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CASE REPORT

Cesarean Section in a Parturient with Pseudocholinesterase Deficiency Suspected by Preoperative Interview and Immune Thrombocytopenic Purpura

A Jlali[1,2*], W Ben Moussa[1], M Gara[1,2], O Ammar[2], M Mehdi[2] and L Grati[1]

[1]*Department of Anesthesia and Intensive Care "B", University of Monastir, Tunisia*

[2]*Research Unit of Genetics, Genotoxicity and Childhood Illness UR12ES10, University of Monastir, Tunisia*

***Corresponding author:** *Dr. Ali Jlali, Department of Anesthesia and Intensive Care "B", Maternity and Neonatology Center, Monastir 5000, Tunisia*

Abstract

The preoperative evaluation of a patient for anesthesia is the cornerstone for perioperative management plans. The following case report details the importance of medical interview in a parturient with suspected pseudocholinesterase deficiency and documented immune thrombocytopenic purpura, and shows that anesthetic complications can be avoided by a rigorous preoperative anamnesis.

Keywords

Immune thrombocytopenia, Pseudocholinesterase deficiency, Cesarean section

Introduction

Pseudocholinesterase (PChE) deficiency is an inherited or acquired condition in which the metabolism of succinylcholine or mivacurium is potentially impaired, and the most described consequence of the disease is prolonged paralysis and apnea. If PChE deficiency is combined with factors endangering the loco-regional anesthesia, like thrombocytopenia, the management of such a patient becomes a challenge.

Patient consent was obtained for publication of this case report.

Case Report

A 39-year-old, 162 cm, 70 Kg, second gravida parturient scheduled for elective cesarean section, was referred to our department at 32 weeks of gestation for preanesthetic evaluation. She had an already immune thrombocytopenic purpura (ITP) first diagnosed at the age of 18 years following a prior history of gingivorrhagia. She remained clinically stable thereafter, and do not required any treatment. Furthermore, in the medical interview, she reported a long "wake-up period" in her first cesarean section, performed three years ago under general anesthesia for fetal distress. No anesthesia records from this delivery were available. We presumed that she had PChE deficiency. A blood sample was drawn and sent off to measure serum PChE activity. The PChE level was 1995 IU/L with the normal range being 5200-12800 IU/L. Blood analysis revealed thrombocytopenia at 41 × 10^9/L. By reading his ancient blood analysis, we find that the platelet count in between pregnancies was about 76 × 10^3/mm^3.

Treatment was initiated in preparation for delivery, with a starting dose of oral prednisone of 20 mg daily. After 14 days of treatment, the platelet number increases to 73 × 10^3/mm^3. Therefore, the corticosteroid dose was decreased to 15 mg/day during 7 days, then adjusted at 10 mg/day and maintained until delivery. Repeated platelet count every week remained stable with values oscillating between 65 × 10^3/mm^3 and 86 × 10^3/mm^3. Cesarean section was performed under spinal anesthesia at 37 weeks of gestation after a platelet count of 82 × 10^3/mm^3 with a mean platelet volume (MPV) at 11.7 fL. In the operating room, basic monitoring with cardioscope, pulse oximeter, and noninvasive

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blood pressure was carried out, and showed normal sinus rhythm of 84 bpm, oxygen saturation (SpO_2) of 98%, and blood pressure of 120/70 mmHg.

Spinal anesthesia was performed at first attempt with a 25 G atraumatic pencil-point spinal needle at the lumber 3-4 interspaces in the sitting position. After backflow of clear fluid, 10 mg of hyperbaric bupivacaine 0.5%, 2.5 µg sufentanil and 100 µg morphine were administrated. A T_4 sensory block to light touch was achieved before surgery. No platelet infusion was given and there were no intraoperatively complications. A corticosteroid cover (hydrocortisone 100 mg IV) was given to prevent adrenal suppression. Neonatal thrombocytopenia secondary to maternal ITP did not occurs. The post-operative recovery period was uneventful, and she was discharged home on postoperative day three. Prednisone was stopped after 7 days of digression. Three months after her cesarean section, the patient returned for a dibucaine inhibition test and a second PChE assay. The PChE value was 2250 IU/L and the dibucaine inhibition test result was 24% (reference range: 81.6-88.3). Moreover, control platelet count on the occasion of this visit was $80 \times 10^3/mm^3$.

Discussion

We report the perioperative management of a cesarean section in a parturient with mild ITP and presumed PChE deficiency. We could find no data about these combined coexisting diseases available in the literature.

Pseudocholinesterase is a glycoprotein enzyme that hydrolyses specifically succinylcholine and mivacurium [1]. Succinylcholine remains the drug of choice commonly used in crash induction. In patients with genotypically normal PChE, time to full 90% recovery from succinylcholine-induced muscle paralysis is about 10 to 12 minutes after a standard 1 mg/kg dose [2].

Once abnormally prolonged neuromuscular-blockade and apnea occur at the time of awakening from general anesthesia, many causes like electrolyte imbalances (hypokalemia, hypocalcemia, hypermagnesemia) and hypothermia should be initially reviewed [3]. If succinylcholine (or mivacurium) was used, PChE deficiency should be suspected, and at a minimum, serum PChE level and dibucaine inhibition test should be drawn 24 to 48 hours later [2,3].

PChE deficiency is divided into inherited and acquired causes. The acquired conditions include advanced age, pregnancy, liver disease, renal disease, malnutrition, hypothyroidism and malignancy. Some medications also affect PChE activity, such as oral contraceptives, monoamine oxidase inhibitors, aspirine, clindamycine and metoclopramide [4]. Unexpectedly, anticholinesterase agents (neostigmine and physostigmine), habitually used to reverse the neuromuscular block, inhibit PChE activity, and can lead to paradoxical worsening of paralysis [5]. Hence, neuromuscular monitoring should be applied even when using succinylcholine [4,6].

It has been shown that pregnancy leads to a reduction in the PChE level. The physiological decrease is usually less than 25% and has a little effect upon the duration of action of succinylcholine in the normal parturient [7]. It starts in the tenth week and could take six weeks postpartum to normalize. Thus, PChE levels should be repeated two months or more after delivery to confirm PChE deficiency [7].

The dibucaine inhibition test (or dibucaine number) represents the percentage of the enzyme (PChE) inhibited by the local anesthetic dibucaine. It is the simplest and most widely used method for cholinesterase phenotyping. A dibucaine number above 80% is labeled as normal, a number between 30 and 70% is labeled as intermediate phenotype (heterozygotes), and a number less than 30% is labeled as atypical phenotype (homozygotes) [8].

In the presented case report, serum PChE activity which was studied before birth giving found 1995 IU/L (normal 5200-12800 IU/L), and after three months, control level increased to 2250 IU/L, thereby confirming the diagnosis of PChE deficiency. The dibucaine inhibition test result was 24%, suggesting an homozygote form. Spontaneous recovery from a typical 1 mg/kg dose of succinylcholine in such a case may be prolonged as much as 6-8 hours [8].

There is no cure for PChE deficiency, and the best and safest management of patients with incidentally prolonged succinylcholine-induced paralysis is to kept the patient sedated and mechanically ventilated until a spontaneous full recovery of neuromuscular function [4,9]. In cases for which general anesthesia is required, succinylcholine must be avoided, and rocuronium may be the best alternative for crash induction [10].

On the other hand, Immune thrombocytopenic purpura (ITP) is a rare disorder accounting for less than 5% of all pregnant thrombocytopenia [11]. Prognosis of ITP in pregnancy is unpredictable, and platelet counts can remain stable or worsen as pregnancy progresses [11]. Thus, the goal of therapy for ITP in pregnant women is to maintain a platelet count of at least $50 \times 10^9/L$ beyond the mid-to late third trimester. Whatever, low platelet count is not always associated with bleeding, and other platelet indices, such as mean platelet volume (MPV) must be considered when predicting haemostatic potential in thrombocytopenic states [12]. Suggested treatment protocol involves steroids as first-line treatment. Intravenous immunoglobulin should be reserved for refractory cases, and platelet infusion must be indicated when there is not enough time for other treatments to take effect [13]. The choice of prednisone alone in our case was based on the good initial response and the possibility of delaying delivery. Fortunately, this

treatment improved the platelet count. Thus, it allowed to perform a spinal anesthesia which is much more advantageous than general anesthesia in this particular case.

Conclusion

Parturients with ITP and PChE deficiency are challenging cases for anesthetists, as they may have complications that influence the selection of both regional and general anesthesia.

The present case shows that anesthetic complications may be avoided by a proper preoperative interview.

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