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Non-gallstone acute pancreatitis and pre-eclampsia: A case report

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ABSTRACT

Acute pancreatitis is rare but well documented in pregnancy and most cases are attributable to biliary disease. We present a case of acute non-gallstone pancreatitis in a patient with acute and severe pre-eclampsia.

A 39-year-old primigravida woman at 33 + 4 weeks' dichorionic diamniotic gestation presented with severe bilateral lower-limb oedema and underwent an emergency caesarean section due to the development of acute severe pre-eclampsia. Postpartum, the woman developed out-of-proportion generalised upper abdominal tenderness with worsening liver function and markedly raised lipase and amylase levels. Imaging confirmed oedema and inflammatory changes in keeping with acute non-gallstone pancreatitis. The patient improved with conservative management and was eventually discharged home on day 13 postpartum.

The development of abdominal pain out of proportion to expected clinical progression should prompt the physician to consider other causes, including acute pancreatitis, in order to provide effective and timely clinical care. The clinical presentation of this woman suggests that pre-eclampsia may be associated with the development of acute pancreatitis. Further prospective study would be needed to establish any association.

1. Introduction

Acute pancreatitis complicates 1 in 1000–3000 pregnancies [1]. It usually occurs during the third trimester [1] or the early postpartum period and is most commonly caused by cholelithiasis [2,3]. We present a case where a patient developed acute postpartum pancreatitis after a pregnancy complicated by acute severe pre-eclampsia. With early diagnosis and supportive treatment, the patient recovered uneventfully.

2. Patient Information

This 39-year-old primigravida woman had had a largely unremarkable antenatal history: spontaneous conception of dichorionic diamniotic twin, normal antenatal blood tests (rhesus positive, serology negative, rubella immune), normal oral glucose tolerance test, low-risk non-invasive prenatal test (Harmony) and a normal morphology scan. Her ultrasound scans at 28 and 32 weeks' gestation also revealed growth concordance between both twins, large for gestation for twin B, without any evidence of fetal compromise on biophysical or doppler assessment on both twins. She was taking pregnancy supplements and thyroxine for subclinical hypothyroidism but was not on any other regular medication during her pregnancy.

The patient first noticed bilateral lower-limb swelling between 18 and 20 weeks' gestation. This progressively worsened over the next month, with swelling extending to the thighs by 24–26 weeks' gestation. Nonetheless, the patient did not report any associated signs and symptoms to suggest an evolving pre-eclamptic picture; she denied any pre-eclampsia symptoms and was normotensive on all her routine antenatal visits. Due to extent of the lower-limb oedema, she had bilateral lower-limb Doppler scans, which were negative for deep-vein thrombosis. The oedema progressed to a stage where her mobility was affected and the pain was intolerable; the patient also experienced some non-specific rigours/chills. Therefore, 24 h later, at 33 + 4 weeks' gestation, she presented to the hospital for assessment and treatment.

Her presentation quickly evolved into a pre-eclamptic picture with severe proteinuria on urine dipstick, acute kidney injury (with a creatinine level at 240 μmol/L) and elevated liver enzyme levels (alanine transaminase at 101 U/L and aspartate transaminase at 111 U/L) and an elevated urate level (0.82 mmol/L). She had a single dose of steroids and proceeded with an emergency lower caesarean section the next day. As per our hospital protocol, she was administered intravenous magnesium sulphate, which continued after delivery.

On day 2 postpartum, she complained of generalised upper abdominal pain, discomfort and distension. Her blood pressure (without treatment) remained stable postpartum; systolic blood pressure was 130–140 mm Hg with stabilising kidney function. However, she recorded worsening liver function.

From the pre-eclampsia point of view, she continued to improve without any treatment; she remained normotensive from day 2

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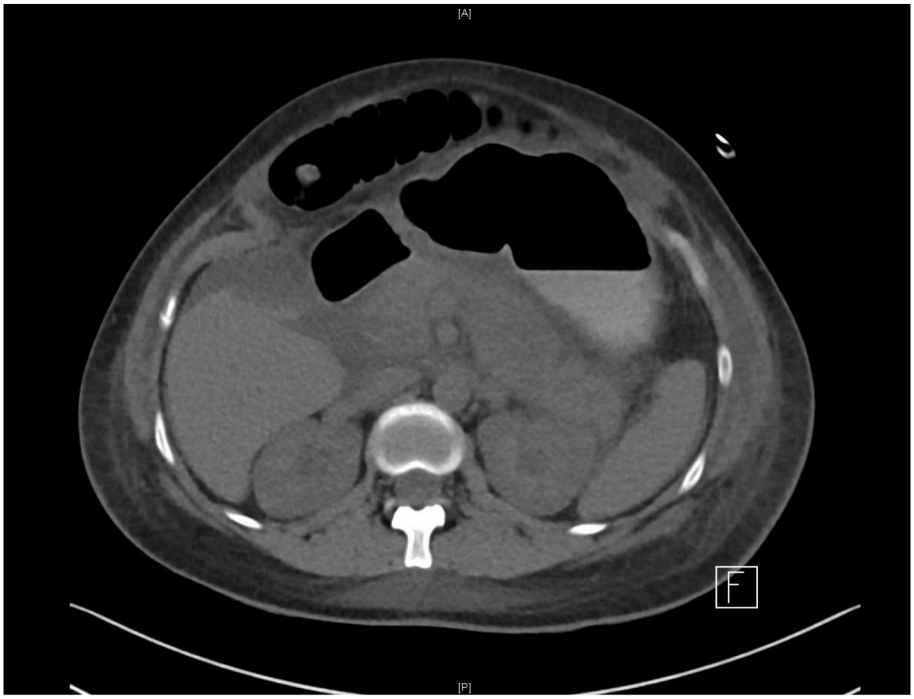

Fig. 1. Diffuse pancreatic oedema with peri-pancreatic fat stranding.

postpartum and her kidney function had returned to premorbid state by day 5 postpartum.

2.1. Diagnostic Assessment and Management

The patient was informed that further testing would be required to investigate her worsening liver function and generalised upper abdominal pain out of proportion with routine caesarean section and the current clinical picture. Serum amylase and lipase levels were recorded at 773 U/L and 8774 U/L respectively, with persistently abnormal liver function tests and hypoalbuminaemia. Ultrasound and CT abdomen scans confirmed pancreatitis: diffuse pancreatic oedema and extensive peri-pancreatic fat stranding and free fluid with an unremarkable biliary tree and only a small calculus within the fundus of the gallbladder (Figs 1 and 2).

Conservative management and supportive treatment were undertaken: she was transferred to the care of the high dependency unit under the care of the general surgeons for closer observation, and was

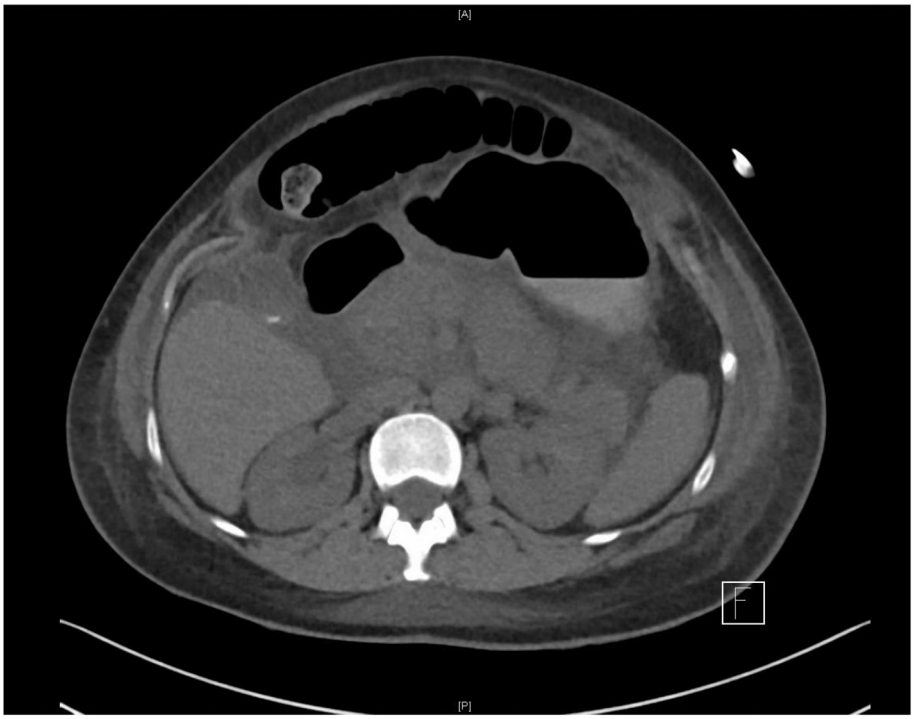

Fig. 2. A small calculus within the fundus of the gallbladder.

fed via a nasogastric tube (with free drainage and regular aspirate) and nil by mouth. She was not treated with any anti-hypertensives or diuretics. She continued to improve clinically and her diet was progressively stepped up. She was discharged on day 13 after the caesarean section.

2.2. Follow-up and Outcome

Four weeks after discharge, the patient was followed up at the general surgery outpatient clinic. She had fully recovered; bilateral lower-limb swelling and upper abdominal pain had completely resolved and she was consuming a full diet. Repeat kidney function, liver function and amylase/lipase tests had all normalised. She was advised to take prophylactic aspirin if she became pregnant again.

3. Discussion

Acute pancreatitis complicates 1 in 1000–3000 pregnancies [1]. While it can occur in any trimester, it is most commonly seen in the third [1]; post-partum occurrence has also been seen, as in our case and others [4–7]. Biliary disease is the most common cause of acute pancreatitis in pregnancy and has been reported to be responsible for 67–100% of cases [1,8,9].

Pancreatitis in association with pre-eclampsia is rare [5,6,10–13]. In a review by Ramin et al. [1], none of the cases of pre-eclampsia was thought to be the cause of pancreatitis and in an older review [9] 9 out of the 98 cases of pancreatitis were thought to be associated with pre-eclampsia but 5 out of these 9 patients received diuretics, which are known to induce pancreatitis [14,15].

Nonetheless, microvascular abnormalities observed with pre-eclampsia and eclampsia can affect cerebral, placental, hepatic, renal and splanchnic circulation as a result of severe disturbances in microcirculation due to endothelial damage [16]. It is thus possible that the pancreatic vasculature can be altered and contributes to acute pancreatitis. Haukland et al. [17] found that pre-eclampsia was associated with increased amylase levels, suggesting a pancreatic involvement. As in this case, the patient developed acute pancreatitis not related to gallstone disease and none of her medications (paracetamol, metronidazole, cephazolin, magnesium sulphate, metoclopramide and oxycodone) was thought to be associated with pancreatitis.

In fact, a recent study by Hacker [18] found pancreatitis in pregnancy to be strongly associated with pre-eclampsia (odds ratio 4.21) and notably with severe pre-eclampsia (odds ratio 7.85).

In conclusion, we have reported a case of pre-eclampsia associated with acute non-gallstone pancreatitis. Out-of-proportion and generalised upper abdominal pain postpartum (with or without existing pre-eclampsia) should alert the clinician to the possibility of inflammatory conditions, including acute pancreatitis, as prompt management has been shown to improve the outcome. Given this, prospective studies should reassess the potential association between pre-eclampsia and pancreatitis.

Contributors

Chris Chan and Monika Mukerji were equal and sole contributors.

Conflict of Interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

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Patient consent

Written informed consent was obtained from the patient for the publication of this case report.

Provenance and peer review

This article has undergone peer review.

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