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First Reported Incidence of Delayed Secondary Abdominal Compartment Syndrome in a Trauma Patient with Scleroderma: A Case Report and Review of the Literature

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Abstract

Background: Scleroderma is a complex immune-mediated rheumatic disease that is characterized by fibrosis of the skin, internal organs, and vasculopathy. Extensive fibrosis, especially in the limited compartment, has been reported to induce acute compartment syndrome frequently reported involving the upper and lower extremities. **Case Presentation**: We present a rare case of a 54-year-old Caucasian female who underwent surgery for abdominal compartment syndrome in the setting of scleroderma. Upon arrival, at the hospital, the patient's health status showed signs of improvement with no indicators of abdominal compartment syndrome until the tenth hospital day. A CT scan showed a new intra-abdominal fluid collection with total lower abdominal anasarca and a stable retroperitoneal hematoma. Following emergency surgery, significant bowel edema without other intra-abdominal injuries was noted. **Conclusion:** Secondary abdominal compartment syndrome may occur in patients with scleroderma without evidence of intra-abdominal trauma or emergent abdominal surgery. Further research is warranted to investigate the relationship between scleroderma and secondary abdominal compartment syndrome.

Keywords

1. Introduction

Abdominal compartment syndrome (ACS) is an increase in intra-abdominal pressure, resulting in intra-abdominal hypertension (IAH) associated with end-organ dysfunction [1]. ACS more commonly occurs after intra-abdominal events such as emergent surgical intervention, trauma, and/or peritonitis. ACS can also occur due to extra-abdominal or systemic conditions, including reperfusion injury, sepsis, shock, excessive resuscitation, and third spacing of fluids that frequently occur among trauma and surgical patients [2]. Secondary ACS is unique in its presentation among patients without a primary intraperitoneal injury or surgical intervention [3]. ACS can lead to a myriad of multiple organ system dysfunctions including the cardiac, respiratory, gastrointestinal, renal, hepatic, ocular, and central nervous systems [1] [4]-[13]. Untreated ACS carries a mortality rate that approaches 100% [10]-[15]. With timely diagnosis and treatment, the mortality range drops between 46% and 66% [9]-[15].

Emergency laparotomy for abdominal decompression is almost always indicated in patients with ACS [1] [4]-[14]. However, the risk factors and clinical signs of ACS remain unclear [15] [16]. Recent studies have reported acute compartment syndrome of the upper and lower extremities among patients with scleroderma [17]. After a thorough review of the English-speaking literature using the PubMed and Google Scholar databases, we report what we believe to be the first known case of secondary ACS in the setting of scleroderma. We report that secondary ACS may occur in patients with scleroderma without evidence of intra-abdominal trauma or emergent abdominal surgery. Further research is warranted to explain this relationship.

2. Case Report

A 54-year-old Caucasian female with a history of scleroderma, diabetes, chronic obstructive pulmonary disease, hypertension, and prior hysterectomy who was not on steroid therapy presented at a Level I Trauma Center after a motor vehicle rollover.

Upon emergency medical service arrival, the patient was hemodynamically unstable with the following vitals: blood pressure (BP) 52/27 mmHg, heart rate (HR) 114 bpm, respiratory rate (RR) 24, oxygen saturation (SaO$_2$) 100%. A right open femur fracture was noted and splinted in the field. The patient received a liter of crystalloid (Lactated Ringers) in transport. In addition, family history and psycho-social history were reviewed. There was no relevant information contributing to this event.

Upon arrival to the emergency room (ER), the patient had the following vitals: BP 99/67 mmHg, HR 110 bpm, RR 24, SaO$_2$ 85%, and was receiving 2 liters per minute through a nasal cannula. Physical examination revealed an intact airway, severe shortness of breath, external hemorrhage due to extensive facial/scalp lacerations, and an open right femur fracture with palpable dorsalis pedis bilaterally. On examination, the abdomen was soft and non-tender to palpation with

hypoactive bowel sounds. A Glasgow Coma Scale score of 13 was noted, with no focal neuro deficits. The patient was intubated, and a mass transfusion protocol was initiated.

Chest radiograph (CXR) showed 3 to 8 rib fractures on the right side with no evidence of pneumothorax or hemothorax. A focused assessment with sonography in trauma did not demonstrate intraperitoneal, thoracic, or pericardial injury. Following stabilization, computed tomography (CT) further noted a subarachnoid hemorrhage, right lateral lower lung contusion, minimal retroperitoneal hematoma, and a comminuted segmented open fracture of the right femur. The patient was taken to the operating room (OR) for the repair of her lacerations and femur fracture and was transferred to the intensive care unit (ICU) for mechanical ventilation.

Hospital day (HD) 3, her respiratory status declined. A CXR noted bilateral infiltrates and right pleural effusion representing evolving adult respiratory distress syndrome (ARDS). Her ventilator settings were adjusted according to the ARDS-net protocol. The right pleural effusion was drained with a thoracostomy tube. Fluids were restricted, and diuretics were administered. Over the next 4 days, her daily CXR images showed gradual improvement.

HD 9, the patient became hypertensive with a BP of 180/100 mmHg. She was placed on nitroprusside (Nitropress, Nexus) drip and a Swan Ganz Catheter (SGC) was inserted.

HD 10, The SGC showed deteriorating hemodynamic parameters (Table 1). Bladder pressures were 28 - 30 mmHg. Her pulmonary status worsened requiring higher ventilator settings to maintain oxygenation. Renal functions were within normal limits. A CT scan showed a new large intra-abdominal fluid collection with total lower abdominal anasarca and a stable retroperitoneal hematoma. No free air or bowel obstruction was seen. In view of the clinical radiographic findings and history of scleroderma, the diagnosis of secondary ACS was made.

The patient was taken emergently to the OR. Upon incising the linea alba a column of fluid rose approximately 10 cm above the abdominal wall and spontaneous evisceration of the intestinal content occurred. Significant bowel edema without other intra-abdominal injuries was noted. Two and a half liters of ascetic fluid were removed. The abdomen was left open. The patient was transferred back to the ICU. The patient's hemodynamics and pulmonary mechanics improved significantly following decompressive laparotomy (Table 1).

A second look procedure was done on HD 13 which showed resolution of the bowel edema. No retroperitoneal hematoma was noted, and no evidence of a missed intra-abdominal injury was noted. The abdominal wall edema had significantly subsided, and the abdominal incision was closed by primary fascial closure. The patient was successfully weaned from the ventilator and transferred to the intermediate care unit on HD 17. She was weaned from her chest tubes within 48 hours, was tolerating a general diet, and participated in physical therapy. Two months later she was transferred to rehab. Table 2 summarizes the global fluid intake and output.

Table 1. Key hemodynamic changes and pulmonary function information before and after decompressive laparotomy.

	Day 9	Day 10	Pre-induction	Intra-operative	Immediately Post-operative	Post-operative Day 1
CO (mL/min)	6.3 - 7.4	6.9 - 7.4			6.2	5.3 - 8.4
CI (L/min/m²)	3.3 - 4	3.3 - 3.8			3.4	2.9 - 4.6
CVP (mmHg)	30 - 35	35 - 42			24	25 - 12
PCWP (mmHg)		32 - 38			20	26 - 14
PAS	51 - 69	32 - 64	63	40	50	36 - 54
PAD	31 - 44	39 - 42	47	30	34	17 - 31
MAP	102 - 115	111 - 120			85	81 - 110
SBP	150 - 178	160 - 203	226	138	147	128 - 178
DBP	80 - 100	58 - 96	108	70	65	60 - 102
PIP	36 - 39	36 - 46	48	35	36	31 - 34
PAW		29 - 33	29		28	19 - 24
PEEP	12	12			10	10
FiO2 %	45	Rapidly increased	100	100	100	Gradually decreased
Bladder Pressure		28	30	-	13	12 - 19
PAP (mmHg)			48 - 50	36	34	

CO: Cardiac Output, CI: Cardiac Index, CVP: Central Venous Pressure, PCWP: Pulmonary Capillary Wedge Pressure, PAS: Pulmonary Artery Systolic Pressure, PAD: Pulmonary Artery Diastolic Pressure, MAP: Mean Arterial Pressure, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, PIP: Peak Inspiratory Pressure, PAP: Peak Airway Pressure, PEEP: Positive End Expiratory Pressure, FiO2: Fraction of inspired Oxygen. PAP: Peak Airway Pressure.

Table 2. Patient's global fluid input/output.

Day	Total Fluid Input	Total Fluid Output
June 30	2 L crystalloid, and 8 units of Pack RBCs	Estimate blood loss: 2500
June 31	Not recorded, Pt on TPN and Levophed infusion	Not recorded
July 1	Not recorded, Pt on TPN	Not recorded
July 2	Not recorded, Pt on TPN	Not recorded
July 3	1560 cc crystalloid	3000 cm³ (Urine, chest tubes)
July 4	1560 cc (TEN = 1200 cm³, Electrolyte replacement = 360 cm³ crystalloid)	3960 cm³
July 5	2016 cc (TEN = 1800 cm³, Electrolyte replacement = 216 cm³ crystalloid)	3627 cm³
July 6	1788 cc (TEN = 1500 cm³, Electrolyte replacement = 288 cm³ crystalloid)	3025 cm³
July 7	2316 cc (TEN = 2100 cm³, Electrolyte's replacement = 216 cm³ crystalloid)	4505 cm³

Continued

July 8	2100 cc (TEN = 2100 cm³, Electrolyte replacement not recorded)	Not recorded
July 9	2268 (TEN = 2100 cm³, Electrolyte replacement = 168 cm³ crystalloid)	2875 cm³
Total	19608 cm³	23492 cm³

TPN: Total Parenteral Nutrition, TEN: Total Enteral Nutrition.

This case report was reviewed by the Institutional Review Board at The University of Texas Health Science Center at Tyler and a determination letter of "Not Human Subjects Research" was given. Patient consent was obtained at the time of treatment.

3. Discussion

Systemic sclerosis is an ongoing and escalating complex immune-mediated rheumatic disease that is characterized by fibrosis of the skin, internal organs, and associated vasculopathy. Compared to any other rheumatological disease systemic sclerosis has higher operative mortality, despite evidence of improved surgical outcomes among patients with the cutaneous subtype [17] [18] [19]. Associated clinical findings include pulmonary arterial hypertension, digital ulceration, and gastroesophageal reflux [17] [18] [19]. Scleroderma is characterized by early microvascular changes leading to activation of mechanisms promoting endothelial cell transition into myofibroblast. The pathogenesis of systemic sclerosis includes a complex autoimmune response involving innate and adaptive immunity with specific/functional auto-antibody production. Progenitor circulating cells (monocytes and fibrocytes) together with growth factors and cytokines participate in disease diffusion and evolution [18]. Progressive fibrosis and ischemia that includes skin and visceral organs result in irreversible damage. Progressive fibrosis of the connective and interstitial tissue has been reported to be a predisposing factor in the development of compartment syndrome found in the hand [17].

3.1. Abdominal Compartment Syndrome

ACS is determined based on its underlying etiology [1] [20]. ACS is defined as a sustained intra-abdominal pressure (IAP) > 20 mmHg that is associated with new organ dysfunction/failure [1]. ACS clinical manifestations and pathophysiology resulting from IAH secondary to aortic aneurysm surgery were described by Kron *et al.* in 1984 [21]. These findings were considered new despite being described over 100 years earlier by Emerson, who developed the experimental foundations of current research on IAP in the 20th century [22] [23].

Cheatham *et al.* listed the signs of ACS as the following: abdominal distension, increased intra-abdominal pressure, oliguria refractory to volume administration, increased peak inspiratory pressure, hypercarbia, hypoxia refractory to in-

creasing FiO2 and PEEP, refractory metabolic acidosis, and increased intracranial pressure [11]. Once developed, ACS can lead to multiorgan disfunction [1] [4]-[21]. ACS results in a drop in cardiac output secondary to the decreased venous return through the inferior vena cava and portal vein [1] [4]-[16] [21]. Respiratory dysfunction is a common sequelae resulting from an elevated diaphragm that compromises the total lung capacity, functional residual capacity, and residual volume [1] [4]-[10] [12] [13] [14] [16] [21]. Early diagnosis of ACS and timely appropriate intervention are required for optimal outcome [1] [4]-[16] [21].

The exact incidence of ACS has not yet been established, [15] but specific patient risk groups have been described. Reported risk factors for ACS include excessive intravascular fluid resuscitation, abdominal surgeries, acute pancreatitis, major trauma, sigmoid volvulus, liver dysfunction, sepsis, obesity, and age [1] [15] [16] [21] [22] [23] [24].

Primary ACS is associated with an injury or disease in the abdomen or pelvis that requires some form of intervention, most commonly, direct surgical repair or control [1] [20] [25]. Patients with primary ACS typically have significant intraperitoneal or retroperitoneal bleeding, solid organ injury, bowel ischemia, major intraabdominal organ transplantation (more commonly the liver), severe hemorrhage (retroperitoneal, intraperitoneal, and pelvic), severe pancreatitis, and have undergone damage control surgery, for example, intraabdominal packing for severe hemorrhage [1] [20] [25]. Repair of the ruptured abdominal aortic aneurysm has also been associated with primary ACS with reported mortality approaching 47% [25] [26]. The incidence of primary ACS has been estimated at 6% [27], with a reported mortality ranging from 40% to 100% [28] [29] [30].

Secondary ACS occurs in patients without an intra-abdominal injury or severe pelvic injury that requires intervention [1] [20]. Interventions for patients include those who undergo initial aggressive fluid resuscitation (3 L or greater), large full-thickness burns, the morbidly obese, those who are pregnant, pediatric trauma patients and those who have undergone intraabdominal packing and/or primary fascial closure after initial surgical exploration [1] [20] [25]-[31]. Secondary ACS after treatment of hemorrhagic shock, without evidence of intra-abdominal injury or complicated traumatic lower extremity vascular injuries, has also been described in the literature [32] [33] [34]. Balogh *et al.* described how aggressive crystalloid fluid resuscitation is a major contributing factor to secondary ACS [35] [36]. Hobson *et al.* reported the occurrence of ACS within 24 hours among burn patients who had received an average of 237 mL/kg of intravenous fluid over a 12-hour period [37]. The true incidence of secondary ACS is unknown. In a retrospective review of a level, I trauma center Maxwell *et al.* noted that the incidence of secondary ACS among ICU admissions was 0.5% and concluded that "it probably occurs more frequently than is currently appreciated" [33]. The mortality of secondary ACS is also not well defined. In an analysis of major trauma victims who developed secondary ACS during standardized shock resuscitation, Balogh *et al.* found a 54% mortality rate and reported

that patients who died failed to respond to decompression with increased cardiac index and did not maintain decreased bladder pressure [35]. The timing of the occurrence of secondary ACS in high-risk groups is unknown, however, it more commonly occurs within 12 to 24 hours after initial volume resuscitation [37] [38].

3.2. Abdominal Wall Compliance

Understanding the pathophysiology, etiology, prognosis, and treatment of elevated IAP in trauma, surgical, and medical patients is essential to better diagnose and treat IAH and ACS. A recent two-part analysis by Malbrain *et al.* has helped define the significance of intra-abdominal volume (IAV) and the relationship between IAV, IAP and abdominal wall compliance (AWC), reporting that due to complex biomechanical effects patients with decreased AWC are at significant risk for IAH and ACS [39] [40]. In a systematic review and meta-analysis of risk factors for IAH and ACS Holodinsky *et al.* identified 25 unique evidence-based risk factors for IAH and sixteen for ACS [41]. Although several of these risk factors appeared to transcend across patient populations, for example, large-volume crystalloid resuscitation and the presence of shock/hypotension, many were specific to the type of patient population under study. The more common clinical settings that impact AWC have been recognized as risk factors for ACS, including, excessive intravascular fluid resuscitation, abdominal surgeries, acute pancreatitis, major trauma, burns, sigmoid volvulus, liver dysfunction, sepsis, obesity, pediatric abdominal wall congenital anomalies [1] [15] [16] [21] [22] [23] [24] [33] [34] [35] [36] [37] [39] [40] [41].

3.3. Abdominal Compartment Syndrome in the Setting of Scleroderma

Due to the unique physiology of systemic scleroderma, we suggest that practitioners apply an approach to scleroderma patients that is similar to circumferential burn victims and consider how this will affect AWC and increase the risk for ACS. With advanced-stage disease leading to fibrosis of internal tissue, the clinical presentation and the associated abdominal distention commonly seen in a patient with secondary ACS may be lacking. The elasticity of the abdominal tissues and the contents within the peritoneal cavity determine the pressure within the abdomen [20]. In this case, elevated pressure was noted without abdominal distention due to the inherent, limited elasticity of the abdominal wall. We propose that a patient presenting with systemic scleroderma is comparable to a burn victim patient and special consideration of altered clinical presentations is warranted.

4. Conclusions

In this case presentation, we diagnosed delayed secondary ACS in a trauma patient without evidence of intra-abdominal trauma. We observed the development of multiple organ failure with deterioration of respiratory functions, BP,

diminished urine output, elevated central venous pressure, and elevated urinary bladder pressure. This patient had typical risk factors including large volume resuscitation associated with IAH and secondary ACS but did not exhibit clinical abdominal distention.

Secondary ACS may occur in patients with scleroderma without evidence of intra-abdominal trauma or emergent abdominal surgery who receive large volume resuscitation with minimal or no evidence of typical abdominal clinical findings associated with ACS. The authors have conducted an extensive search of the English-speaking literature using PubMed and Google Scholar and have failed to find a single case report of scleroderma cited as a case or predisposing risk factor associated with secondary ACS. We believe this may be the first recorded case in the literature and suggest this report may provide novel clinical information that will assist surgeons in the care of the poly-trauma scleroderma patient.

Consent

Patient consent was obtained at the time of treatment.

Conflicts of Interest

The authors declare no conflicts of interest.

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List of Abbreviations

ACS: Abdominal Compartment Syndrome

ARDS: adult respiratory distress syndrome

AWC: abdominal wall compliance

BP: blood pressure

CT: computed tomography

CXR: chest radiograph

ER: emergency room

HD: hospital day

HR: heart rate

IAH: Intra-Abdominal Hypertension

IAP: intra-abdominal pressure

IAV: intra-abdominal volume

ICU: intensive care unit

OR: operating room

RR: respiratory rate

SaO_2: oxygen saturation

SGC: Swan Ganz Catheter