorded by observation and Image J on postoperative day 14.

Patient Code	GT POD 14Obs	GT POD 14 IJ
1	95	83.3
2	100	100
3	100	100
4	100	100
5	80	64.1
6	85	81.6
7	35	41
8	100	100
9	90	93.6
10	80	68.5
11	85	90.3
12	80	68.7
13	40	60.1
14	100	100
15	100	99.3
16	45	31.9
17	98	94.6
18	90	83
19	40	23.9
20	50	62
21	35	43
22	95	90.4
23	90	92.1
24	85	76.1
25	80	77.8
26	60	63.2
27	80	83.6
28	99	93.1
29	45	62.1
30	80	74.3
31	100	100
32	98	88.3
33	95	88.2
34	85	83.6
35	95	83.5
36	85	79.7

Continued

37	75	87.6
38	90	87.6
39	98	94.6
40	100	100
41	65	75.7
42	100	98.5
43	40	22.7
44	35	32.2
45	95	87.2
46	98	97.3
47	95	87.2
48	60	62.3
49	40	33.9
50	10	32.3
51	90	82
52	100	100
53	90	84.9
54	60	41
55	20	33.7
56	96	89.1
57	98	92.1
58	10	22.7
59	23	34.1
60	85	87.7
61	95	80.8
62	85	75.7
63	90	87.1
64	95	93.1
65	99	97.3
66	80	71.1
67	85	78
68	90	81.3
69	85	91.4
70	100	100
71	95	87.1
72	100	100

Table 5. Comparison of proportion of ulcer management using observational method and Image J® method in different ulcer aetiology.

Ulcer aetiology	*DAY* 5			*DAY* 8			*DAY* 14		
	Observation	*Image J*	*p-value*	*Observation*	*Image J*	*p-value*	*Observation*	*Image J*	*p-value*
	mean ± SD	*mean ± SD*		*mean ± SD*	*mean ± SD*		*mean ± SD*	*mean ± SD*	
Trauma (n = 28)	92.35 ± 10.64	90.44 ± 8.84	0.114	90.32 ± 12.88	88.21 ± 9.44	0.135	86.14 ± 19.84	84.04 ± 18.04	0.114
Burns (n = 26)	83.50 ± 11.14	81.75 ± 8.78	0.399	77.42 ± 15.33	74.55 ± 10.87	0.215	74.77 ± 22.83	72.54 ± 20.81	0.224
Cellulitis (n = 6)	95.17 ± 3.54	88.10 ± 8.23	0.047	77.88 ± 9.91	75.60 ± 12.13	0.493	60.00 ± 35.36	64.18 ± 28.51	0.340
Post ex tumour (n = 5)	92.80 ± 7.36	90.12 ± 10.44	0.281	86.40 ± 19.40	87.30 ± 15.55	0.754	85.00 ± 23.97	78.90 ± 30.42	0.154
Flap site (n = 3)	90.00 ± 5.00	84.50 ± 5.44	0.116	83.33 ± 5.77	77.70 ± 5.34	0.141	93.67 ± 3.21	87.07 ± 2.10	0.014
Fascitis (n = 2)	60.00 ± 14.14	47.35 ± 19.59	0.188	45.00 ± 21.21	36.95 ± 9.83	0.500	25.00 ± 21.21	27.50 ± 6.79	0.920
SSG door site (n = 2)	96.50 ± 2.12	91.45 ± 6.15	0.327	95.00 ± 7.07	97.30 ± 3.82	0.500	100.00 ± 0.00	99.65 ± 0.49	0.500

A variety of factors are believed to adversely influence skin graft take; haematoma, shearing movements [12], inadequate compliance, deficient blood supply [13], are examples. Infection is the second most frequent cause of Skin graft loss [14].

The percentage graft take, noted and documented, often goes a long way to influence management plans of whether to continue with wound dressing or to re-graft the wound. Therefore, the take of the skin graft, traditionally estimated by observation needs to be as precise as possible and dependable.

In this study, there was no significant difference between the estimation of graft take, made by observation and estimation made using Image J® digital programme. The fact that the residents who were estimating the graft take by observation, were blinded to the results by the Image J, removed biases and influences. Thus it can be said that the practice of estimating graft take by observation as done at NRPSBC is acceptable.

In this study, the estimation by observation was made by senior residents in plastic surgery. Therefore it can be said that in as much as the method of estimation by observation was found to be reliable, this reliability depended on the experience of the persons making the estimation.

5. Conclusion

Graft take ideally must be made by an objective method such as an image analyzer. However the method of estimation of graft take by observation is an acceptable practice and can be relied upon to make decisions on patient management.

Author Contribution

All authors have contributed to all process in this research, including preparation, data gathering and analysis, drafting and approval for publication of this manuscript.

Conflicts of Interest

This was a secondary finding in work (Evaluation of bacterial infection of split-thickness skin grafts at the Korle Bu Teaching Hospital) which was presented as a dissertation submitted to the Faculty of Surgery of the WEST AFRICAN COLLEGE OF SURGEONS in part-fulfilment of the requirements for the award of the Final Fellowship of the West African College of Surgeons (FWACS) in Plastic Surgery in October 2017. Financial Assistance was sort for and received from the Management of the National Reconstructive Plastic Surgery and Burns Centre of the Korle Bu Teaching Hospital.

References

[1] Andreassi, A., Bilenchi, R., Biagioli, M. and D'Aniello, C. (2005) Classification and Pathophysiology of Skin Grafts. *Clinics in Dermatology*, **23**, 332-337. https://doi.org/10.1016/j.clindermatol.2004.07.024

[2] Ratner, D. (1998) Skin Grafting: From Here to There. *Dermatologic Clinics*, **16**, 75-90. https://doi.org/10.1016/S0733-8635(05)70488-5

[3] http://practicalplasticsurgery.org/docs/Practical_12.pdf

[4] Valencia, I.C., Falabella, A.F. and Eaglstein, W.H. (2000) Skin Grafting. *Dermatologic Clinics*, **18**, 521-532. https://doi.org/10.1016/S0733-8635(05)70199-6

[5] Mutalik, S. and Ginzburg, A. (2000) Surgical Management of Stable Vitiligo: A Review with Personal Experience. *Dermatologic Surgery*, **26**, 248-254. https://doi.org/10.1046/j.1524-4725.2000.09182.x

[6] Schneider, C.A., Rasband, W.S. and Eliceiri, K.W. (2012) NIH Image to ImageJ: 25 Years of Image Analysis. *Nature Methods*, **9**, 671-675. https://doi.org/10.1038/nmeth.2089

[7] Abramoff, M., Magalhaes, P. and Ram, S. (2004) Image Processing with ImageJ. *Biophotonics International*, **11**, 36-42.

[8] Collins, T.J. (2007) ImageJ for Microscopy. *Biotechniques*, **43**, 25-30. https://doi.org/10.2144/000112517

[9] Girish, V. and Vijayalakshmi, A. (2004) Affordable Image Analysis Using NIH Image/ImageJ. *Indian Journal of Cancer*, **41**, 47.

[10] https://imagej.nih.gov/ij/docs/menus/analyze.html

[11] Zekri, A. and King, W. (1995) Success of Skin Grafting on a Contaminated Recipient Surface. *European Journal of Plastic Surgery*, **18**, 40-42. https://doi.org/10.1007/BF00183697

[12] McGregor, A. and McGregor, I. (2000) Free Skin Grafts. In: *Fundamental Techniques of Plastic Surgery*, Churchill Livingstone, Edinburgh, 35-59. https://doi.org/10.1016/B978-0-443-06372-5.50007-3

[13] Browse, N.L., Gray, L., Jarrett, P.E. and Morland, M. (1977) Blood and Vein-Wall Fibrinolytic Activity in Health and Vascular Disease. *British Medical Journal*, **1**, 478-481. https://doi.org/10.1136/bmj.1.6059.478

[14] Thornton, J. and Gosman, A. (2004) Selected Readings in Plastic Surgery. Vol. 10, Selected Readings in Plastic Surgery, Inc., Dallas.

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Three Reverse Adipofascial Cross Finger Flaps Used for Coverage of Raw Areas on Two Fingers of a Patient with Blast Injury of the Hand—Case Report

Medhat Emil*, Anood Alqaydi, Dalal AlMansoori, Dalia Medhat Habib, Marwa Alawadhi, Abrar Alteneiji, Omar Alameri

Department of Plastic and Reconstructive Surgery, Zayed Military Hospital, Abu Dhabi, United Arab of Emirates
Email: *medhatemil1@hotmail.com

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Abstract

We report a case of blast injury to the left hand which resulted in fractures of the fingers with exposure of bones and joints of the phalanges. We used three reverse adipofascial cross finger flaps raised at the same time from 2 fingers to reconstruct adjacent fingers of the patient. The patient recovered well post-operatively and had good range of movement of the fingers. This avoided the complications of the use of regional or distal flaps. To our knowledge, this is the first case reported in which three reverse adipofascial cross fingers flaps are raised at the same time, two of them from an injured finger, to cover three raw areas on two fingers of a patient.

Keywords

Adipofascial Flap, Reverse Cross Finger Flap, Blast Injury Hand, Simultaneous Flaps Fingers

1. Introduction

The first article that dealt with cross-finger flap was published in the year 1950 by Micheal Gurdin and John W. Pangman after the 2nd world war. They termed the procedure as "trans-digital flap". It was carved more on the lateral and volar surfaces of the donor digit [1]. Ten years later, Hoskins DH, published an article in which he described the design of the classic cross finger flap [2].

In 1978, Ivan Pakiam described the adipofascial cross finger flap [3] and further modifications took place in the design and shape of the flaps along the his-

tory [4]. In this case report we used 3 reverse adipofascial cross finger flaps to reconstruct 2 fingers with exposed bones and joints in a patient of war blast injury.

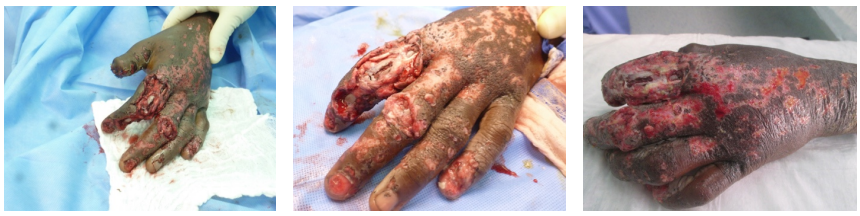
2. Case Report

A 27 years old young soldier was involved in a mine blast injury during the Yemen war which resulted in crush injury of his left hand, burns with multiple foreign bodies of the left upper limb and intraoccular foreign bodies. The patient was initially stabilized in the field hospital and transferred next day to the national military hospital for further management.

On examination, the patient was found having facial burns with foreign bodies in both the globes. He sustained also burns of the left shoulder and arm with multiple puncture wounds and lacerations. He had bad crush injury of the left hand with amputated terminal phalanx of the left thumb and the amputated stump had already been closed with sutures in the field hospital. The patient had also multiple fractures of the left hand with fracture proximal, middle and terminal phalanges of the left index finger and intraarticular fracture of the proximal interphalangeal joint. There was loss of the central slip of the extensor tendon with exposure of the bones of the proximal and middle phalanges of the index finger at the dorsal aspect. The middle finger also had tissue loss at the dorsal aspect of the proximal interphalangeal joint with intraarticular undisplaced fracture head of the proximal phalanx. There was also injury of the nail beds of the left index, middle and little fingers (**Figure 1** & **Figure 2**).

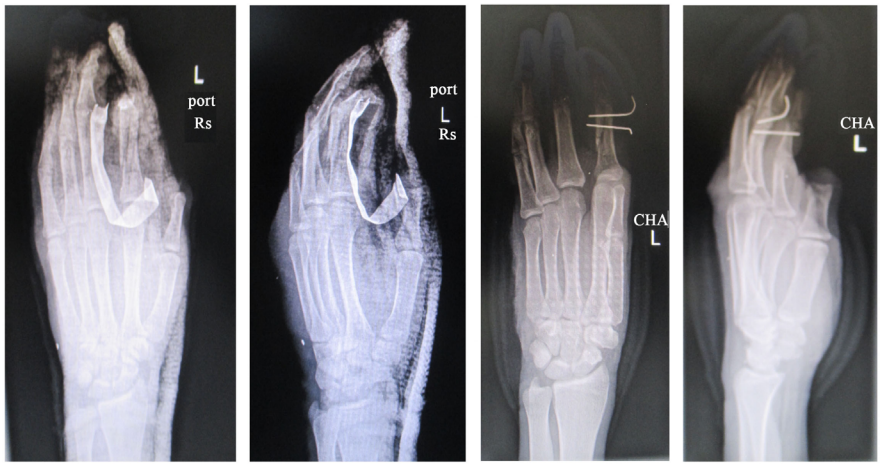
3. Operative Procedure

The patient was taken on the same day of admission to the operation theater and removal of foreign bodies from the eyes was done by the ophthalmologists. Debridement of the dirty wounds and burnt area of the upper limb and hand was done by the Plastic Surgery team. He was taken again to the operation theatre two days later for further debridement and repair lacerations of the nailbeds and arm wounds.



(a) (b) (c)

Figure 1. The hand injury sustained. (a) shows crush injury of the left hand with amputated terminal phalanx of the left thumb; (b) and (c) show exposure and fracture of the bones of the proximal and middle phalanges of the index finger with loss of the central slip of the extensor tendon at the dorsal aspect, tissue loss at the dorsal aspect of the proximal interphalangeal joint of the middle finger and injury of the nail beds of the index, middle and little fingers.



(a) (b) (c) (d)

Figure 2. X-rays of the patient. (a) & (b) Preoperative X-rays anteroposterior and oblique views; (c) & (d) Postoperative X-rays anteroposterior and oblique views.

After two more days when the wounds were clean and the burn on the left upper limb was found improving conservatively, the patient underwent under general anaesthesia the coverage procedure after fixing the longitudinal fracture of the proximal phalanx of the index finger by two transverse K-wires.

Two reverse adipofascial cross finger flaps were designed from the dorsal aspect of the proximal and middle phalanges of the left middle finger to cover the dorsal aspects of the proximal and middle phalanges of the left index finger.

A third reverse adipofascial cross finger flap was designed from the dorsal aspect of the middle phalanx of the left ring finger to cover the dorsal aspect of the proximal interphalangeal joint of the left middle finger.

The technique used in each of the flaps was the same. Incisions were made longitudinally in the skin at the radial side of the donor finger and turned transversely proximally and distally at the dorsal aspect of each of the donor phalanges. Dissection was done between the dermis and the underlying tissues under magnification raising the skin as full thickness skin graft till the ulnar aspect of the finger. From this aspect, the underlying subcutaneous tissue was raised as a flap in the opposite direction and dissected from the underlying paratenon of the extensor tendon till the radial aspect of the finger placing the base of the flap next to the recipient finger. Following meticulous haemostasis, the flaps were reversed and sutured to the recipient sites with 5/0 Vicryl sutures. The donor defects were covered with the previously elevated full thickness skin. The reversed sides of the subcutaneous adipofascial flaps were surfaced with split thickness skin grafts harvested from the left thigh. Holes were made in the graft to prevent haematoma formation. A soft dressing was applied with volar Plaster of Paris slab (**Figure 3**).

The patient was put on dressings and 3 weeks later division of the three flaps was done. There was raw area at the ulnar aspect of the left index finger which was grafted.

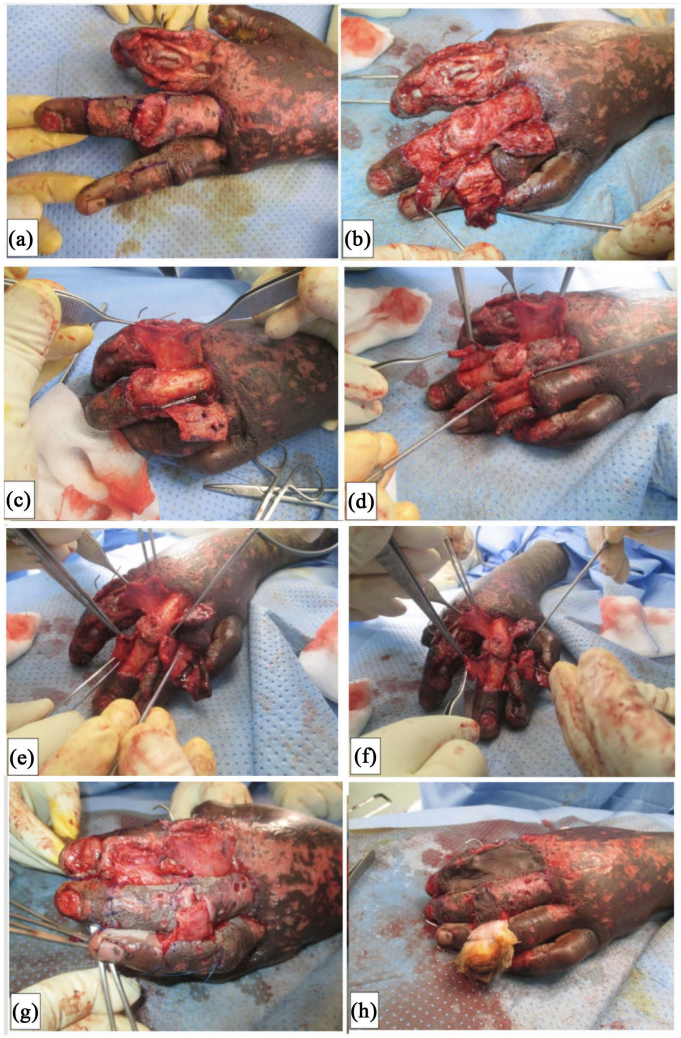
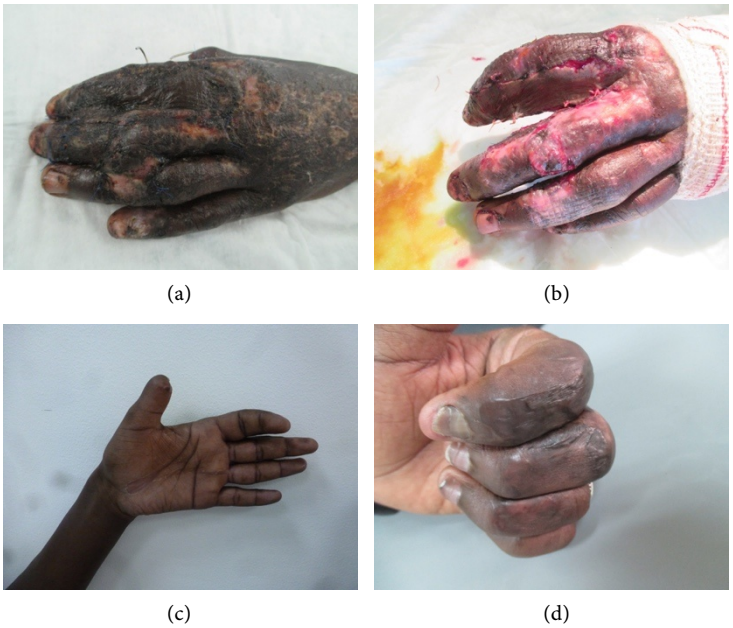


Figure 3. (a) shows K-wires fixation of the longitudinal fracture of the proximal phalanx of the index finger. Skin marking done; (b) Skin incised at the radial aspect of the middle and ring fingers and reflected and kept attached at the ulnar aspect of both the fingers; (c) The first reverse adipofascial flap at the proximal phalanx of the middle finger elevated from the ulnar aspect of the phalanx and reflected to cover the proximal phalanx of the index finger. Note the punctures in the flap and skin from the blast injury effect; (d)-(f) show the three reverse adipofascial cross finger flaps which were designed from the dorsal aspect of the proximal and middle phalanges of the left middle finger and the dorsal aspect of the middle phalanx of the left ring finger to cover the dorsal aspects of the proximal and middle phalanges of the left index finger and the dorsal aspect of the proximal interphalangeal joint of the left middle finger; (g) shows the three flaps after reversing and suturing them to the recipient sites. The previously reflected skin is sutured back proximally and distally to its original site but not at the radial side of the phalanges where the pedicles of the flaps are; (h) shows split thickness skin graft surfacing the reversed side of the adipofascial flaps.

The patient was put on physiotherapy till he got good range of movement of his left hand. Extension of the left index finger was limited to 20 degrees but he had full flexion with normal grip of the hand. The postoperative follow up period was 103 days and the patient went back to his home country (**Figure 4**).



(a) (b)

(c) (d)

Figure 4. (a) Two weeks post operatively. The grafts are settling on the recipient's sites; (b) Three weeks post operatively; immediately post division of the three flaps and skin grafting of the ulnar aspect of the index finger; (c) & (d) The left hand 3 months post operatively with slight limitation of extension with full flexion and normal grip of the hand.

4. Discussion

Many procedures are prescribed for coverage of raw areas of the hand. Distant flaps as the groin flap can be used but have the complications of stiffness of the shoulder and elbow joints [5]. Bulkiness of the flap is one of the drawbacks and subsequent defattening is usually required in most of the cases [6].

The reversed radial forearm flap can be used as fasciocutaneous or fascial flap with preservation of the radial artery but it has the disadvantage of having a large scar over the forearm and often the need for skin grafting to close the recipient site [7]. The reverse posterior interosseous flap is another option but in addition to the scarring on the forearm, it has the disadvantages of difficulty of dissection, inconstant anatomy of the artery, possibility of injury of posterior interosseous nerve and venous congestion of the flap. The flap also needs long time of elevation and a long learning curve [8].

Raising flaps from the hand itself can avoid such complications and provides tissues of similar texture and pliability. For these reasons we preferred to use the reverse adipofascial cross finger flaps rather than distant or regional flaps in our case.

The reverse adipofascial flaps are based on dorsal cutaneous branches from the proper digital arteries of the fingers. On his cadaveric study on 180 digits, Jefferson Silva showed that there are 2 constant branches in the proximal and middle phalanges from each proper digital artery and they have consistent sites of origin at predictable distances from the proximal interphalangeal joint for the long fingers [9]. We raised our three flaps; one from the proximal phalanx and

two from the middle phalanges based on this anatomical concept. In spite of having many puncture wounds of the blast injury in our flaps, they all were viable and healed well which shows the good blood supply of the flaps.

The adipofascial flaps can be raised in different ways. They can be used as homodigital flaps within the same finger as turnover flaps or as heterodigital flaps from one finger to the adjacent one.

The homodigital flap can be used to cover the dorsal aspects of the middle [10] and terminal [11] phalanges of the fingers. The flap is elevated from the dorsal aspect of a phalanx and turned over to cover the phalanx distal to it. In our case, this type of flap could not be used as there was no soft tissue on the dorsal aspect of the proximal phalanx of the index finger to be turned over to cover the middle phalanx of that finger so we had to use a heterodigital type of flaps i.e. flaps from adjacent fingers.

Heterodigital flaps can be formed of either only of the adipofascial tissue between the dermis of the overlying skin and the underlying paratenon over the extensor tendon of the phalanges or can be formed of that tissue with the overlying skin after de-epithelializing it [12]. In the last option skin graft is applied on both the turned over flap and the paratenon of the donor site as one piece.

We feel that this technique can have high possibility of implantation dermoid cyst formation if no adequate de-epithelialization of the flap is done. Also applying skin graft on both the donor and recipient digits may be unnecessary if only skin graft can be applied on the recipient digit in the procedure we used. This will avoid disfigurement of the donor digit and maintain its normal hairy skin shape. For these reasons we preferred that our flaps to be formed only of the tissue between the dermis of the overlying skin and the underlying paratenon over the extensor tendon of the phalanges. After turning the flap 180 degrees like a page of a book and flap insetting is performed on the recipient site, the previously elevated overlying skin is turned back to cover the donor site without the need to do skin grafting of the donor digit.

5. Conclusion

The reverse adipofascial cross finger flap is a reliable procedure for covering raw areas of adjacent fingers. It provides soft tissue of proper thickness with good pliability and keeps the upper limb free of stiffness of distant flaps. To our knowledge, this is the first case reported in which three of these flaps were used to cover raw areas on two fingers of a patient at the same time.

Conflicts of Interest

The authors declare that they have no conflict of interest.

Compliance with Ethical Standards

All procedures performed in this study involving human participant were in accordance with the international ethical standards. Approval from the hospital

Scientific and Research Committee was obtained.

Informed Consent

Additional informed consent was obtained from the participant for whom identifying information is included in this article.

References

[1] Gurdin, M. and Pangman, W.J. (1950) The Repair of Surface Defect of Fingers by Transdigital Flaps. *Plastic and Reconstructive Surgery*, **5**, 368871.

[2] Hoskins, H.D. (1960) The Versatile Cross-Finger Pedicle Flap. *JBJS Case Connector*, **1**, 261-277.

[3] Pakiam, A.I. (1978) The Reversed Dermis Flap. *British Journal of Plastic Surgery*, **31**, 131-135. https://doi.org/10.1016/s0007-1226(78)90062-0

[4] Fang, F. and Chung, K.C. (2014) An Evolutionary Perspective on the History of Flap Reconstruction in the Upper Extremity. *Hand Clinics*, **30**, 109-122. https://doi.org/10.1016/j.hcl.2013.12.001

[5] Graf, P. and Biemer, E. (1992) Morbidity of the Groin Flap Transfer: Are We Getting Something for Nothing? *JPRAS*, **45**, 86-88. https://doi.org/10.1016/0007-1226(92)90162-q

[6] Arner, M. and Möller, K. (1994) Morbidity of the Pedicled Groin Flap a Retrospective Study of 44 Cases. *Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery*, **28**, 143-146. https://doi.org/10.3109/02844319409071192

[7] Hansen, A.J., Duncan, S.F.M., Smith, A.A., Shin, A.Y., Moran, S.L. and Bishop, A.T. (2007) Reverse Radial Forearm Fascial Flap with Radial Artery Preservation. *Hand*, **2**, 159-163. https://doi.org/10.1007/s11552-007-9041-7

[8] El-Sabbagh, A.H., Zeina, A.A.El-M., El-Hadidy, Al-M. and El-Din, A.B. (2011) Reversed Posterior Interosseous Flap: Safe and Easy Method for Hand Reconstruction. *Journal of Hand and Microsurgery*, **3**, 66-72. https://doi.org/10.1007/s12593-011-0042-y

[9] Braga-Silva, J. (2005) Anatomic Basis of Dorsal Finger Skin Cover. *Techniques in Hand and Upper Extremity Surgery*, **9**, 000-000. https://doi.org/10.1097/01.bth.0000173726.99670.f0

[10] Naji, S.H. (2001) Dorsal Adipofascial Turnover Flap for Dorsal Defects of Finger Injury. *Medical Journal of Babylon*, **8**, 67-72.

[11] Ozdemir, R., Kilinç, H., Sensöz, O., Unlü, R.E. and Baran, C.N. (2001) Innervated Dorsal Adipofascial Turnover Flap for Fingertip Amputations. *Annals of Plastic Surgery*, **46**, 9-14. https://doi.org/10.1097/00000637-200101000-00003

[12] Robbins, T.H. (1985) The Use of De-Epithelialised Cross-Finger Flaps for Dorsal Finger Defects. *British Journal of Plastic Surgery*, **38**, 407-409. https://doi.org/10.1016/0007-1226(85)90253-x

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Historical Case Report: 45 Years of the First Plastic Surgery in Morbid Obese in Brazil and Their Weight Loss

José Humberto Cardoso Resende[1], Cristine Mara Fragoso Dos Santos Oliveira[2], Heitor Dos Santos Leão[2], Bruno Viana Martins[2], Brenda Martins Fernandes[2], Valentina Ruvieri Silveira[2], Juliana Araújo Naves[2], Ítalo Julierme Barros Duarte[2], Emídio Silva Falcão Brasileiro[3]

[1]UNIFAN, Coordinator of the Work, Aparecida de Goiânia, Brazil
[2]UNIFAN, Academics of Medicine, Aparecida de Goiânia, Brazil
[3]UNIFAN, Coordinator of Research, Aparecida de Goiânia, Brazil
Email: jresen99@hotmail.com

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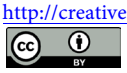

Abstract

Before the 70s, in Brazil, each city had its morbidly obese, considered as the "excessive fats", in very small numbers if we compare it with the current percentage. There was no classification of the degree of obesity by the body mass index (BMI) as we have today. By chance, on a Saturday in June 1975, at the Outpatient Clinic of the 23rd Infirmary of Santa Casa da Misericórdia Hospital in Rio de Janeiro, arrived the patient I. S., 41 years old, 1.70 m tall, supported by her two children, weighing 210 kg in weight body. Knowing that bariatric surgery only appeared in the 1980s, before that, patients with morbid obesity were left to their own devices, with hypertension and diabetes. The patient I. S. was hospitalized for 3 years in our Plastic Surgery Service, having received nutritional monitoring, had sporadic discharges and undergone 9 reparative plastic surgeries. She was discharged weighing 71 kg, with self-esteem recovered and happy to start a new life, without hypertension and diabetes.

Keywords

Obesity, Hypertension, Diabetes, Plastic Surgery, Slimming

1. Introduction

This manuscript will serve to guide future doctors who will be able to deepen their studies on this disease called OBESITY. After 45 years of the first plastic

surgery in morbidly obese in Brazil, we thought it was a good idea to honor the colleagues who were present during this time of tireless professional work at the Santa Casa da Misericórdia Hospital in Rio de Janeiro, Brazil. This was the first step before we founded the Chapter of Plastic Surgery in Obesity at the Brazilian Society of Plastic Surgery and, later, we implemented the "Obese Workshop" at the Federal Hospital of Servers of the State of Rio de Janeiro.

We know that subsistence and nourishment are our source of life. During the medical career, we learned that this is a pathology that kills if it is not treated in time. Morbid obesity involves, in addition to overeating, a lack of physical exercise, which causes numerous disorders resulting from hypertension and diabetes, and affects the psychological and social sector of patients. Due to bodily deformity and the difficulties of mobilization, they remain in a form of servitude all the time, which prevents them from enjoying life's pleasures, gradually causing paralysis, as shown in this career first case (**Figure 1**). The constraints, according to Scherer (2012) [1], and the upsets caused by the disease, leave the patient without opportunities in the world. For countless reasons, we know that there are no job opportunities for the morbidly obese. Thus, we consider this disease to be a public health problem [2]. Words banned by the disease: thinness, elegance, health, happiness, relationships, joys, in addition to the ban on coming and going. There are no spaces in the chairs of cinemas and theaters, buses, stretchers in hospitals, bathrooms, airplanes, all due to the lack of attention from public agencies. Even struggling a lot, sending projects, we were unable to fulfill the dream of an appropriate hospital for 40% of the overweight population and 10% of morbidly obese people. What values are being developed in the current "Society"? (Carvalho, 2010) [3].

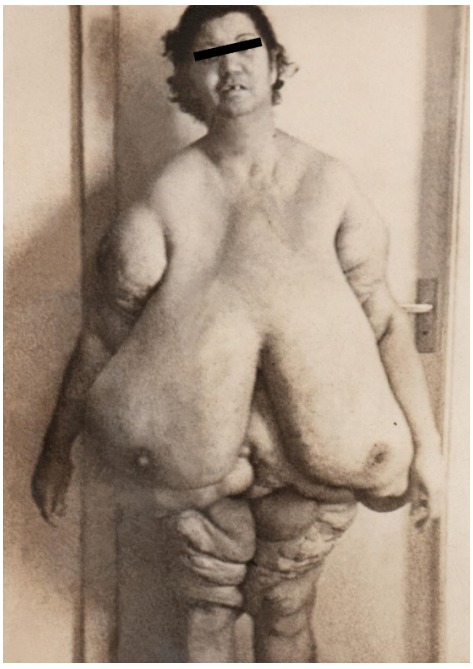

Figure 1. 1st day of hospitalization.

They are considered disabled (Medeiros, 2018) [4] in some tasks and cannot exercise their rights equally to the slender. In the case reported, we admitted for philanthropic reasons, even without future planning. The patient said: "Doctor, you saw me, be the *mechanic*, I'm in the *workshop*". On the following Monday, with the authorization of Prof. Xavier Lopes, we started to create a nutritional and surgical protocol to attend to her. We are very grateful to our head nurse, the Dominican Sister Lúcia Sezures, who was the right hand in this period. One day, she said: "Doctor, don't worry, the food here is so bad that the patient will lose weight anyway". I had already finished my 2-year residency in general surgery and was in the second year of plastic surgery. We idealize performing body relief surgeries concomitantly with weight loss using protein and vitamin supplements every 6 hours [5]. The largest surgical piece excised was the breasts with 32 kg. All laboratory tests of the patient, upon admission, were altered, including blood pressure and blood glucose, 170 × 110 mmHg and 210 mg/dL, respectively. Although obese, she had a 34% hematocrit.

We felt compelled to remember in honor of the courageous team that helped us at a time when everything was new and undergoinga learning stage. Anesthetists: Dr. Kleber Sardenberg (RJ), Dr. Marcos Botelho (RJ) and Dr. Marco Antonio Garambone (RJ); Surgeon: Dr. José Humberto Cardoso Resende (RJ); Assistants: Dr. Elmo Glória Filho (RJ), Dr. Hermes Galvão de Sá Filho (PB), Dr. Zeneide Alves de Souza (AP); Nurse: Sister Lúcia Sezures; Location: 23rd Infirmary of Santa Casa da Misericórdia Hospital in Rio de Janeiro.

2. Material

45 years ago, it was not usual to document the pre and postoperative surgeries. If there was any kind of lawsuit, which was rare, black and white photos were accepted. At that time, we used the old "slides"—slides (sheets) of an image projector presentation. Even so, we are amazed to have kept and organized, at least, the main results of the reparative surgeries that, today, I call "body relief".

After some Plastic Surgery Services rejected the lady I. S.'s case, I took courage and made a plan that I thought was more adapted to what I knew. A careful pre-operative, which I follow to this day, and the most important: to not put the patient's life at risk. Nutrition was controlled with protein and vitamin supplements [6]. After she lost 20 kg in 6 months, we checked her laboratory exams, which would allow us to start surgery. All results were already within normal limits. We decided to do only one body part at a time, a technique recommended until today [7]. Then, in the 6th month, we marked the extirpation of the larger part, the breasts (**Figure 2**), which had two layers of tissue, skin and subcutaneous cell, never seen before in professional life. After 4 hours of procedure, we ended with the removal of 18 kg of the right breast and 14 kg of the left breast, making a total of 32 kg of breast pieces. We can observe the reduction of the layers seen on the back (**Figure 3** and **Figure 4**). Some photos were not found or we did not think it was due to the impossibility of identification due to the number of overlapping layers. The preoperative and postoperative periods

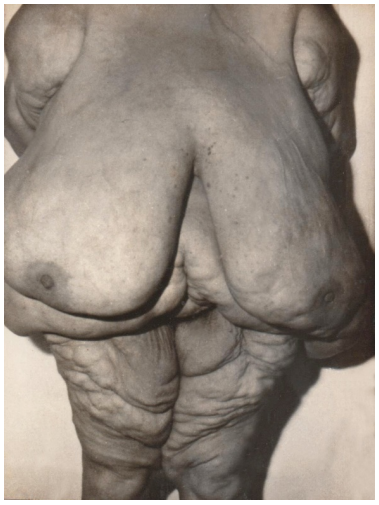

Figure 2. Preoperative of the breasts (2 layers).

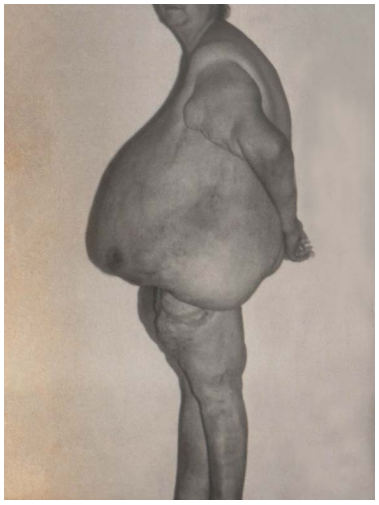

Figure 3. Preoperative profile after 1st weight loss.

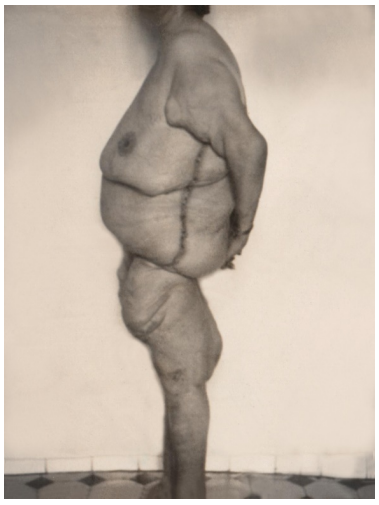

Figure 4. Postoperative of the 1st breast surgery and postoperative with 4 days of longitudinal abdominal surgery.

went smoothly, having been assisted by 4 surgeons and 3 anesthetists. After 6 months, we studied and performed the second surgical intervention, the first to remove the abdomen. When we put the patient on the table, the anesthetist said: "Dr. José Humberto, the belly fell to the sides of the table". Thus, we concluded that, as it was relief surgery, but not yet described in the literature, we decided to make the longitudinal surgical incision, which started in the armpit and ended near the pelvic region (**Figures 5-7**). At the end of this surgical time, she had her only cardiac arrest for 3 years, which was soon reversed. Six months later, we performed the second repair of the breasts and, with an interval of 3 months, we performed the second abdominal repair, with a transverse incision. Also in the 2nd year of hospitalization, we underwent surgery on the thighs and the pubic mound (**Figure 8** and **Figure 9**). In the last six months of the 3rd year of hospitalization, we performed the last surgeries to decrease the diameter of the arms (**Figure 10** and **Figure 11**). For the purpose of comparing pre and postoperative periods, at the end of the 3 years, we show the result (**Figure 12** and **Figure 13**). The patient arrived weighing 210 kg and was discharged with 71 kg. She arrived wearing a giant dress, which fit 4 people inside (**Figure 14**), and was discharged wearing "jeans" and mid-heel shoes [8]. It is worth remembering that, at that time, we did not have a bibliographic review that would assist us in choosing the technique. For this reason, we chose linear incisions with the intention of "body relief" [9] [10] for the patient.

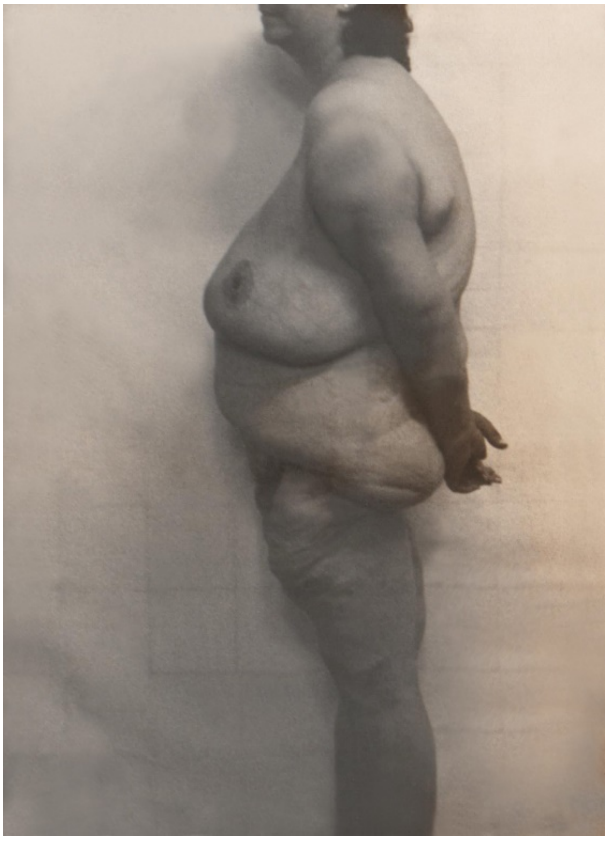

Figure 5. Postoperative profile at 6 months.

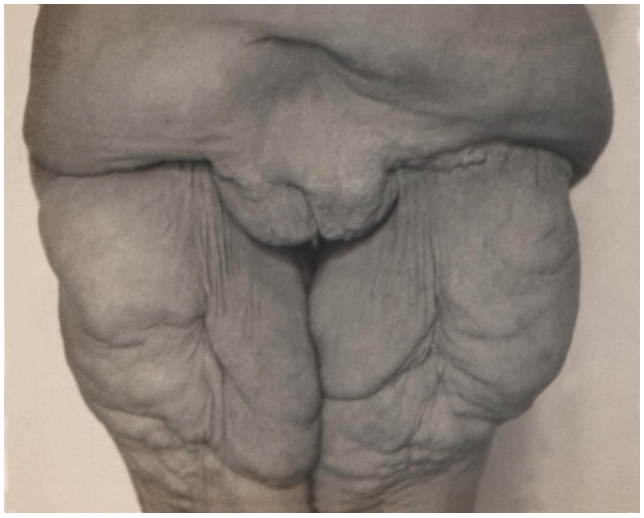

Figure 6. Preoperative of thighs and pubic mount.

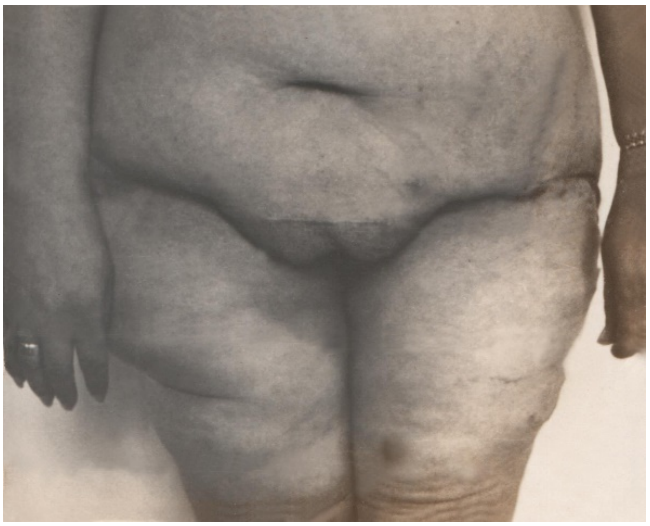

Figure 7. Postoperative of thighs and pubic mount and abdominal postoperative with transverse incision.

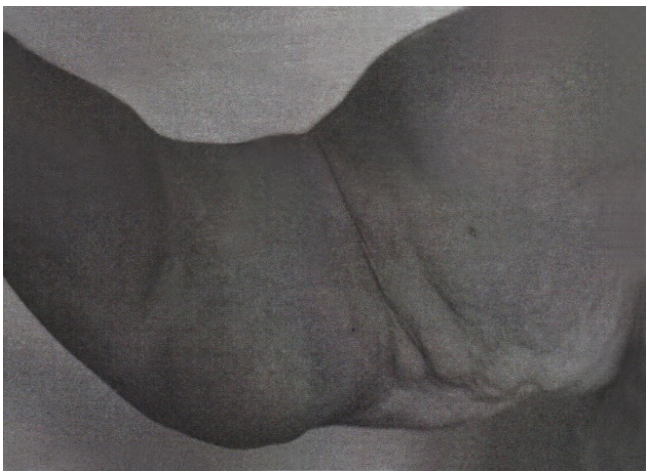

Figure 8. Preoperative arm.

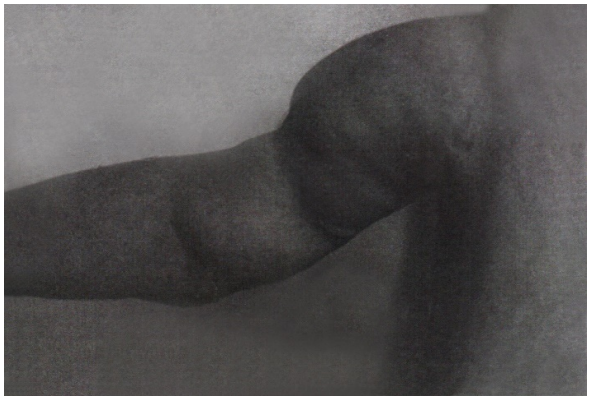

Figure 9. Postoperative arm.

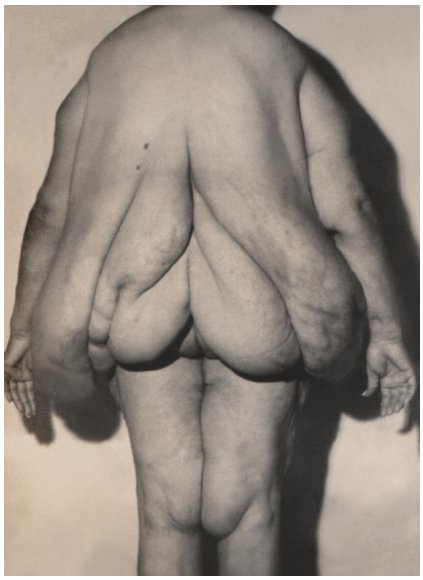

Figure 10. Preoperative of the back.

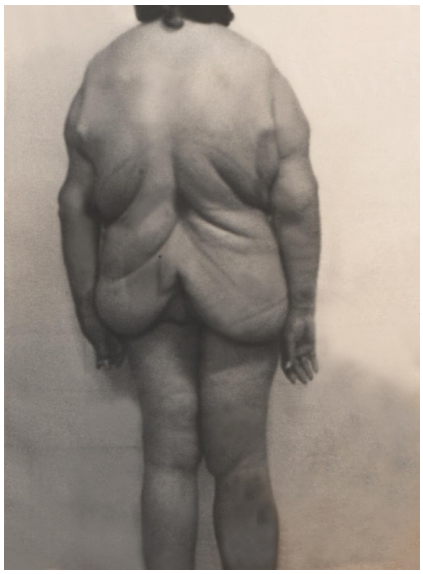

Figure 11. Post-operative of the back after the 1st surgery of the breasts and abdomen.

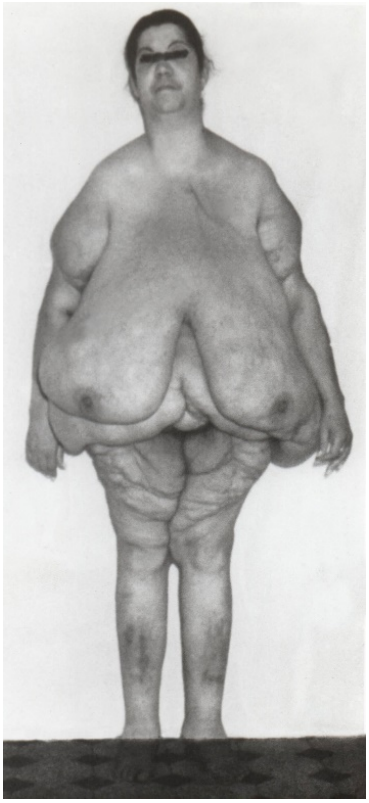

Figure 12. Preoperative of the 1st surgery. Hospitalization—210 kg.

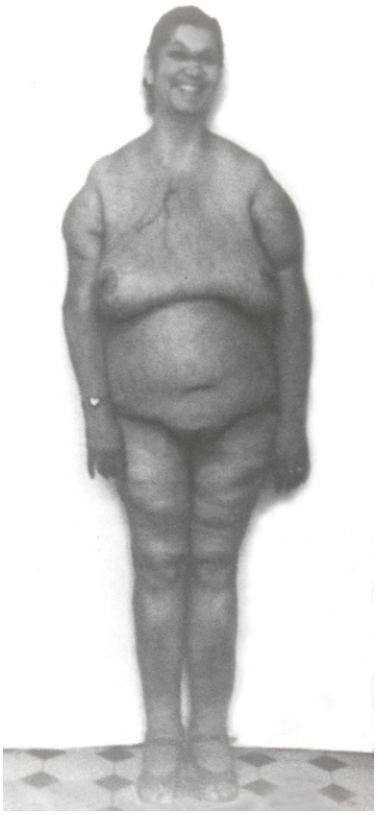

Figure 13. Last postoperative of the 9 reparative surgeries performed. High—71 kg.

Figure 14. Dress worn on the 1st admission (4 people inside).

3. Discussion

Perhaps today, after the bodily relief of the breasts, I would recommend bariatric surgery, even though I know that weight loss depends on a great psychological preparation, because when they are slowly monitored they are more advantageous. We have already published several articles on the subject, but even so, there is a large population increase at the moment. Recalling this case report will serve to open doors for more publications and awaken young doctors to this specialty that covers cases like this, which, one day, will be considered public health. Saying that there is nothing to do is an easy solution. It was difficult to face the case, with no future perspective and uncertain results. Even today, after 45 years, we still consider it a complicated case, but one that could not be ignored in view of the patient's age and the psychological severity she was experiencing. We always respect different or even contrary opinions. Today, perhaps, the postoperative results could be more acceptable; however what we achieved was the best for that moment or that time.

In this reported case, we had to develop a solution taking into account the patient's age, 41, her willingness to live and finish raising her children. It was not a common case and we did not have many resources at the time. Despite numerous opinions from more experienced teams, none of them gave us plausible alternatives. The idea of remembering the case came with the interest of encouraging younger specialist doctors to have the courage to help others even know-

ing the risks and the uncertain outcome. Discernment and dedication in hard times, using common sense, respecting science and having faith will provide a better result. A hypertensive and diabetic woman who was discharged with all normal laboratory tests [11].

4. Conclusion

As professionals and as human beings, we are very happy to see the result of this reported case. Monitoring and being part of this process, from the arrival of patient I. S., weighing 210 kg, carried by relatives, until being discharged, wearing jeans and 71 kg is the real payment for the well being provided to her. Seeing a person suffering from hypertension, arriving with 310 mg/dL of blood glucose and blood pressure 170 × 110 mmHg, leaving the hospital with 90 mg/dL of blood glucose and 130 × 80 mmHg of blood pressure is the best final balance we could have obtained, giving us the certainty, it was worth it! At that time, we still did not have the collaboration of bariatric surgery or liposuction (1980) [12]. The doors will always be open for us to evolve and, certainly, with the appearance of many new techniques for improvements in all specialties. It is important to remember that 9 surgical acts were performed, without bariatric and without liposuction, which would have contributed a lot. The best gratitude was to witness the degree of joy, satisfaction of the patient and the social inclusion that we felt during the ultimate discharge.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Scherer, P. and Santos, A. (2012) Weight That Is Not Measured on the Scale: The Repercussions of Obesity on the Subjects' Daily Lives. Masters Dissertation, Pontifical Catholic University, Rio Grande do Sul, Brazil.
http://hdl.handle.net/10923/5192

[2] Resende, J.H.C., de Moura, Á.I., Mariano, A.C.A., Silva, H.K., Silva, H.L., Campos, I.A., de Lima, L.C.F., Gabriel, M.B. and Silverio, W.O. (2019) Gigantomasty in Female Workers: "Public Health Cases". *Modern Plastic Surgery*, **9**, 1-7.
https://doi.org/10.4236/mps.2019.91001

[3] De Carvalho, P.S., *et al.* (2007) Bariatric Surgery Cures Metabolic Syndrome? *Arquivos Brasileiros de Endocrinologia e Metabologia*, *São Paulo*, **51**, 79-85.
http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0004-27302007000100013&lng=en&nrm=iso

[4] Medeiros, B., *et al.* (2018) The Importance of Accessibility in Organizations. *Proceedings of the VI Scientific Day of Campos Gerais in Ponta Grossa*, Paraná, 24 September 2018, Poster. Edition v. 16.
https://www.iessa.edu.br/revista/index.php/jornada/article/view/684

[5] Ministry of Health (2008) Bariatric Surgery in the Treatment of Morbid Obesity. *Brazilian Health Technology Assessment Bulletin*, **III**.

https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=2ahUKEwjMzvSbnPDoAhXsH7kGHRIPDIYQFjAAegQIAhAB&url=http%3A%2F%2Fwww.rebrats.saude.gov.br%2Finstitucional%2Fbrats%3Fdownload%3D104%3An-05-cirurgia-bariatrica-no-tratamento-da-obsidade-morbida&usg=AOvVaw0FMS6Khv4gkl9oIm0bfWpq

[6] Rocha, L.J.L.F., De Vilhena, J. and De Vilhena Novaes, J. (2009) Morbid Obesity: When Eating Goes far Beyond Food. *Psicologia em Revista*, *Belo Horizonte*, **15**, 77-96. http://pepsic.bvsalud.org/scielo.php?script=sci_arttext&pid=S1677-11682009000200006&lng=pt&nrm=iso

[7] Younis, F., *et al.* (2020) Endoscopic Treatment of Early Leaks and Strictures after Laparoscopic One Anastomosis Gastric Bypass. *BMC Surgery*, **20**, 33. https://doi.org/10.1186/s12893-020-0686-2

[8] Paul, M.A., Opyrchał, J., Knakiewicz, M., Jaremków, P., Duda-Barcik, Ł., Ibrahim, A.M.S., *et al.* (2020) The Long-Term Effect of Body Contouring Procedures on the Quality of Life in Morbidly Obese Patients after Bariatric Surgery. *PLoS ONE*, **15**, e0229138. https://doi.org/10.1371/journal.pone.0229138

[9] Resende, J.H.C. (2003) Gigantomastia. In: *Plastic Surgery—Fundamentals and Art—Aesthetic Surgery*, MEDSI, São Paulo, Vol. 1, 555-561.

[10] Resende, J.H.C. (2008) Resende's Technique for Correcting Gigantomastia—Body Relief Surgery, Treated Plastic Surgery in Obesity. Rubio, Rio de Janeiro, 357-365.

[11] Robles-Cervantes, J.A., Yanes-Dias, S., Cardenas-Cama-Rena, L. (2004) Modification of Insulin, Glucose and Cholesterol Levels in Nonobese Women Undergoing Liposuction: Is Liposuction Metabolically Safe? *Annals of Plastic Surgery*, **52**, 64-67. https://doi.org/10.1097/01.sap.0000096448.59407.43

[12] Hong, Y.G., Kim, H.T., Seo, S.W., *et al.* (2006) Impact of Large-Volume Liposuction on Serum Lipids in Orientals: A Pilot Study. *Aesthetic Plastic Surgery*, **30**, 327-332. https://doi.org/10.1007/s00266-005-0010-7

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Morbid Obesity: A Review on the Reasons for Impediments to Physical Exercises and Social Activities

José Humberto Cardoso Resende[1], Mattheus Duarte da Veiga Jardim[2], Brena Thamyres de Andrade Irineu[2], Leydeane Rosa Gomes Gontijo[2], Lucas Radi Cruvinel[2], Zeno Augusto de Sousa Neto[2], Rodrigo Gonçalves de Oliveira[2], Daniel Soares de Araújo[2], Emídio Silva Falcão Brasileiro[3]

[1]UNIFAN, Work Coordinator, Aparecida de Goiânia, Brazil
[2]UNIFAN, Academics of Medicine, Aparecida de Goiânia, Brazil
[3]UNIFAN, Research Coordinator, Aparecida de Goiânia, Brazil
Email: jresen99@hotmail.com

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Abstract

Morbid obesity is regarded as a disease due to excess body weight, causing a silence of life as a whole and entailing the most varied disabilities for the person, such as: physical, social, psychological, affective, etc. It represents cases of "public health", thereby involving competent bodies in the development of solutions that encompass various medical specialties and other health fields, in addition to influencing the mind of these people, causing depression that, due to metabolic involvement, can evolve to the death of the individual. The participation of multidisciplinary health focuses on weight loss, freely and spontaneously, or on the indication of bariatric surgery. We know how difficult it is to lose weight. In order to achieve successful procedures, we recommend the "Obese Workshop" or pre- and post-surgical follow-ups close to the patients, with a view to avoiding recurrences or the "accordion effect" (very common), which can interfere with the Body Mass Index (BMI). Everyone, males and females, complained of tiredness and the impossibility of any physical exercise, even the lightest and simplest to be performed, in addition to the fact that they cannot attend gyms and are unable to open a simple door handle.

Keywords

Obesity, Hypertension, Diabetes, Bariatric, Illness, Physical Exercise

1. Introduction

In this manuscript, we will focus only on morbid obesity, which, according to the Brazilian Society of Diabetes, grade III will take place when the BMI is higher than 40 [1], in the last 5 years, or 35 to those resistant for 2 years with chronic diseases [2].

Before the 1980s, we did not have the collaboration of bariatric surgery. Weight loss was conducted only with dietary restrictions, with which we almost never reached success. With the onset of Gastric *Bypass*, Gastric Band and duodenal surgeries [3], everything came to provide hope for people condemned by hypertension and diabetes, due to the capacity to improve metabolism [4] [5]. In 2019, the most widely used restrictive surgery was Gastric *Bypass* in a large proportion optional compared to other techniques.

We know that morbid obesity prevents patients from doing any type of physical exercises due to the difficulty in walking, deambulating and even getting out of bed [5] (**Figure 1** and **Figure 2**). Differently from the obese, the morbid individuals do not lie that they eat a lot. They were followed-up for two years. The main complaint of all men was pendulous abdomen, while for women, it was gigantomasty (large and heavy breasts).

Everyone complained about the huge effort they had to make to perform minimal physical exercises, such as: opening doors or taking water from the filter. They are hypertensive and diabetic patients who, at the slightest effort, complain of breathing difficulties and tiredness. We draw the attention of the health authorities to their duty to adapt, in clinics and hospitals, the width of the doors and the safety of the toilets, which can be prepared in more reinforced ways, without the risk of breaking, which could cause greater complications for the physical state of the patients [6]. Among the risks inherent to the morbidly obese individuals, we have dozens of associated diseases, such as: diabetes, sleep apnea, high blood pressure, stroke, atrial fibrillation, heart diseases, asthma, hernias, gastric reflux, pancreatitis, infertility, varicose veins, besides the most striking: social stigmatization [7]. Patients report that they are not invited to events, they have difficulty in making a simple gesture and even the fact that they deal with restrictions when buying clothes [8]. Commonly, obesity leads to alcoholism, drugs and depression.

Pharmacological treatments would only work if accompanied by physical exercises, which we cannot achieve in cases of morbidity III [9]. During these more than 40 years caring for the obese patients and coordinating the "Workshop", I was able to evaluate the degree of difficulty encountered by the morbidly obese patients. I was once called to assist an obese man and asked where he was. They [staff] took me to the ambulance, opened the rear door, and then I perceived that the patient was lying there on the floor of the car. The patient weighed 205 kg, so that he could only be weighed on a load weighing equipment, which was neither conventional nor decent.

Before a bariatric surgery, in several cases, we are called upon to perform the

surgical healing of hernias and eviscerations, in order to allow the accomplishment of the restrictive surgery later (Figure 3 and Figure 4).

Usually, we firstly perform body relief surgery on giant breasts, where we remove more than 10 kg of body mass (Figure 5 and Figure 6), and then indicate bariatric surgery, with the purpose of reducing intraoperative surgical risks.

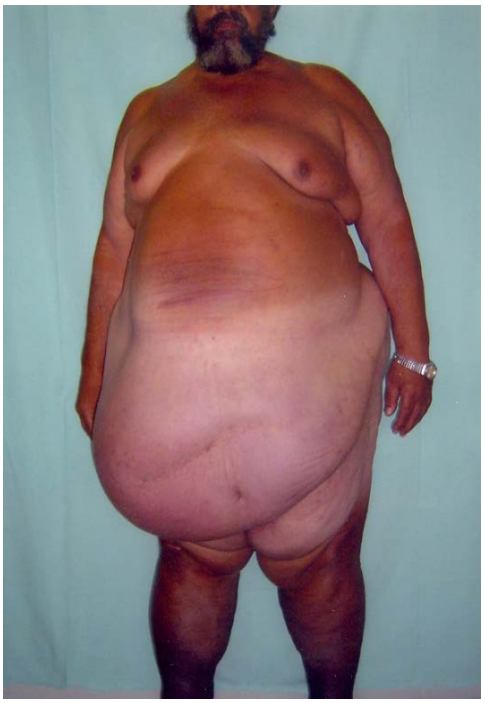

Figure 1. Morbid obese male patient with giant abdomen.

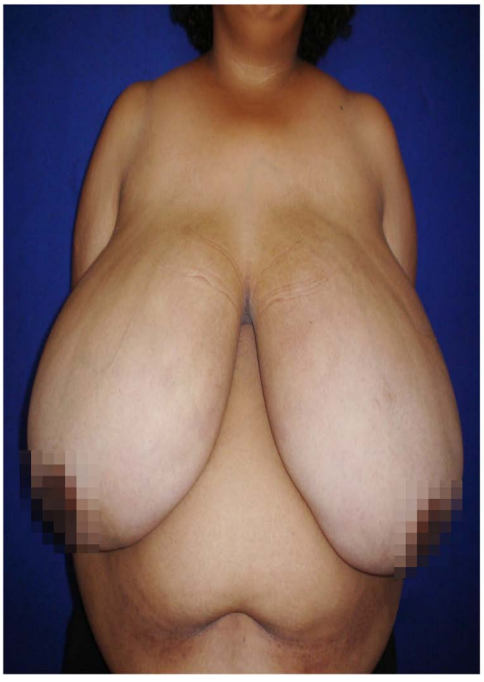

Figure 2. Morbid obese female patient with gigantomasty.

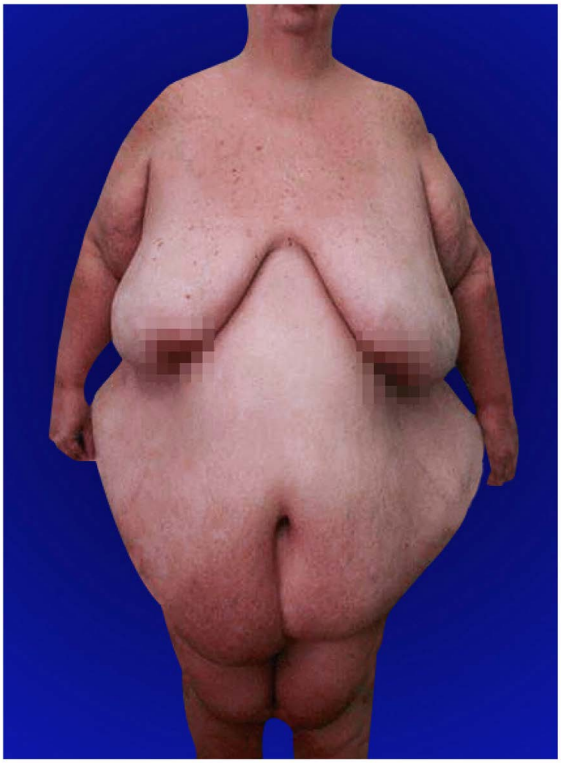

Figure 3. Obese male patient with hernia and evisceration.

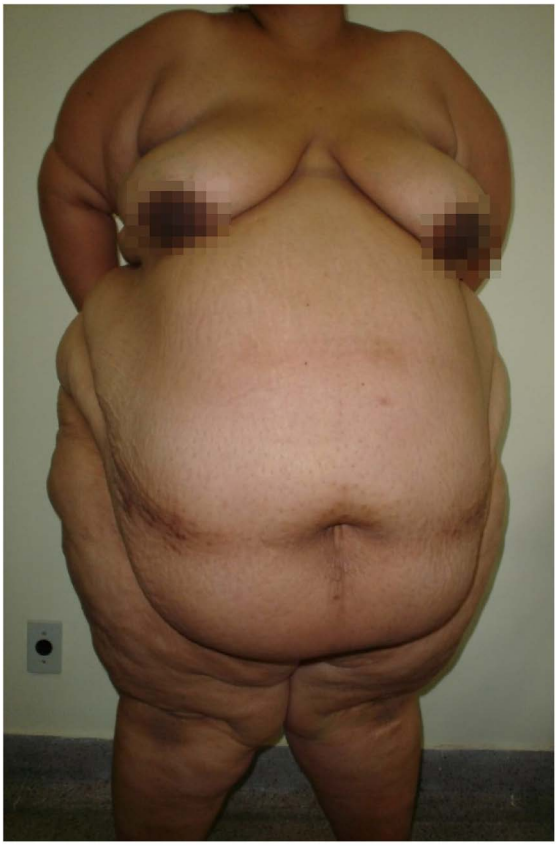

Figure 4. Obese female patient with hernia and diastase in straight abdominal muscles.

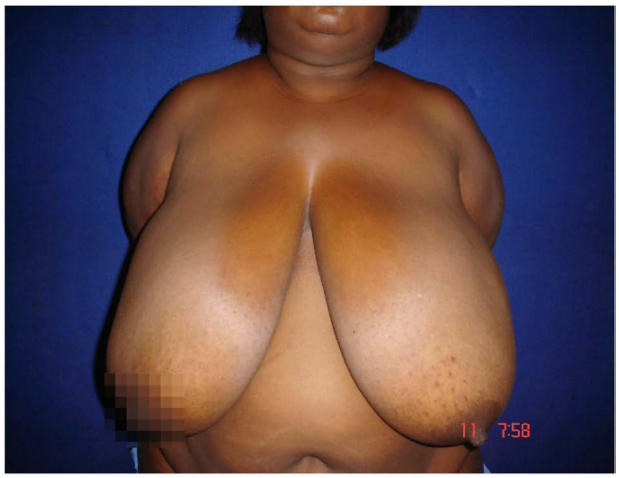

Figure 5. Preoperative of gigantomasty.

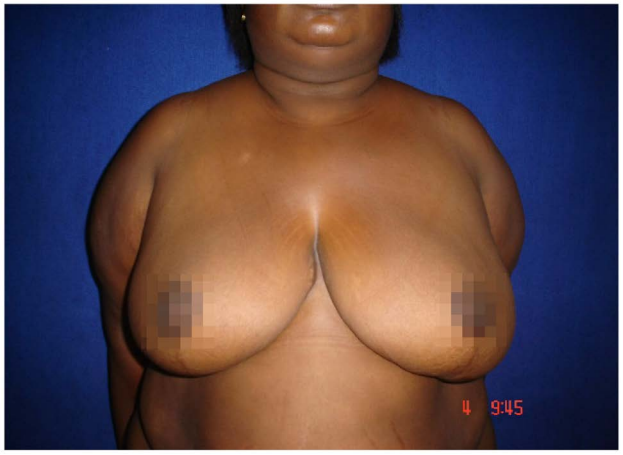

Figure 6. Postoperative with 5 days.

In many cases, the morbidly obese men have a thin voice and have their genitals covered up. Breasts are not always considered to be organs of identification when it comes to morbidly obese people. In **Figure 6**, we can note that there was total graft of the areola-papillary complex and decreased weight of the breasts, thereby greatly improving the quality of life of patients, even before the bariatric surgery is done.

All patients who lose more than 50 kg, during the postoperative period, request plastic surgery to have again the same type of body that they had before obesity, as if, by miracle, this could happen. We remember that, in any case, weight loss brings great satisfaction to most patients because they have reached a better quality of life, as well as the power to reintegrate into society in terms of clothing [10]. In this manuscript, we are not addressing morbid obesity in adolescence or childhood, where emotional and psychological problems (bullying) are sometimes even greater [11]. Ethics and respect have always been priorities in our studies, without any identification of the patients and with all Informed Consent Forms duly signed.

2. Material

We selected 10 patients, 4 men and 6 women, where we noticed the difficulty in identifying them in the first observation, as they are similar except for the beard and the amount of body hair (**Figure 1**). In men, we identified more hernias and eviscerations in the abdominal part (**Figure 3**) than in women. In contrast, in women, we identified breast gigantism in 80% of them (**Figure 2**) or breast ptosis without conditions for conventional mammoplasty, when we use the Resende Technique [12] [13] to remove the excess skin, with subsequent areola grafting. Firstly, we recommend surgery for giant breasts [14], before bariatric surgery, or, in cases of abdominal gigantism (**Figure 4**), excision of the abdominal fat apron. We recommend special beds, adapted bathrooms and a lot of attention in the postoperative period. In all cases, body relief surgeries are highly complex and multidisciplinary teams should be convoked, including nutrition professionals [14].

We recommended weight loss of at least 5 kg before restrictive surgery. This is because it was noticed that, after losing 5 kg of body mass, there was a significant improvement in hypertension and a decrease in blood glucose levels. During the anamnesis, we heard the main complaints of the 10 patients:

- "I feel very tired".
- "I can't even get up".
- "I can't find suitable clothes to buy".
- "I have impediments to having sexual intercourses".
- "I can't get in cars".
- "I'm ashamed of myself".
- "I find difficulties in showering".
- "I always feel hungry".
- "Nobody wants to date me".
- "He separated from me because I'm chubby".
- "I'm rejected by society".

3. Discussion

The visual absurdity is so intriguing and complex that it will depend on the degree of experience of each medical team, involving anesthesia and the type of surgery that must be performed first. It will depend on the degree and shape of each body, in order to define whether plastic surgery first enters to perform body relief or whether bariatric surgery is performed before. We always choose the technique that is most suitable for each case of morbid obesity. Unfortunately, many patients progress to death before they can achieve their dreams. The team needs to be prepared to decide what is best for the patient.

We know that these cases concern public health and we still do not have an exclusive hospital for obese people in our country. This would be necessary because 40% of the Brazilian population is overweight, besides the fact that we have a high rate of deaths from hypertension and diabetes. Accordingly, a new

field is introduced in the surgical specialties for this discussion. We highlight the importance of the care that every morbidly obese person requires, as well as the dangers, including legal ones, that can arise. We also draw attention to the fact of how a successful surgical procedure can lead to considerable improvements in the quality of life of patients with this illness.

4. Conclusions

In all patients, males and females, we observed a considerable improvement in the metabolic status and in the appearance of body vanity. It is as if we gave back to each individual the will to live and the hope of being reintegrated into society. A male patient made the following observation: "When I was fat, I was ugly, with these skin folds, I am horrible, but only until I reach all the plastic surgeries I need!" With the involvement in studies on obesity, we can state that the patients who attended the "Obese Workshop" performed better than those who arrived at the surgery without emotional preparation and without knowing all the phases that involve the metabolic and plastic surgeries in question.

We conclude that, firstly, we should be focused on saving lives, and then try to reintroduce them into society. Moreover, we should manage to achieve a plastic surgery team prepared and trained to continue making corrections of the excess skin left by weight loss.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Milech, A., Oliveira, J. and Vencio, S. (2015-2016) Brazilian Diabetes Society Guidelines.
https://www.google.com.br/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=2ahUKEwjWxLWm2tnoAhW2GrkGHcnuAxMQFjAAegQIAhAB&url=https%3A%2F%2Fwww.diabetes.org.br%2Fprofissionais%2Fimages%2Fdocs%2FDIRETRIZES-SBD-2015-2016.pdf&usg=AOvVaw35_kNhv9afootr01FW2UjS

[2] Federal Council of Medicine (2020) FCM Details List of Comorbidities That May Lead to Indication for Bariatric Surgery.
https://portal.cfm.org.br/index.php?option=com_content&view=article&id=25939

[3] Tonatto-Filho, A.J., *et al.* (2019) Bariatric Surgery in the Brazilian Public Health System: The Good, the Bad and the Ugly, or a Long Way to Go. Yellow Sign! *ABCD, Arquivos Brasileiros de Cirurgia Digestiva*, **32**, e1470.
https://doi.org/10.1590/0102-672020190001e1470

[4] Brazil. Ministry of Health (2020) Homepage DATASUS. Hospital Information Systems—SUS (SIH/SUS).
http://tabnet.datasus.gov.br/cgi/deftohtm.exe?sih/cnv/qiuf.def

[5] Streb, A.R. and Del Duca, G.F. (2020) Obesity and Physical Exercise: Latent Challenges. *Biomotriz*, **13**, 1-5.
http://revistaeletronica.unicruz.edu.br/index.php/BIOMOTRIZ/article/viewFile/8190/pdf_147

[6] Dias, C. and Verona, A. (2019) Overweight, Obesity and Education in Brazil. *Health Magazine* (*Sta. Maria*), Brazil, **45**.

[7] De Melo, M.E. (2020) Diseases Triggered or Aggravated by Obesity. Brazilian Association for the Study of Obesity and Metabolic Syndrome—BASOMS. https://abeso.org.br/wp-content/uploads/2019/12/5521afaf13cb9.pdf

[8] Marcon, E.R., Gus, I. and Neumann, C.R. (2011) Impact of a Minimum Supervised Exercise Program on Cardiometabolic Risk in Patients with Morbid Obesity. *Arquivos Brasileiros de Endocrinologia e Metabologia*, *São Paulo*, **55**, 331-338 http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0004-27302011000500006

[9] Moraes, J. da M., Caregnato, R.C.A. and Schneider, D. da S. (2014) Quality of Life before and after Bariatric Surgery. *Acta Paulista de Enfermagem*, *São Paulo*, **27**, 157-164. http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0103-21002014000200012&lng=pt&nrm=iso

[10] Luz, D.M.D. and Encarnação, J.N. (2008) Advantages and Disadvantages of Bariatric Surgery for the Treatment of Morbid Obesity. *Brazilian Journal of Obesity*, *Nutrition and Weight Loss, São Paulo*, **2**, 376-383. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=4&ved=2ahUKEwjv8Jvc5dnoAhUQE7kGHeXhAfcQFjADegQIAhAB&url=http%3A%2F%2Fwww.rbone.com.br%2Findex.php%2Frbone%2Farticle%2Fview%2F99%2F97&usg=AOvVaw2AmpoxAiCFYbSeYUSMJ9Iv

[11] Sousa, P.M.L. (2006) Adolescent Obesity: Psychological Aspects and school Performance. Dissertation (Master)—Pedagogical Psychology Course, Faculty of Psychology and Educational Sciences, University of Coimbra, Coimbra, Portugal. http://hdl.handle.net/10400.8/218 Acessed on April 08

[12] Resende, J.H.C. (2003) Gigantomasty. In: Plastic Surgery—Fundamentals and Art—Aesthetic Surgery. MEDSI, São Paulo, vol. 1, 555-561.

[13] Resende, J.H.C. (2008) Resende's Technique for Correcting Gigantomasty—Body Relief Surgery, Treated Plastic Surgery in Obesity. Ed. Rubio, Rio de Janeiro, 357-365.

[14] Resende, J.H.C., de Moura, Á.I., Mariano, A.C.A., Silva, H.K., Silva, H.L., Campos, I.A., de Lima, L.C.F., Gabriel, M.B. and Silverio, W.O. (2019) Gigantomasty in Female Workers: "Public Health Cases". *Modern Plastic Surgery*, **9**, 1-7. https://doi.org/10.4236/mps.2019.91001

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