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PREGNANCY WITH PINK BLOOD: A CASE REPORT

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ABSTRACT

A 24 year old female P1L1 presented to our hospital on first post operative day of emergency caesarean delivery with complaint of fever with chills and rigors. The per-operative notes showed the presence of thick pinkish maternal blood when compared to cord blood, which turned opaque white while sampling. On examination she had pallor and raised blood pressure. Her lipid levels were very high and ultrasound showed bilateral mild pleural effusion with minimal ascites with increase echogenicity of peripancreatic fat. She was started on injectable antibiotics following which she improved in symptoms. She was also started on fenofibrate and orlistat following which her lipid profile improved and the blood stopped turning opaque white and she was then discharged against medical advice due to financial constraints.

KEYWORDS: Hypertriglyceridemia, Pink blood.

INTRODUCTION

Severe hypertriglyceridemia is rarely encountered in pregnancy and has a poor prognosis for both mother and baby (1). It is associated with an increased risk of acute pancreatitis and pre-eclampsia in the mother and macrosomia in the baby (2-5). Most of the cases reported in literature who developed severe hypertriglyceridemia in pregnancy were either familial or due to previously known genetic mutations in the mother (6). We report a case of severe, pregnancy induced and non-familial hypertriglyceridemia and discuss the technical issues involved in management.

CASE REPORT

A 24-year-old primi para, was referred to our hospital on first post operative day of emergency caesarean delivery at term, done for non-reassuring fetal heart during labour. During caesarean delivery, her blood was found to be pink as compared to the fetal blood which was dark red in color (Fig. 1,4,5). Within 3-4 minutes, the blood would turn opaque white and coagulate (Figure 2-3). The patient had a body mass index of 24 kg/m2 and maternal observations showed elevated blood pressure readings.

Fig. 1: Showing Blood Sample Immediately

Fig. 2: After 5 minutes

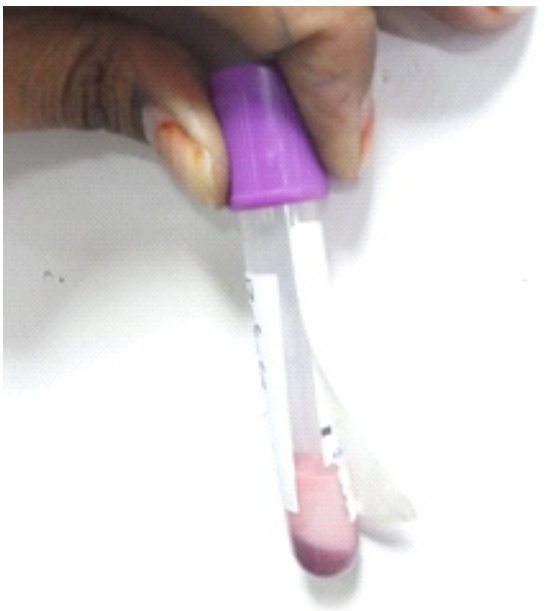

Figure 3: After 10 minutes

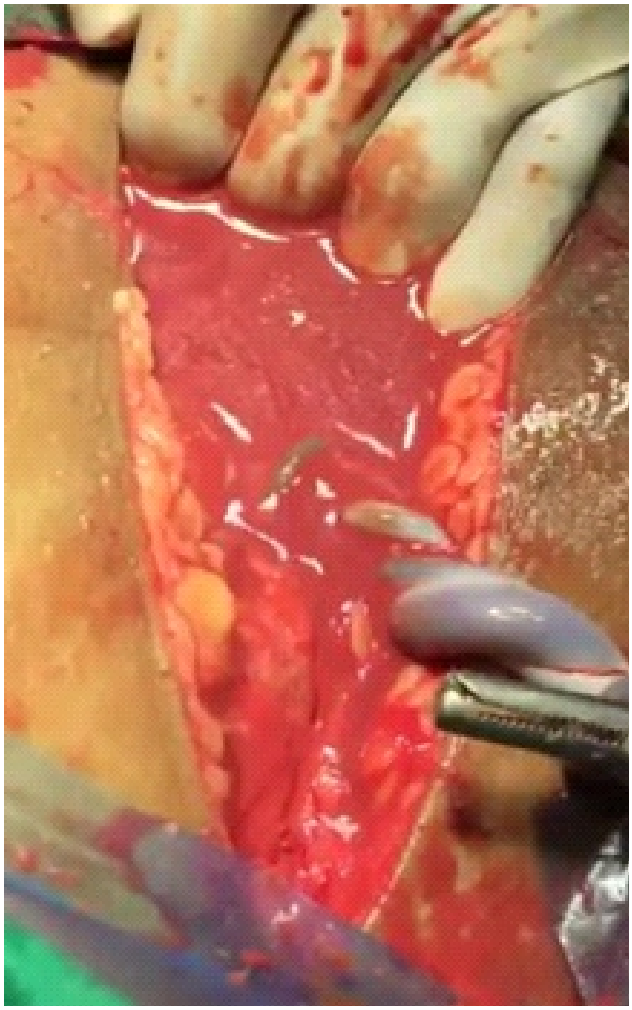

Fig. 4: Pink Blood of Mother during Caesarean section

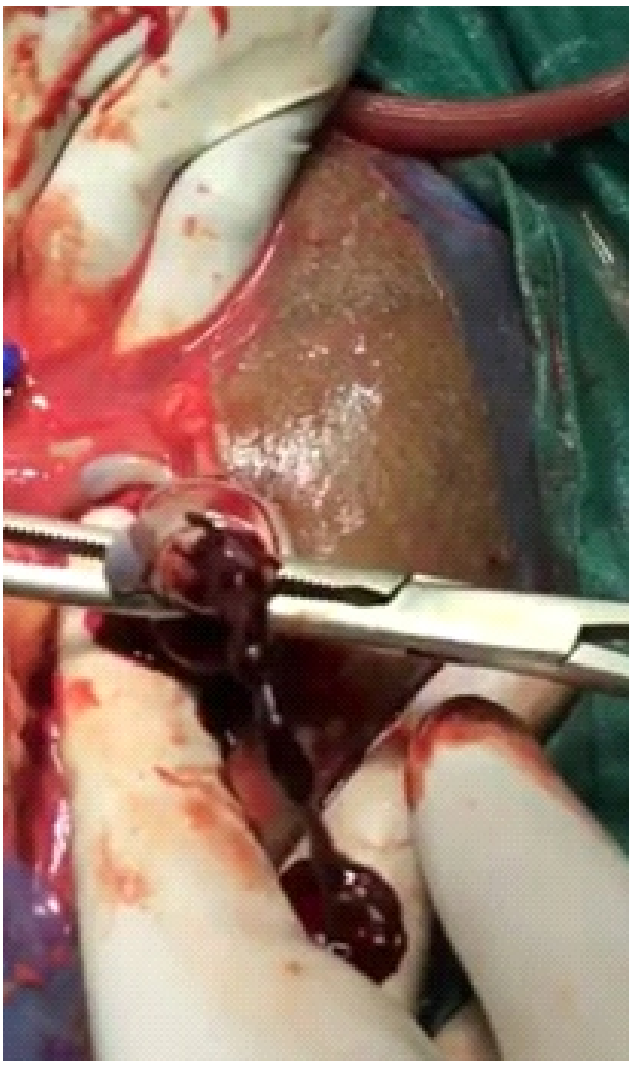

Figure 5: Dark Red Cord Blood Compared to Pink Blood of Mother

She was diagnosed as having lipaemic plasma clinically. Processing her blood sample for investigations proved challenging as the automated machines were displaying error in sample analysis. The blood samples were progressively tested in dilutions and at a dilution of 1:10, the auto analysers were able to process the sample which showed very high levels of triglycerides and cholesterol. (Table 1) There was no personal or family history of hypertriglyceridemia and the reason for high lipid levels remained elusive.

Multidisciplinary management was instituted for the case. Table 2 shows the treatment regime tailored for management. The patient was gradually tapered off medication in the next three months. Her blood pressure and other laboratory indices came back to normal and she was off treatment at sixth month of follow up.

Parameter	Day 1	Day 8	Ref. Val.
Serum Triglycerides	2775 mg/dl	1297 mg/dl	*Borderline High: 150-199 mg/dl* *High: 200-499 mg/dl* *Normal: <150 mg/dl* *Very High: >=500 mg/dl*
Serum Cholesterol	726 mg/dl	559 mg/dl	*Borderline High: 200-239 mg/dl* *Desirable: <200 mg/dl* *High: >=240 mg/dl*
Serum HDL (High Density Lipoprotein)	21 mg/dl	134 mg/dl	*High: >= 60.0 mg/dl* *Low: <40.0 mg/dl*
Serum V.L.D.L (Very Low Density Lipoprotein)		259mg/dl	*2-30 mg/dl*
Serum LDL (Low Density Lipoprotein)		166 mg/dl	*<= 100 mg/dl*
Serum Amylase	648 U/L	181 U/L	30-110 U/L
Serum Lipase	1309 U/L	364 U/L	23-300 U/L

Table 1: Different Lab Investigations

Treatment given	Mechanism	Benefit
Low fat diet 15-20% of calories from fat/day	Reduces substrates for exogenous triglyceride synthesis pathway	Effective in triglyceride lowering
Omega 3 acid ethyl esters 3-4 g/day orally	Reduce hepatic triglyceride synthesis Increase fatty acid oxidation in the liver and skeletal muscle Enhance lipoprotein lipaseactivity	Reduce triglyceride by 25-50% via several mechanism Helps avoid deficiency of key omega-3 fatty acids including docosahexanoic acid and eicosapentoic acid
Parenteral nutrition	Less increase in triglyceride from intravenous carbohydrate ingestion compared to enteral carbohydrate nutrition	Provides source of calories
Statins (Tab Atorvastatin+fenofibrate 20/160 mg)	Atorvastatin is 3-Hydroxy-3- methylglutaryl-coenzyme A reductase (HMG CoAreductase) inhibitor Fenofibrate is peroxisome proliferator receptor alpha activator (PPAR α), PPAR α activates lipoprotein lipase and reduces apoprotein CIII	Decrease production of cholesterol Increase lipolysis and elimination of triglyceride rich particles from plasma
Tab orlistat 120 mg 8 hourly	Gastric lipase inhibitor Reduc es intestinal free fatty acid concentration	Reduces chylomicron synthesis
Heparin	Release lipoprotein lipase from the endothelium in the plasma	Lowers triglyceride levels
Antihypertensive (tab Nifedipine 20mg×12 hourly +tab metoprolol×12.5 mg once a day		Control of blood pressure

Table 2: Treatment Regime in Management

DISCUSSION

Plasma lipids start increasing from the first trimester of pregnancy and progressively rise so that by the end of third trimester, serum triglycerides have risen by 200-300% and serum cholesterol has risen by 25-50% (7). Due to rise in blood volume however, hypertriglyceridemia is rarely seen. Genetic predisposition, mutations, alcohol consumption, liver disorder, renal disease, type 2 Diabetes mellitus are known risk factors for severe hypertriglyceridemia.[1,8]

Hypertriglyceridemia poses a grave threat to maternal and fetal health irrespective of its cause. 4-6% of acute pancreatitis during pregnancy occur due to hypertriglyceridemia (5). Women who have plasma triglyceride >1000 mg/dl or those having hypertriglyceridemia before pregnancy are at increased risk of developing acute pancreatitis during pregnancy. Other complications are pancreatic pseudocyst, pancreatic necrosis, significant electrolyte derangement, preeclampsia, hyperviscosity syndrome, fetal macrosomia, intrauterine fetal death, preterm labor.

This patient presented in casualty with fever with chills and rigors and raised blood pressure on first post operative day of emergency caesarean delivery. On further investigations diagnosis of hypertriglyceridemia induced acute pancreatitis was made, as the patient was having very high lipid levels and high serum amylase and lipase levels with ultrasound findings suggestive of pancreatitis. No predisposing factor for such severe hypertriglyceridemia was present in this patient.

Patient was managed with dietary modifications, antibiotics, lipid lowering drugs like statins and fibrates and omega 3 fatty acids.

Diet and nutritional supplements are very important in the manangement of hypertriglyceridemia. Diet should be isocaloric and low in fat with <20% of calories from fat. Omega-3 fatty acids contain eicosapentoic acid and docosahexanoic acid, which downregulate hepatic lipogenesis and stimulate fatty acid oxidation in the liver and skeletal muscle reducing triglyceride level by 25-30%.

Statins and fibrates (gemfibrozil) are prescribed if dietary changes alone are not enough to treat hypercholesterolemia and hypertriglyceridemia. In pregnancy, most fibrates are contraindicated as they carry risk of teratogenicity. But as per few reports, gemfibrozil has been used successfully, in the patients who were refractory to dietary medication and omega-3 fatty acids. It is reported to decrease the risk of developing hypertriglyceridemic pancreatitis with no side effects, especially in the third trimester.

In diabetes-associated hypertriglyceridemia insulin increases removal of triglycerides from the plasma as it activates lipoprotein lipase (7). In resistant cases, other treatments are used like plasmapheresis, plasma exchange, total parenteral nutrition, and intravenous heparin (1,3).

Immediate and aggressive management should be initiated in patients who present with complications associated with hypertriglyceridemia. Plasma lipid levels rapidly decline after delivery in case of gestational hypertriglyceridemia (10-20% decrease in triglyceride levels within the next 24 hours). So, if gestational age permits, pregnancy can be interrupted.[9] In this case hypertriglyceridemia was undiagnosed during antenatal period and casesarean delivery was done for non-reassuring fetal heart rate during labour at term and patient presented to our hospital with features hypertriglyceridemia induced pancreatitis post-delivery. Here the patient was managed by multidisciplinary team approach and improved.

There is controversy around recurrence risk of hypertriglyceridemia in subsequent pregnancies. For contraception, the best way is to use copper intrauterine devices. Also, progestin can be given at a very minimum dose but under regular monitoring.[1] Genetic etiological testing is not indicated for routine hypertriglyceridemia cases (6). However, in cases of subsequent pregnancy, the recurrence risk may necessitate the need for clinical and laboratory monitoring second trimester onwards (3).

CONCLUSION

Due to physiologic changes during pregnancy triglyceride levels rise and typically increase by 2-3 fold in third trimester. In patient who have underlying defect in lipid metabolism or insulin resistance, severe hypertriglyceridemia can develop. Dietary management is one of the best therapeutic modalities, but early delivery is advised and lipid lowering drugs may be used after the delivery. In cases with persistent hypertriglyceridemia, the monitoring in the post pregnancy stage becomes essential till the triglyceride levels return to normal. Complications in terms of acute pancreatitis must be ruled out since it carries a high risk of mortality.

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