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Case Report

A case report on antepartum and postpartum management of Wilson's disease

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ABSTRACT

Wilson's disease incidence is about one in 30,000 live births. Usually occurs between the ages of 5 years and 35 years. It was described first in the year 1854 by Friedrich Theodor von Frerichs (German pathologist) and is named after Samuel Wilson (British neurologist). With our experience in this case, we are explaining the antepartum and postpartum management of a rare case of Wilson's disease.

Keywords: Wilson's disease, Autosomal recessive, Copper, Management

INTRODUCTION

Wilson's disease, also known as hepatolenticular degeneration, is a genetic disorder which is characterized by the excess accumulation of copper in the body. Wilson's disease is due to the sequence variation occurring in the Wilson disease protein (ATP7B) gene.[1] This protein helps in transporting excess copper into bile, and it is excreted in waste products. The condition is autosomal recessive disorder. For a person to be affected, they must inherit a mutated copy of the gene from both of their parents. Diagnosis of the Wilson's disease usually difficult and frequently involves a combination of blood tests, urine tests, and a liver biopsy.

CASE REPORT

We reported a case of successful management of Wilson's disease during both antenatal and postnatal periods. 26 years old, gravida 2, para 1, live 1, known case of Wilson's disease came to Panimalar Medical College and Hospital for antenatal checkup after pregnancy confirmation. Patient was on D-penicillamine previously. Liver function test (LFT) and coagulation profile done and was within normal limits.

Medical gastroenterology opinion obtained and D-penicillamine was withheld due to the risk of teratogenicity during first trimester and was started on tablet zinc ascorbate 50 mg per orally thrice daily. Patient was advised copper restricted diet. Monthly follow-up with medical gastroenterologist was done. Dating scan, nuchal translucency (NT) scan was done and was normal. Anamoly scan done and anamolies ruled out. Growth scan done at 36 weeks showed reduced liquor (amniotic fluid index (AFI) was 6.5 cm) and was admitted for hydration therapy and steroid coverage. Two doses of injection betamethasone 12 mg IM covered 24 hours apart. On admission non-stress test (NST) was reactive. After 3 days, AFI was 5 cm and the patient was induced with two doses of prostaglandin E2 (PGE2) gel on 14 September 2024. Post induction cardiotocogram (CTG) was reactive. ARM was done and minimal clear liquor drained and was started on injection oxytocin.

Patient was taken up for emergency lower segment caesarean section (LSCS) in view of non-progression of labour. On post-operative day (POD) -3, dressing removed and wound was healthy. Patient was restarted on zinc ascorbate per orally thrice daily. Medical gastroenterologist opinion sought and orders followed, advised follow-up after 1 month.

Table 1: Investigations of the patient on admission.

Investigations	On admission	Normal range
Haemoglobin (g/l)	12.1	11.5 to 15.5
Platelet count ($\times 10^9$/l)	2.45	1.5 to 4.5
Total bilirubin	10	0-17
AST (U/l)	10	8-40
ALT (U/l)	12	8-40
Albumin (g/l)	36	35-55
INR	1.0	0.9 to 1.9
ALT	60	40 to 110

DISCUSSION

Wilson's disease, an autosomal recessive disease is due to a mutation in the Wilson disease protein (ATP7B) gene. This protein helps in transport of excess copper into bile, where it is excreted in waste products. If there is a suspicion of Wilson disease is high, the ceruloplasmin level will be less than 20 mg/dl (normal 20 mg/dl to 40 mg/dl).[2] Urinary copper levels will be raised more than 100 mcg/dl. These two lab findings with Kayser-Fleischer rings are usually enough for diagnosis, but if there is the possibility of an alternate diagnosis, order a liver biopsy for liver copper levels; this the most accurate test for Wilson disease. Genetic testing can be used to screen family members of those affected. Wilson's disease is usually treated with dietary changes and medications.[3] Dietary changes include eating a low-copper diet and not using copper cookware.[4] Medications used include chelating agents, such as trientine and D-penicillamine, and zinc supplements.[5] Complications of Wilson's disease are liver failure and kidney problem. Liver transplant can be the treatment option for those with liver failure.[6] Patients become symptomatic between 5 and 35 years of age. The main sites for copper accumulation are the liver and the brain.[7] Patients may have abnormal liver function tests such as raised aspartate transaminase, alanine transaminase, and bilirubin levels.[8] If the liver damage is marked, albumin may be decreased because of an inability of damaged liver cells to produce albumin.[9] Prothrombin time may be prolonged as the liver is unable to produce proteins known as clotting factors.[10]

CONCLUSION

With our experience in this case, we are explaining the management and approach towards a case of Wilson's disease in both antepartum and postpartum periods.

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