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Pseudocholinesterase Deficiency: A Case Report and Literature Review

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ABSTRACT

A 72-year-old male underwent neck dissection and parotidectomy with facial nerve preservation. Endotracheal intubation was facilitated with succinylcholine. Prolonged muscle paralysis which was first detected after failure to stimulate the facial nerve with electrocautery, lasted five hours. Laboratory tests indicated pseudocholinesterase (PChE) deficiency. A thyroidectomy one month later was performed uneventfully using rocuronium as a muscle relaxant. Literature review revealed a total of 40 PChE deficiency cases being reported since 1956.

Keywords: Prolonged Paralysis; Succinylcholine; Pseudocholinesterase Deficiency; Dibucaine Number; Case Review

1. Introduction

Pseudocholinesterase (PChE), also referred to as butyrylcholinesterase, serum cholinesterase, plasma cholinesterase, and false cholinesterase, is a liver-derived plasma protein capable of hydrolyzing esters including muscle relaxants (*i.e.* succinylcholine, atracurium, mivacurium) and ester type local anesthetics (*i.e.* procaine, chloroprocaine, tetracaine, cocaine) with a serum half-life of eight to 12 days. A deficiency of this enzyme, either inherited and/or acquired, can lead to significantly pro-longed activity of these medications.

We encountered a patient with prolonged paralysis after exposure to an intubation dose of succinylcholine for parotid gland resection. Succinylcholine was indicated to facilitate an endotracheal insertion with a laryngoscope with the expectation that muscle tone should quickly be regained for the surgeons to preserve the facial nerve during neck dissection. The importance of early recognition of prolonged paralysis is stressed and its management, as well as a literature review of reported PChE cases, is discussed.

2. Case Report

A 72-year-old patient presented for resection of a left parotid gland lesion. His past medical history was notable for benign prostatic hypertrophy, acid reflux, and basal cell cancer of the face. There were no prior surgeries or anesthetics. Family history was noncontributory. He was married with four children and six grandchildren. He denied use of tobacco, alcohol, or illicit drugs. There were no allergies at that time. His medications included only 0.5 mg dutasteride daily. Present illness at the time of surgery included a painful skin lesion overlying the angle of the left aspect of the mandible with an accompanied parotid mass. He was scheduled for an excision of the left facial skin lesion, left partial parotidectomy with facial nerve dissection, and left lymph node biopsy. His height and weight were 188 cm and 110 kg, respectively. On physical exam, his heart sounds were regular without murmurs, rubs, or gallops; lungs were clear bilaterally; abdomen was soft and non-tender; no lower extremity edema.

After premedication with 2 mg midazolam and 100 mcg fentanyl intravenously (iv) and preoxygenation, general anesthesia was induced with 200 mg propofol with 100 mg lidocaine iv. Succinylcholine 200 mg iv was administered to facilitate the placement of an endotracheal tube. General anesthesia was maintained with desflurane and 50% oxygen. Initial vital signs were stable with an SaO_2 of 100%, heart rate at 85/min and regular, blood pressure of 145/75 mmHg, and a temperature of 36.3˚C. Volume controlled ventilation was used with a tidal volume of 750 mL, a respiratory rate of 10/min, and peak inspiratory pressure of 20 cm H_2O. The skin lesion, actinic keratosis, was excised using an elliptical incision and the tissue was submitted to pathology. The anterior aspect of the sternocleidomastoid muscle was mobilized and the anterior aspect of the external auditory canal was identified along with the posterior belly of the digastrics muscle, revealing the left facial nerve structure. At this point, approximately one hour into the procedure, the

surgeon was unable to create muscle contractions by direct stimulation of the facial nerve with an electrocautery. The anesthesiologist confirmed muscle paralysis with an ulnar nerve stimulator via a train-of-four (TOF) technique without any twitch.

Given the use of succinylcholine and the patient's otherwise unremarkable preoperative condition, PChE deficiency was suspected. A blood sample was submitted to measure serum PChE level. The surgical team continued to carefully identify and protect the facial nerve, and the lateral aspect of the parotid gland was removed and submitted to pathology. The wound was irrigated and closed in layers. The patient's paralysis continued at the end of the two-hour and eleven-minute operation. He was transported to the post-anesthesia care unit with a propofol infusion (100 mcg/kg/min) under mechanical ventilation. At five hours after induction of general anesthesia, an adequate TOF stimulation was obtained. No fresh frozen plasma was used. The propofol infusion was then terminated and the endotracheal tube was removed after the patient was able to follow commands, sustain a head lift, and register adequate tidal volumes.

The serum PChE level submitted during surgery was 1100 IU/L (normal 3300 - 7600 IU/L), supporting the diagnosis of PChE deficiency. The patient was counseled in regards to the function of PChE and what it means to be deficient. The patient was instructed to wear a Medic Alert bracelet and to obtain further testing for him and his family.

He was discharged home on post-operative day one, and returned approximately one month later for an uneventful total thyroidectomy using rocuronium instead of succinylcholine for muscle paralysis. Before the second surgery, a repeated plasma PChE test revealed a level of 1100 IU/L, effectively confirming his PChE deficiency. He declined additional testing, including dibucaine number and genetic testing.

3. Discussion

PChE deficiency is one of many causes of prolonged paralysis during general anesthesia. In the current case, failure to induce muscle contraction with direct electrostimulation of the facial nerve led to the identification of the prolonged paralysis. The differential diagnoses for prolonged paralysis can be seen in **Table 1**. While most are common, treatable, and avoidable causes of pro-longed muscle blockade, PChE deficiency after the exposure of potential offending muscle relaxants should also be suspected.

PChE deficiency can be either inherited or acquired. The inherited etiologies of PChE deficiency is attributed to genetic mutations at a single autosomal location on the long arm of chromosome 3. The genetics of PChE deficiency were first documented in the 1950's; five autosomal alleles on locus E_1 are now known to be responsible for synthesizing PChE. The five alleles are: usual (E_1^u), atypical (E_1^a), fluoride resistant (E_1^f), K variant (E_1^k) and silent (E_1^s). The usual allele codes for a normal PChE quantity and quality while the others to a changeable degree do not. These five alleles give rise to 15 phenotypes that produce varying degrees of PChE deficiency. To further illustrate the commonality of this disorder, it has been reported that 96% of the population is homozygous for the usual enzyme while 4% posses at least one abnormal allele. This translates to 1:3000 in the general population possessing two abnormal alleles and 1:2400 belonging to those with a clinically significant prolonged paralysis after exposure to PChE dependent muscle relaxants [1]. On the other hand, there are many acquired etiologies for having low PChE levels (**Table 1**). However, as common as these modifiers may be, more often a patient possessing any of these risk factors will have a normal response to paralytics. Imerman *et al*. points out that a reduction to 5% of normal PChE activity would be required to increase paralysis by one hour after administration of succinylcholine [2]. Furthermore, Foldes and colleagues documented that liver disease alone, resulting in PChE levels 20% of normal, merely increases neuromuscular blockade threefold [3]. It is unlikely that any of the above acquired etiologies could produce a clinically significant prolongation of paralysis, illustrating that a genetic basis of PChE deficiency should be considered most likely.

Table 1. Causes of pseudocholinesterase deficiency [2,7-11, 14,17,18].

Inherited	Aquired	Iatragenic
Atypical	Liver failure	Cyclophophamide
Gene alleles	Renal failure	Diethylstilbestrol
	Extremes of age	MAO inhibitors
	Pregnancy	Oral contraceptives
	HELLP	Organophosphates
	Malnutrition	Alkylating agents
	Hypothyroidism	Esmolol
	Hypothermia	Clucocorticoids
	Malignancy	Metaclopramide
	Burns	Pancuronium
	Hypoalbuminemia	Phenelzine

To verify a suspected PChE deficiency, one should first obtain a serum PChE level, the normal reference range being 3300 - 7600 IU/L. Of note, a repeated test approximately one week or more after exposure to the offending agents is warranted for confirmation, because PChE level can be artificially decreased initially [4]. Our patient's PChE level immediately after the exposure of succinylcholine was 1080 IU/L and was confirmed low (1100 IU/L) one month later. Also, a dibucaine number (DN) helps to decipher whether the PChE deficiency is genetic in origin, and can imply hetero- or homozygosity. Dibucaine is a local anesthetic which inhibits PChE activity when mixed with a blood sample. The percentage of PChE inhibited will yield a DN; 80 - 100 indicates normal PChE function, 40 - 70 indicates heterozygous, and less than 30 indicates homozygous for atypical genotype. From a practical standpoint, PChE level and DN have been shown to be sufficient in determining a genetic origin of PChE deficiency. However, molecular testing is also available, including gel-electrophoresis and immunoelectrophoresis, and this method has been shown to improve biological diagnosis in one-third of patients; however, the clinical implications are lacking [5].

The management of patients with prolonged paralysis due to PChE deficiency is mainly conservative. Patients are maintained with mechanical ventilation under sedation while the effect of the paralytic is cleared [3]. Theoretically, if urgent reversal of paralysis becomes necessary, for example to assess neurologic function, fresh frozen plasma (FFP) or packed red blood cells can be utilized to provide exogenous PChE [6,7]. Of note, anticholinesterase agents (*i.e.* neostigmine and physostigmine) inhibit PChE activity and can lead to paradoxical worsening of paralysis [7]. If PChE deficiency is suspected, the patient and family should be counseled with recommendation of further diagnostic tests and a Medic Alert bracelet should be issued [4].

In a literature review searching for the key words "pseudocholinesterase deficiency" in PubMed (http://www.ncbi.nlm.nih.gov/pubmed/:lastaccessedMay 5, 2012), a total of 39 articles featuring 40 case reports on PChE deficiency were found and are summarized in **Tables 2** and **3** [1-39]. The earliest description of PChE deficiency was reported in 1956 [3] and the latest case report was published in 2011 [20]. The length of paralysis ranged from 50 minutes [28] to more than ten hours [34]. Only one case reported the use of FFP to facilitate recovery from paralysis [6]. No mortality or lasting complications were reported. PChE levels were checked in all but six cases, ranging from 5 - 2765 IU/L. DN was checked in only 12 of the 40 cases ranging from 19 - 81 [1,4,8,9,12,14,20,24,26,33,38]. Two cases reported a "normal" DN [8,9] and one with a "very low" value [1]. In terms of etiology, 20 cases were considered genetic [1, 4,7,12,14,19,20,23,24,26,27,29,34,35,37,38]. Six were due to acquired causes such as liver disease [28], pregnancy [33], HELLP syndrome [17], malnutrition [8], or from administration of cyclophosphamide [10] or diethylstilbestrol [9]. The remaining 15 cases were from an unknown etiology [2,6,13,21,22,25,27,30,32,36,39]. Medical staff in only 18 cases inquired about family history; eight reported a family member with a similar experience [1,4,7, 29,37]. None of these case reports performed genetic testing on family members.

Table 2. Summary of the reported cases of pseudocholinesterase deficiency.

ID	Year	Age[a] /Sex	Procedure	Agent	Additional agent	Clinical sign	Ventilation support (min)	FFP
1	1960 [29]	1.25/F	Lumbar meningocele	SCh	None	PA	190	No
2	1962 [37]	7/F	Tonsillectomy	SCh	None	PA	75	No
3	1962 [37]	6/M	Tonsillectomy	SCh	None	PA	150	No
4	1962 [37]	4/F	Tonsillectomy	SCh	None	PA	270	No
5	1965 [28]	73/M	Laparotomy	SCh	None	PA	50	No
6	1966 [6]	24/M	Scapula tumor biopsy	SCh	None	PA	210	Yes
7	1966 [6]	17/M	Laminectomy	SCh	None	PA	240	No
8	1967 [35]	23/W	Bronchoscopy	SCh	None	PA	180	No
9	1968 [36]	41/M	Gastroscopy	SCh	None	PA	100	No
10	1973 [27]	47/F	Cholecystectomy	SCh	None	PA	630	No

Continued

11	1977 [1]	21/F	Laparoscopy	SCh	None	PP	225	No
12	1977 [1]	42/F	Dilatation and curettage	SCh	None	PP	210	No
13	1978 [9]	78/M	TURP	SCh	None	Hypoventilation	210	No
14	1982 [26]	31/F	Laparotomy	SCh	None	PP	180	No
15	1982 [39]	40/F	Tubal ligation	SCh	None	PP	345	No
16	1987 [25]	26/M	Electro convulsive therapy	None	Atrac	n/a	n/a	No
17	1987 [25]	80/M	Electro convulsive therapy	None	Atrac	n/a	n/a	No
18	1987 [25]	30/F	Electro convulsive therapy	None	Atrac	n/a	n/a	No
19	1989 [38]	9/M	Tonsillectomy	SCh	None	PP	210	No
20	1999 [10]	2.5/F	Central line insertion	SCh	None	PP	90	No
21	1999 [33]	28/F	Dilatation and curettage	SCh	Mivac	PP	140	No
22	1999 [34]	4 wks/M	Pyloromyotomy	SCh	Atrac	PA	1140	No
23	2001 [2]	41/M	Renal transplantation	SCh	Atrac	PP	300	No
24	2002 [31]	19/F	Breast lumpectomy	Mivac	None	PP	360	No
25	2003 [4]	19/F	Dentoalveolar resection	SCh	Mivac	PP	300	No
26	2004 [17]	27/F	Cesarean section	SCh	None	PP	180	No
27	2004 [8]	71/M	Esophagoscopy	SCh	Mivac	PP	270	No
28	2005 [13]	17/F	Removal of hardware	SCh	Mivac	PA	270	No
29	2005 [24]	29/M	Electro convulsive therapy	SCh	None	PP	Unknown	No
30	2007 [14]	39/M	Digit amputation	SCh	None	PP	150	No
31	2007 [12]	67/M	Electro convulsive therapy	SCh	None	PP	150	No
32	2007 [7]	35/F	Endotracheal intubation	None[b]	None	None	Extubated on day 13	No
33	2009 [22]	80/M	Ventro-peritoneal shunt	None	None	n/a	n/a	No
34	2009 [23]	30/F	Cesarean section	SCh	None	PP	360	No
35	2010 [30]	70/M	Electro convulsive therapy	SCh	None	PP	240	No
36	2010 [32]	26/F	Cesarean section	SCh	None	PA	540	No
37	2011 [19]	26/M	Nasal septum repair	Mivac	None	PP	420	No
38	2011 [19]	7/M	Tonsillectomy	Mivac	None	PP	240	No
39	2011 [20]	60/M	Electro convulsive therapy	SCh	None	PP	120	No
40	2011 [21]	26/M	Electro convulsive therapy	None	Roc	n/a	n/a	No
41	**Present case**	72/M	Parotidectomy	SCh	None	PP	300	No

Note: [a]Year-old (unless otherwise indicated); [b]Known family history of PChE-D: No paralytic was used; **Abbreviations**: F, female; M, male; wks, weeks old; TURP, transurethral resection of the prostate; SCh, succinylcholine; Mivac, mivacurium; Atrac, atracurium; Roc, rocuronium; PA, prolonged apnea; PP, prolonged paralysis; FFP, fresh frozen plasma.

Table 3. Summary of the reported cases of pseudocholinesterase deficiency (continued).

ID	Initial PChE (IU/L)	Dibucaine number	Cause	Family history of PChE-D	Follow-up
1	25[c]	n/a	Genetic	Yes	None
2	60.5[c]	n/a	Genetic	Yes	Family testing with PChE-D
3	68.5[c]	n/a	Genetic	Yes	Family testing with PChE-D
4	22[c]	n/a	Genetic	Yes	Family testing with PChE-D
5	5[d]	n/a	Amoebic liver disease	No	None
6	2[e]	n/a	Unknown	No	None
7	1[e]	n/a	Unknown	Unknown	None
8	12.3[c]	n/a	Genetic	No	Family testing with PChE-D
9	n/a	n/a	Unknown	Unknown	None
10	0.22-0.84 pH/hr	n/a	Unknown	Unknown	None
11	800	27	AH	Yes [f]	None
12	"Normal"	"Very low"	AH	Yes[g]	None
13	1300	"Normal"	Diethylstilbestrol	Unknown	PChE normalized after d/c diethylstilbestrol
14	40	32	Genetic	Unknown	PChE 84, dibucaine 27 (6 weeks postpartum)
15	538	n/a	Unknown	Unknown	None
16	5000	n/a	Unknown	Unknown	None
17	n/a	n/a	Unknown	Unknown	None
18	n/a	n/a	Unknown	Unknown	None
19	389	20	Genetic	Unknown	None
20	400	n/a	Cyclophosphamide	Unknown	PChE 4533 IU/L after d/c cyclophosphamide
21	600	78	Pregnancy vs. OHSS	Unknown	PChE 7600 IU/L (4 months post-op)
22	<200	n/a	Genetic	No	Family testing with PChE-D
23	1660	n/a	Unknown	Unknown	None
24	4794	n/a	Unknown	No	None
25	799	48	AH	Yes[h]	None
26	2500	n/a	Pregnancy vs. HELLP	No	PChE 5200 IU/L (16 days post-op)
27	838	"Normal"	Malnutrition	Unknown	None
28	n/a	n/a	Unknown	Unknown	None
29	2896	81	Genetic	Unknown	See note[i]
30	2765	23	AH	No	None
31	375	19	AH	Unknown	None
32	n/a	n/a	Genetic	Yes	None
33	5	n/a	Unknown	Unknown	None
34	1123	n/a	Genetic	Unknown	PChE 1479 IU/L (3 years post-op)
35	n/a	n/a	Unknown	Unknown	None
36	<100	n/a	Unknown	Unknown	None
37	3393	n/a	Genetic	No	None
38	2558	n/a	Genetic	No	None
39	1125	23	Genetic	No	None
40	2942	n/a	Unknown	Unknown	None
41	1080	n/a	Genetic	Unknown	PChE 1100 IU/L (4 weeks post-op)

Note: [c]μL CO_2/mL; [d]μmols/mL/hr; [e]mM acetylcholine hydrolyzed/hr/L; [f]Father with heterozygosity: Mother with borderline heterozygosity; [g]Children with heterozygosity; [h]Clinical history of mother; [i]A reduced dosage (50% - 75%) of succinylcholine was administered to provide paralysis and allowed faster recovery; **Abbreviations:** PChE, pseudocholinesterase; PChE-D, pseudocholinesterase deficiency; AH, atypical PChE homozygosity; OHSS, ovarian hyperstimulation syndrome; HELLP, hemolysis, elevated liver enzymes, low platelets syndrome.

4. Conclusion

PChE deficiency is a rare cause of prolonged paralysis, regarding which any clinician utilizing paralytics should be educated. When suspecting the diagnosis, supportive care should be given to ensure patient safety, a PChE level and DN should be obtained, and genetic testing should be offered if indicated. Genetic counseling should be offered not only to the patient but also to members of family.

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