Non-Bacterial Thrombotic Endocarditis in a CAPD Patient

K.B.		Modi
I.S.		Henderson

Renal Unit, Stobhill Hospital, and Renal Unit, Royal Infirmary, Glasgow, UK

Dr. K.B. Modi, MD, MRCP, Renal Unit, Stobhill Hospital, Glasgow G21 3UW (UK)

Dear Sir,

We report a case of non-bacterial thrombotic endocarditis (NBTE or marantic endocarditis) causing cardiac failure and death in a patient with end-stage chronic renal failure managed with continuous ambulatory peritoneal dialysis (CAPD).

The patient was a 60-year-old male who developed chronic renal failure due to accelerated hypertension detected in July 1979. Regular home haemodialysis was commenced in March 1981. He suffered from recurrent fluid overload and cardiac failure due to unsatisfactory control of systemic hypertension and excessive fluid-intake and required several admissions for ultrafiltration. He was noted to have a long systolic murmur, best heard at the aortic area, which was shown to be due to aortic valve sclerosis after clinical and echocardiographic assessment in March 1984. In August 1984, the patient chose to change from home haemodialysis to CAPD. With CAPD he had an initial improvement in dyspnoea but later developed progressive tiredness. Hypoalbuminaemia was a major problem in spite of high protein intake and repeated intravenous plasma protein supplementation. His PD effluent fluid revealed an albumin concentration of 22–26 g/litre and this persistently high albumin loss in PD fluid in the absence of peritonitis was considered to result from long-standing congestive cardiac failure. In March 1985, during an admission for investigation of progressive tiredness, lethargy, low-grade pyrexia and weight loss (9 kg over the previous 8 months), a short early diastolic murmur was noted. Repeat echocardiogram revealed a tricuspid aortic valve with marked vegetations and left ventricular dilatation in addition to previously noted hypertrophy. Repeated blood cultures were negative. He died suddenly during an attack of pulmonary oedema.

Postmortem examination revealed a partially calcified tricuspid aortic valve, the valve cusps being covered

by large, crumbling vegetations which were sterile on culture (fig. 1). The other cardiac valves were unremarkable apart from a calcified mitral valve ring. There was no evidence of recent myocardial infarction.

To our knowledge, this is the first case of NBTE in a CAPD patient. NBTE is characterised by sterile, verru-

Fig. 1. Postmortem examination of heart showing marantic vegetations.

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cose, thrombotic vegetations adherent to the closure sites of the valve leaflets. The mitral valve is most commonly affected, followed by the aortic valve. It is usually associated with wasting

diseases including chronic tuberculosis, kwashiorkor, marasmus or malignancy, especially pancreatic, gastric or lung carcinoma. The suggested pathogenesis of NBTE includes platelet aggregation to injured endothelium with subsequent thrombus formation, and the relatively high association with malignant diseases may be due to the frequently increased coagulability of blood which occurs in these conditions [1, 2].

CAPD has been increasingly used in patients with end-stage renal failure aged over 50 years. These patients are more likely to have aortic valve sclerosis due to systemic hypertension and premature degenerative changes. Some patients on CAPD also suffer from persistent hypoalbuminaemia due to excessive loss of protein in PD fluid. According to Blumenkrantz et al. [3] an average of 8.8 g of protein is lost via CAPD daily in established CAPD patients. Our patient had an unusually high peritoneal protein loss in the absence of peritonitis and in spite of an 80-gram protein diet and intravenous plasma protein supplementation, he suffered from protein malnutrition.

NBTE is usually diagnosed at autopsy but is not necessarily preterminal. With echocardiography, antem-ortem diagnosis is possible and many of its complications such as embolism, valvular dysfunction or bacterial endocarditis are treatable.

This case illustrates a possibly increased risk of NBTE in CAPD patients suffering from protein malnutrition.

References

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