cryptogenic aneurysms — where a definitive infectious focus elsewhere in the body cannot be detected.

Weisman\(^1\) reports that in a postmortem examination of a patient with cavernous sinus thrombophlebitis a polymorph infiltration in the adventitia and media of the carotid artery with intimal proliferation was found.

According to Rout \(^2\) only 18 cases of bacterial intracavernous carotid aneurysms secondary to cavernous sinus thrombophlebitis had been reported in the literature; 10 of these occurred in children. Staphylococci from septic foci that could have led to the thrombophlebitis were isolated in 9 cases. The diagnoses in 5 of the 6 cases described by Rout \(^3\) were made after ophthalmoplegia caused by cavernous sinus thrombosis failed to clear after 4 - 6 weeks of antibiotic therapy. Arteriography demonstrated the aneurysms. It is therefore suggested that if long-term antibiotic therapy is instituted for an infective aneurysm, repeat angiography should be performed to monitor the size of the lesion. Of a total of 11 patients who had follow-up angiograms, in 5 the aneurysms progressively enlarged, in 5 they disappeared and 1 patient developed thrombosis of the internal carotid artery. It is suggested that in patients in whom the ophthalmoplegia improves and the size of the aneurysm decreases on repeat angiograms 4 - 6 weeks after admission, conservative management can be followed.\(^3\) Digital subtraction angiography can be done in order to reduce the risks of repeated arterial catheterisation. Carotid ligation is indicated in all cases where the size of the aneurysm is shown on angiography to have increased.

The incidence of rupture of an intracavernous aneurysm is lower than elsewhere in the body since the dura mater of the cavernous sinus supports the aneurysm and its course is therefore more benign.

REFERENCES


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**Infective endocarditis in pregnancy**

**A report of 3 cases**

J. DOMMISSE

**Summary**

Three pregnant patients with infective endocarditis presented with haematuria. The importance of this symptom is stressed and a brief review of immune-complex glomerulonephritis is given. The role of antibiotic therapy in preventing infective endocarditis is discussed.


**Case reports**

**Case 1**

A 21-year-old patient had a history of rheumatic fever at the age of 12 years with resultant mitral incompetence, mild mitral stenosis and aortic incompetence. She had had 1 previous uncomplicated delivery.

She attended the antenatal clinic regularly and at 16, 20 and 24 weeks was noted to have haematuria. This was confirmed by microscopy. She was treated for supposed urinary tract infection although urine cultures were negative.

At 36 weeks' gestation labour was induced with vaginal prostaglandin E\(_2\) for suspected fetal growth retardation. This was confirmed when an infant weighing 1240 g was delivered by forceps.

She was discharged from hospital, apparently well, on day 6 but was readmitted to a medical ward 6 weeks later with haematuria and proteinuria. Echocardiography revealed mitral valve vegetation. Blood cultures were negative but a diagnosis of immune-complex glomerulonephritis owing to infective endocarditis was assumed and the patient treated with intravenous antibiotics.

The mitral valve was later replaced and histology of the stenosed valve revealed a septic thrombosis on one valve leaflet.

**Case 2**

This patient, aged 26 years, booked for antenatal care at 20 weeks. No cardiac lesion was detected. She had had 1 previous normal delivery.

Haematuria was noted at the 34- and 37-week visits. Urine culture was negative and no treatment was prescribed.

Four days later she was found unconscious and after admission to hospital computed tomography revealed a left-sided cerebral infarct. Mitral incompetence was detected and \textit{Streptococcus mitis} grown in blood culture.

Labour started and she was delivered by forceps of a 2810 g infant. The patient died 7 days later without regaining consciousness.

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Case 3
This patient, aged 19 years, was known to have a ventricular septal defect and booked at 18 weeks. She developed proteinuria and haematuria at 32 weeks gestation. Blood culture revealed Gram-positive cocci in chains, probably Peptostreptococcus. She was treated with intravenous penicillin and later netilmicin with good effect. The haematuria and proteinuria resolved. She was delivered of a 3780 g baby spontaneously at term. The ventricular septal defect will be repaired later.

Discussion
Infective endocarditis during pregnancy is an uncommon occurrence. In a recent literature survey, Seaworth and Durack1 noted only 92 reported cases during the last 40 years. Forty-nine of these occurred during pregnancy, 18 in the puerperium and 25 after abortion. Sugrue et al.2 cited a series of 3915 pregnancies in patients with cardiac disease in which there were only 2 cases of infective endocarditis unequivocally related to the pregnancy.

The 3 patients reported here all presented during the antenatal period with haematuria. There were no other associated symptoms or signs to suggest infective endocarditis. Two patients were inadequately investigated; this resulted in progressive deterioration and a fatal outcome owing to cerebral embolus in 1 and the development of intractable cardiac failure necessitating valve replacement in 1. Patient 3 responded well to treatment and the outcome was favourable.

Transient haematuria is not uncommon in pregnancy. There are a number of known causes such as urinary tract infection, but so-called 'idiopathic haematuria' may occur and resolve spontaneously. However, the importance of haematuria in a patient with known cardiac valvular disease must be stressed. Such cases must be fully investigated and infective endocarditis excluded.

Haematuria, proteinuria and renal dysfunction were previously thought to be caused by glomerular lodgement of microscopic emboli.3 More recent studies suggest that these conditions are more likely to be the result of an immune mechanism.4 Circulating immune complexes have been detected in up to 90% of patients with infective endocarditis.5,7 This type of immune-complex glomerulonephritis is often associated with infections caused by less virulent organisms.5,7 Such infections run a longer course, producing a greater antibody response. This subject was well reviewed by Neugarten and Baldwin8 in 1984.

Infective endocarditis may develop during pregnancy, at the time of delivery or in the puerperium, but as symptoms only manifest some time later it is not usually possible to relate the source of infection to any specific event during pregnancy.

While haematuria and proteinuria may be due to pregnancy complications such as gestational proteinuric hypertension (pre-eclampsia) or urinary tract infection, the patient with known cardiac valvular disease should be more extensively investigated. Appropriate investigations include repeated blood cultures, echocardiography and possibly an attempt to identify circulating immune complexes.8

The use of prophylactic antibiotics during delivery would obviously not have prevented the above 3 patients from developing infective endocarditis. Over the past 5 years, during which 650 cardiac patients were delivered in this centre, we have encountered no other cases of infective endocarditis and none specifically related to parturition. Our present policy is therefore not to administer prophylactic antibiotics routinely to all cases at delivery. Intravenous penicillin and amikacin are, however, administered during labour and for 24 hours after delivery to high-risk cases, that is patients with prolonged labour or whose membranes have been ruptured for more than 12 hours, traumatic vaginal deliveries, manual removal of the placenta, caesarean section and patients with prosthetic heart valves.

This policy is in keeping with the regimen advocated by the American Heart Association.9 Seaworth and Durack1 also concluded that: 'Antibiotics need not be given for the prevention of infective endocarditis before most common obstetric and gynecological procedures, including uncomplicated vaginal delivery.'

REFERENCES