diagnostic manoeuvre. In this case full investigation produced no evidence of a recognised aetiology. A local tuberculoid reaction has been reported in association with malignant granulosa cell tumour of the ovary[1] and with a neurocytoma.[2] Localised tuberculoid granulomata in lymph nodes draining a neoplasm have been reported less frequently.[3-5] In none of these cases, however, were hepatic granulomata mentioned.

Granulomata may be produced by the deposition of toxic material; by cell-mediated immune reactions of a delayed hypersensitivity-type; or by the deposition of antigen-antibody complexes. Although Symmers has suggested that lymph node granulomata associated with carcinoma may represent a reaction to irritant lipoidal or lipoprotein material produced in the tissue drained,[5] definitive evidence is lacking. In this case the co-existence of localised lymph node granulomata and adenocarcinoma again suggests a cause-and-effect relationship. Probably also the hepatic granulomata represent a similar reaction.

I thank Dr D N Croft for his assistance and permission to publish this report.

[1] Schattenberg, H J, and Harris, W H, *American Journal of Pathology*, 1964, **22**, 539.
[2] Hartz, P H, and Van Der Sar, A, *American Journal of Clinical Pathology*, 1945, **15**, 473.
[3] Gherardi, G H, *Archives of Pathology*, 1950, **49**, 163.
[4] Nadel, E M, and Ackerman, L V, *American Journal of Clinical Pathology*, 1950, **20**, 952.
[5] Symmers, W St C, *American Journal of Pathology*, 1951, **27**, 493.

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Pseudomonas endocarditis treated surgically in a patient dependent on haemodialysis

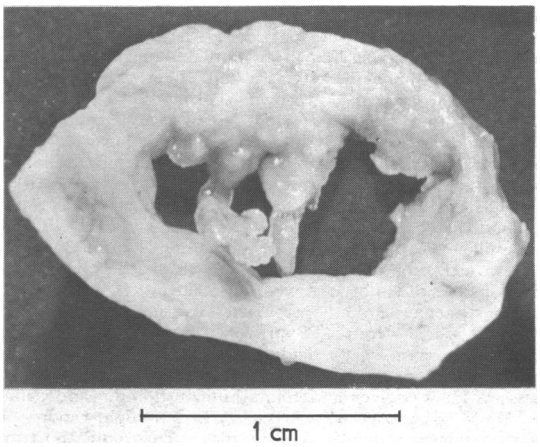
This case represents the only reported survival after aortic valve surgery for *Pseudomonas aeruginosa* endocarditis in a patient dependent on haemodialysis.

Case history

A woman aged 49 started on haemodialysis treatment in 1968. At that time only an innocent systolic ejection murmur was audible in the mitral and aortic areas. She was admitted to hospital on 1 August 1973 with rigors. Her temperature was 37·6 , the systolic murmur was unchanged, and there was no evidence of endocarditis. Septicaemia was diagnosed and treatment started with 500 mg cloxacillin 6-hourly and a single 80-mg dose of gentamicin. Subsequently *Ps aeruginosa* was isolated from her blood and also from the Kiil dialyser at her home. From 4 August to 9 October, when aortic valve reconstruction was performed, blood cultures repeatedly grew *Ps aeruginosa*. Many positive blood cultures were obtained while the patient was receiving gentamicin and tobramycin, when mean levels of over 5 μg/ml (peak level 12 μg/ml, trough level not below 2·4 μg/ml) were being maintained by giving 80 mg of either gentamicin or tobramycin intravenously at the end of each dialysis and additional doses between dialyses. Colistin 100 mg twice a day intravenously was given in addition to the aminoglycoside, but she remained feverish with positive blood cultures. Colistin was stopped for fear of its neurotoxic effects and tobramycin discontinued because of beginning bilateral deafness. Tobramycin was restarted five days later.

At this stage the prognosis was poor. She was pyrexial, increasingly breathless, weak, and apathetic. Though there was as yet no evidence of endocarditis she remained in heart failure. Her clotted arteriovenous fistula was explored as the possible source of her septicaemia but cultures were negative. On 19 September a faint aortic diastolic murmur was heard: the diagnosis of infective endocarditis was then made. She was then treated with colistin and sulphamethoxazole (2 g initially followed by 1 g twice daily), as these drugs are synergistic in vitro,[1] but her condition deteriorated, she developed an allergic rash, and the sulphonamide was stopped. Colistin was given alone and the dose increased until neurotoxicity developed. It was at this time that blood cultures became negative for the first time for many weeks. They remained negative thereafter, but that was not foreseen at the time and it was supposed that the organism was still active.

The patient's condition was rapidly worsening and clearly surgery was imperative. For it to be successful a maximum effort had to be made to clear the site of infection, since rapid destruction of aortic valve collagen might be taking place, as happens in eye infections. Carbenicillin 30 g intravenously, followed after 12 hours by 20 g and after 24 hours by another 20 g, was therefore given. Its sodium content, however, precipitated pulmonary oedema, which required urgent dialysis. At operation, performed by Mr L D Abrams (Queen Elizabeth Hospital, Birmingham) on 9 October, there was a large defect in the centre of the non-coronary cusp surrounded by small vegetations (see fig). The other two cusps were perfect and no other damage was noted. The non-coronary cusp was excised and a bicuspid transformation of the aortic valve was carried out.



Aortic valve cusp showing defect and vegetations.

Her postoperative course was stormy. Colistin was stopped after a week, and she remained apyrexial with negative blood cultures. Culture of the excised valve cusp was negative and histology showed only patchy fibrosis. Two years later she remains well with no evidence of aortic valve regurgitation.

Discussion

Pseudomonas endocarditis has a 50% mortality, the prognosis being worse in left-sided disease (aortic or mitral valves, or both, or left atrium).[2] Since the advent of antibiotics heart failure is the commonest cause of death in infective endocarditis,[3] and valvular surgery is imperative if there is progressive haemodynamic deterioration.[4 5] The gross pathology of endocarditis due to this organism lies between the proliferative, friable, acute type and the tougher vegetations of the subacute type due to streptococci, but in our case there was destruction of the valve.

Although the first of the continuing series of negative blood cultures began during colistin treatment we do not know whether colistin alone was responsible for sterilising the lesion or whether massive doses of carbenicillin contributed to it.

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[1] Russell, F E, *Journal of Clinical Pathology*, 1963, **16**, 362.
[2] Reyes, M P, *et al*, *Medicine*, 1973, **52**, 3, 193.
[3] Robinson, M J, and Ruedy, J, *American Journal of Medicine*, 1962, **32**, 922.
[4] Stason, W B, *et al*, *Circulation*, 1968, **38**, 514.
[5] Krestchmer, K P, and Lawrence, G H, *American Journal of Surgery*, 1969, **118**, 273.

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