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Original Research Paper

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USAGE OF FRESH FROZEN PLASMA TO REVERSE ACQUIRED PSEUDOCHOLINESTERASE DEFICIENCY – A CASE REPORT

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ABSTRACT Pseudocholinesterase deficiency is very rare to find in routine practice , although acquired deficiency may be found in some patients. 25 year old primigravida with 36 weeks of gestation with impending Eclampsia with suspected HELLP syndrome was posted for emergency LSCS i/v/o non reassuring non stress test. Patient was induced under GA with all standard ASA monitoring under Rapid sequence induction with inj. Succinyl choline 75mg . Patient did not have any signs of respiratory efforts even after 2.5 hours. We suspected acquired pseudocholinesterase deficiency and decided to give a trial of reversing with fresh frozen plasma as it contains pseudocholinesterase enzyme. As soon as FFP was given patient had spontaneous respiratory efforts and was taking adequate tidal volume and hence extubated uneventfully.

KEYWORDS : Pseudocholinesterase , HELLP syndrome , Fresh frozen plasma

INTRODUCTION

Pseudocholinesterase deficiency is one of the rare acquired or inherited defect in the enzyme which is produced by liver .(1)

Drugs like succinylcholine and mivacurium are metabolized by this enzyme.(2)

Inherited form of enzyme transfers in autosomal recessive manner secondary to mutations in butyrylcholinesterase gene. Patients may present as heterozygotes or homozygotes(3)

Acquired in several disease states like Malnutrition, Pregnancy& postpartum period,HELLP syndrome,burns, liver and kidney disease, Hemodialysis , Malignancy , chronic infections. Drugs like steroids, cytotoxic agents can decrease the production of enzyme.(4)

METHODS

25 year old primigravida with 36 weeks of gestation was posted for emergency LSCS I/v/o severe pre eclampsia with ?HELLP syndrome with twin gestation with non reassuring NST.

She had come with complaints of dizziness, blurring of vision since 2 days which was associated with 3-4 episodes of vomiting.

She was a recently diagnosed case of PIH on labetalol 100mg BD. She was started on MGSO4 on Pritchard regimen as her BP was 160/100mmhg on admission and having signs of impending eclampsia.

Investigations shows HB- 11.2, TC- 14400, Platelet count – 50,000, BUN/creat – 15.3/1.1, SGOT/SGPT - >350/>350, PT/INR – 15.3/ 1.19 , Fundoscopy – WNL.

Patient was taken inside OT after taking informed consent , ICU/venti standby was asked .

All standard ASA monitors were attached including Neuromuscular monitoring .

Patient was induced under GA (6.5mm cuffed ET tube) with inj.Propofol 80mg + inj. Scholine 75 mg (1.5mg/kg) under RSI .

Procedure was done uneventfully within duration of 1 hour, with blood loss of 400ml . Patient was hemodynamically stable throughout the procedure.

No supplemental muscle relaxant was administered as there was no signs of spontaneous respiratory efforts by the patient and TOF count was 0-1.

Patient was observed for one more hour after surgery . MAC was reduced, all the causes of delayed recovery were ruled out like hypoglycemia, hypothermia, hypermagnesemia, hypokalemia, Acidosis(on ABG) . Total duration of Scholine time – 2.5 hours

We suspected Acquired Pseudocholinesterase deficiency in this patient and decided to give fresh frozen plasma . 300 ml of FFP was given slowly over 30 minutes.

Patient started taking spontaneous breaths, TOF count increased to 4 ,ratio of >90%

Once the patient started taking adequate tidal volume breaths, all the inhalational agents and N2O was stopped ,100% o2 was given.

After confirming signs of complete reversal of neuromuscular blockade - Patient was extubated.

We observed the patient inside OT for 1 hour and shifted to recovery.

In recovery room - multipara monitors were attached and observed for 4 hours . Patient was comfortable and vitally stable hence shifted to ward

CONCLUSION

Pseudocholinesterase deficiency is an infrequently encountered genetic or acquired condition, discovery is typically only after exposure to succinyl choline or mivacurium . So the family history of prolonged mechanical ventilation post surgery must be enquired.(5)

Prolonged neuromuscular blockade should receive treatment with mechanical respiratory support until the spontaneous return of muscle function is felt to involve less risk than reversal with transfusion of plasma .(6)

Patients may be unaware that they have Pseudocholinesterase deficiency if there is no prior exposure to succinyl choline or mivacurium.(7)

Laboratory analysis of Pseudocholinesterase deficiency can be performed qualitatively by dibucaine test , which inhibits normal variant of the enzyme by 80%. Heterozygotes inhibition by 50-60%. 20-30% for homozygotes. (8)

Quantitative testing can be done to determine actual amount of the enzyme present in the plasma . (normal levels 4000-13,500 U/L)(8)

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