A Case Of Severe Obstetric Cholestasis With Subsequent Severe Preeclampsia

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Background

Obstetric cholestasis (OC) and preeclampsia are important diagnoses to make, as they are associated with increased fetal morbidity and mortality. OC carries known risk of stillbirth, and increased rates of preterm birth. They both also carry a risk of recurrence in subsequent pregnancies. These conditions are managed in part by planning delivery of the fetus, whilst optimising fetal maturity & wellbeing and maternal wellbeing. Adequate monitoring of patients and timely intervention remains vital in patient outcomes.[1]

Case

A 37 year old G6P4, presented at 28+0 weeks complaining of pruritis without rash. She had previously had 4 term spontaneous vaginal deliveries, complicated only by postpartum haemorrhage. Clinical history and biochemical markers were consistent with OC, and she was prescribed ursodeoxycholic acid.

On clinic review at 29+2 she was mildly hypertensive and subsequently admitted for investigation and monitoring. She was otherwise asymptomatic. Abdominal ultrasound showed a gallbladder distended with sludge, suggestive of cholestasis. Obstetric ultrasound showed a growth restricted fetus, with normal amniotic fluid and abnormally low middle cerebral artery pulsatility index.

She quickly progressed to hypertensive crisis, with a diagnosis of severe preeclampsia with likely HELLP syndrome based on evolving biochemistry. Magnesium sulphate and labetalol infusions were started, and she was steroid loaded. She was transferred to a tertiary referral hospital with appropriate neonatal facilities for ongoing care.

Due to deteriorating biochemistry and cardiotocography, she underwent an uncomplicated emergency LUSCS at 29+4, delivering a 1.29kg baby boy. Apgars were recorded as 6 (1min), 8 (5min). Proteinuria was never observed during her care. Bile acids taken on the day of presentation, returned after delivery at **385 micromol/ml**.

Gestational age, time	28+4	29+1	29+2 1252	29+2 1716	29+3 0130	29+3 1820	29+3 2355 Pre-delivery	29+4 0800 Post-delivery
Hb (115-155)	136		142	147	141	147	132	134
Plt (150-450)	140		111	120	122	150	119	120
Creatinine			64		75	94	84	69
Urea (2.7-8)			4.0		4.6	5.6	6.2	5.4
Bili (2-24)	18	25	20	27	16	15	12	13
ALP (30-110)	286	351	343	406	315	351	289	274
ALT (0-55)	231	289	227	227	268	393	367	364
AST (0-45)			191	201	302	446	409	387
GGT (0-60)	117	139	141	148	125	146	138	142
LDH (<250)			349	342	410	452	384	445
Uric Acid			0.45	0.56	0.51	0.56		0.53
Coags			NAD			NAD		

Discussion

We observed severe disease in this patient, of two processes traditionally thought of as separate. Research has begun to postulate links between the development of severe preeclampsia in the setting of known severe OC. Raz et al[1] studied the disease course of a group of women with mild, moderate and severe OC, compared to a control group. Severe OC was defined as a bile acid of >40. Preeclampsia was generally seen to develop 2-4 weeks after the onset of OC. Severe, but not mild, OC was thought to be a major risk factor for the development of severe preeclampsia.

In their series of patients, all developed significant proteinuria prior to the development of their hypertension. Of interest in our patient was that we never observed proteinuria throughout her disease course. Additionally, her bile acids returned as 385 several days after the patient had been transferred – an exceptionally high result.

A diagnosis of HELLP syndrome (haemolysis, elevated liver enzymes, low platelets) was debated. Whilst thought to be a subset of preeclampsia, the diagnosis was not achieved based on SOMANZ criteria[2], wherein the platelets did not drop below 100, nor did the LDH rise above 600. In this case, the discussion around diagnosis of HELLP syndrome is purely academic.

Conclusion

While often independent disease processes, with different aetiologies, research has suggested that severe OC may be a risk factor for severe preeclampsia. Severe OC should therefore prompt vigilance in screening for preeclampsia and related entities. This case was of interest, in that it displayed severe and quickly-escalating disease process with some odd pathology findings.

References

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