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Lesson of the week

Postpartum eclampsia of late onset

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Late onset postpartum eclampsia can occur in normotensive women with uncomplicated pregnancies, not just in women with pre-eclampsia

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Pre-eclampsia and eclampsia occur in 6% to 8% of all pregnancies.[1] The British eclampsia study confirmed 383 cases of eclampsia during 1992 and warned of the severe consequences of the condition.[2] In 1997 Leitch and colleagues showed that over a 60 year period the incidence of eclampsia had fallen from 74/10 000 to 7.4/10 000, although the incidence of postpartum eclampsia had increased.[3]

A US study identified 229 cases of postpartum pre-eclampsia or eclampsia between 1992 and 2002[4]; 151 of these cases were diagnosed after readmission to hospital with new symptoms and signs after delivery, and 16% (24/151) of these had eclampsia. Other work from the United States identified 89 cases of eclampsia during 1996 to 2001[5]; 29 cases (33%) presented in the postpartum period. In both these US studies most cases developed symptoms more than 48 hours post partum.

Most antenatal and intrapartum cases of eclampsia present to obstetricians, but postpartum cases are more likely to be encountered by non-obstetricians. We present a case of postpartum eclampsia.

Case report

A 27 year old woman in her second pregnancy delivered a healthy male baby weighing 3690 g at 38 weeks by elective caesarean section. Clinically significant proteinuria was present from 30 weeks onwards. In her first pregnancy she had had pre-eclampsia requiring delivery at 38 weeks. This second pregnancy was by a different partner. She was discharged on the third postpartum day with blood pressure of 130/68 mm Hg and no protein in her urine.

She visited her general practitioner on the ninth postpartum day because her baby was unwell with a rash, and she was seen in the accident and emergency department as the mother of a child requiring neonatal assessment. As the mother too looked ill, she was also examined. She was noted to have low grade fever, bradycardia, and hypotension. Her urine showed moderate proteinuria (2+) on dipstick examination. Chest radiography and blood and urine cultures were normal. She was admitted under the care of the general physicians and started on intravenous ceftriaxone for suspected infection. She received 500 ml of gelatin followed by 500 ml of normal saline. Overnight her blood pressure rose to 186/92, which was treated initially with nifedipine. Her blood pressure continued to rise—to a maximum of 200/112 mm Hg. She developed a headache and suddenly lost vision in both eyes. This was followed by a generalised tonic clonic convulsion. Blood analysis—including full blood count, urea and electrolytes, and glucose—was normal, as were her thyroid function tests, anticardiolipin, extractible nuclear antigens, double stranded DNA antibodies, and complement concentrations. Blood urate was not assessed. Urinary catecholamines were negative. Doppler examination of renal vasculature and renal ultrasonography were normal.

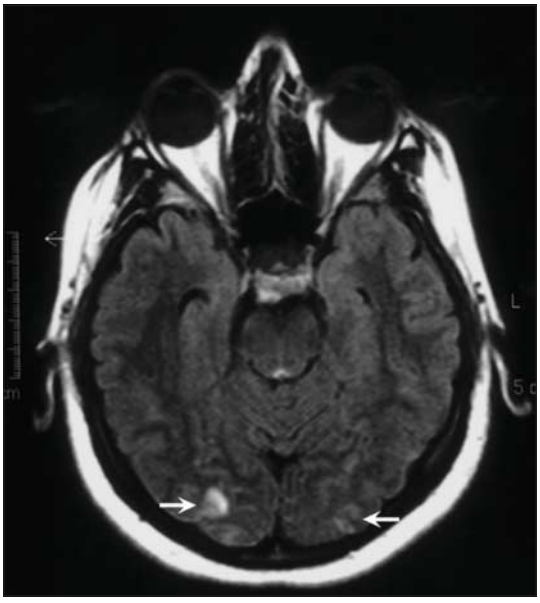
The consulting obstetricians diagnosed eclampsia, and the patient was started on intravenous hydralazine and later magnesium sulphate. A magnetic resonance scan of the brain showed small areas of high signal intensity on T2 weighted images, involving both cortical and subcortical white matter of the right posterior parietal and both occipital lobes (figure). There was no evidence of veno-occlusive disease. The scan suggested hypertensive posterior cerebral encephalopathy. The patient regained her vision within 24 hours after her blood pressure was controlled, and she was prescribed labetalol when she was discharged home 10 days later.

Discussion

Eclampsia is a poorly understood multisystem complication of pregnancy that substantially contributes to maternal morbidity and mortality. The typical clinical picture is of generalised tonic clonic seizures during the third trimester, labour, or early puerperium in women who already have hypertension, proteinuria, and oedema.

The previously controversial existence of a delayed postpartum variant of eclampsia is now acknowledged by most experts.[2–8] Convulsions with initial presentation more than 48 hours but less than four weeks after delivery are commonly referred to as late postpartum eclampsia.[6]

Our patient had convulsions on the ninth postpartum day. Her response to magnesium sulphate, the magnetic resonance scan showing hypertensive



Axial FLAIR magnetic resonance image showing high T2 signal intensity abnormality involving bilateral parieto-occipital cortex and subcortical white matter (arrows)

posterior cerebral encephalopathy, the exclusion of metabolic and infectious causes strongly support the diagnosis of eclampsia.

Postpartum eclampsia can present with a variety of clinical and neurological symptoms and signs. Lubrasky[6] and Chames[5] reported that 44% and 79% of their respective patients with late onset postpartum eclampsia had not been identified as having pre-eclampsia before seizure onset. They reported that severe and persistent headache, visual symptoms, epigastric or right upper quadrant pain, and hypertension can present as prodromal symptoms before the onset of eclampsia.[4–6] Our patient had these symptoms.

Eclampsia should be considered in any postpartum woman who develops any of these prodromal symptoms. Further indicators include convulsions up to four weeks after delivery, hypertension, or proteinuria. This is important as eclampsia is amenable to treatment with magnesium sulphate.

The differential diagnosis includes epilepsy, cerebral venous thrombosis, intracerebral haemorrhage, phaeochromocytoma, space occupying lesions, and metabolic disorders.[9] Neuroimaging, especially magnetic resonance imaging, shows micro-ischaemic injury patterns in parieto-occipital lobes.[10] This form of posterior leukoencephalopathy syndrome can cause cortical blindness, which may be reversible with control of hypertension and magnesium sulphate therapy, as in our case. Lesions on a magnetic resonance scan cannot predict whether damage leading to cortical blindness is permanent or likely to be reversible.

Magnesium sulphate remains the drug of choice for preventing and treating eclamptic seizures. If such seizures are not treated appropriately, grave complications such as intracerebral haemorrhage and death can occur.

Late onset postpartum eclampsia can occur in normotensive uncomplicated postpartum women as well as in women with pre-eclampsia. The presence of prodromal symptoms should be thoroughly investigated, even in the absence of antecedent pre-eclampsia. We suggest that such patients seen in accident and emergency units within 14 days of delivery should be assessed by an experienced obstetrician.

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Competing interests: ABM co-edited a publication for the Royal College of Obstetricians and Gynaecologists in 2003 on pre-eclampsia, but without financial gain.

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ALIAS (a landing in an alphabet soup)

All medical specialties use short forms and acronyms, and I had thought O&G (obstetrics and gynaecology) must be one of the top contenders. However, a conference on HIV management really opened my eyes.

No drug used in the management of HIV does not have a short form of its own. Almost all the best combinations have now been used up, like web addresses, and the newer discoveries will have to make do with numbers or symbols.

Initially, it seemed a piece of cake: abacavir is called ABC, atazanavir is called ATV, and indinavir is called IDV. No problems. But then matters took a turn for the worse with zidovudine being called AZT. I can understand the Z, but what are A and T doing there? Next I was faced with didanosine and tenofovir. If I were asked to devise their acronyms, I would have said DDN and TNF. However, no one had asked me but had apparently consulted somebody with a massive word ataxia, with the result that didanosine is called ddl and tenofovir is TDF. Even better, the drug ritonavir is simply known as /r (yes, it starts with an oblique sign).

We seemed to be returning to sanity when we were told SQV meant saquinavir, but then we learnt that SQV also meant Fortovase, which is also known as FTV. Just as I was recovering from that blow, I was given a knockout punch—SQV and FTV can both stand for SGC. This must be a world first—a short form for an acronym.

After that, matters grew more surreal. Can you guess what ddC and d4T could mean? Well, ddC means zalcitabine (I knew you would guess that one), and d4T means stavudine. Don't ask me where that 4 comes from or what that weird capitalisation is all about. For that matter, do not even think about how FTC can mean emtricitabine or 3TC can mean lamivudine.

At the end of the conference, feeling dazed and confused, I was surrounded by a dozen upstarts, who started discussing all those alphabets knowingly. Reeling from this onslaught, I retaliated with a devastating riposte: "You say you must use ABC or XYZ if the CD4 count comes down. Let me ask you a simple question. What is the full form of CD, as in CD4 and CD8, and just how many of these CDs are there?"

I proudly tilted my nose upwards and walked out triumphantly.

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