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Ascending aortic false aneurysm following cannulation for perfusion

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Branchini, B., Zingone, B., and Vaccari, M. (1976). *Thorax*, **31**, 234–237. **Ascending aortic false aneurysm following cannulation for perfusion.** A case of false aneurysm originating from the ascending aortic cannulation site in the absence of mediastinal infection is described. Surgical treatment was carried out by means of limited cardiopulmonary bypass and hypothermic circulatory arrest, but the patient died early in the postoperative period. The technical failures responsible for the unsuccessful outcome are emphasized.

In most cardiac surgical centres the ascending aorta is the preferred site for arterial return from the pump-oxygenator following the initial suggestion of Nuñez and Bailey (1959) and DeWall and Levy (1963).

Cannulation of the ascending aorta is considered to be simple and expeditious and allows forward perfusion through large-bored cannulae; it is not hampered by aorto-iliac disease and is commonly said to carry a low complication rate (Flick *et al.*, 1971).

This report deals with a rare complication of the procedure, an aneurysm originating at the aortic cannulation site, and the fatal outcome of the case raises a few points which are worth noting.

CASE REPORT

A 54-year-old woman with a past history of rheumatic heart disease and a previous closed mitral valvotomy had had her mitral valve replaced six weeks previously at another hospital. At operation there had been problems because of friable tissues: the right ventricle was entered while pericardial adhesions were being dissected. Teflon was required to close the leak from the sutured ventricular wall. The mitral valve was replaced uneventfully, but Teflon felt was also needed to close the apical vent stab wound and the aortic cannulation site. The patient was eventually discharged after an uncomplicated postoperative course.

Four weeks after surgery a small lump appeared in the upper third of the sternotomy scar and steadily increased in size over the following days. The patient returned to hospital where an aortogram was performed; this demonstrated an aneurysm of the ascending aorta originating at the level of the cannulation site, feeding the superficial lesion through several fistulous tracts (Fig. 1). The patient was referred to our unit for surgical treatment.

On admission (12 December 1974) she was febrile (39°C) but had not had any fever over the previous weeks. The heart rate was 114/min. with a blood pressure of 130/70 mmHg. There were no signs of heart failure, and auscultation of the heart revealed normal prosthetic sounds and no murmurs. A dark-blue orange-sized mass in the upper third of the sternotomy scar (Fig. 2) exhibited an obvious pulsation synchronous with the radial pulse. An electrocardiogram and portable chest radiographs were not contributory. ESR was 77 mm/hour and WBC 10 100/mm^3, of which 82% were neutrophils; haemoglobin was 10·5 g/dl.

Vitamin K_1 and fresh frozen plasma were administered, together with an 80 mg dose of heparin, while emergency surgery was being arranged. Blood cultures were later reported to be sterile.

The operation started some five hours after admission, with the plan to cool the patient down to 25°C, arrest the circulation, enter the chest, cross-clamp the aorta above the aneurysm, and close the defect in the aortic wall while rewarming on bypass.

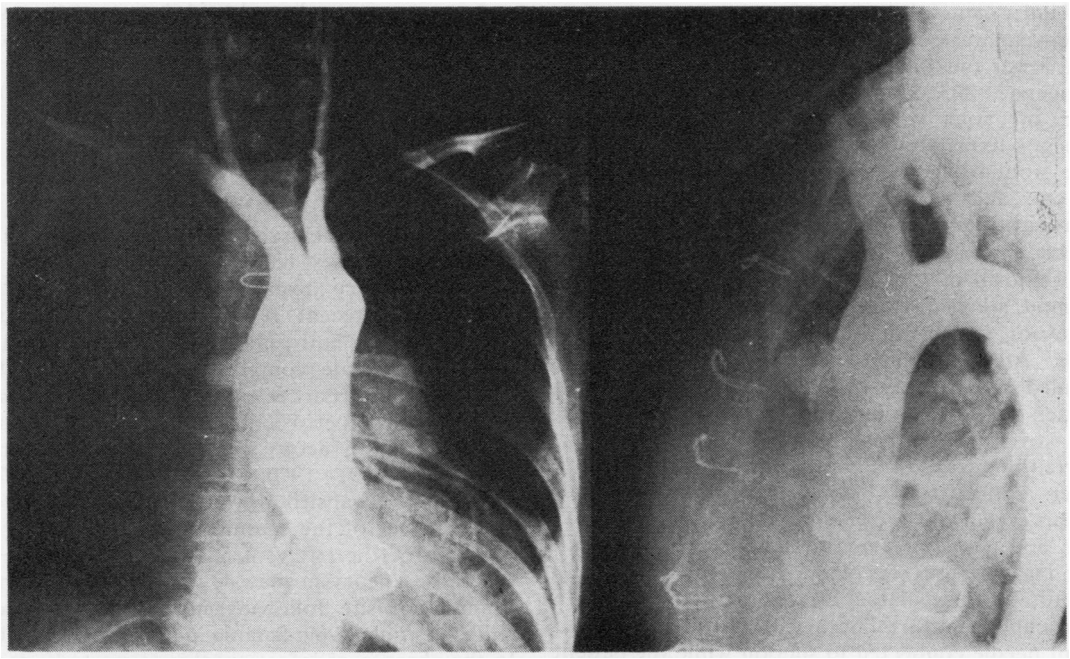

FIG. 1. *Aortogram demonstrating ascending aortic aneurysm; note the lack of correlation with Fig. 2, due to clots within the aneurysm.*

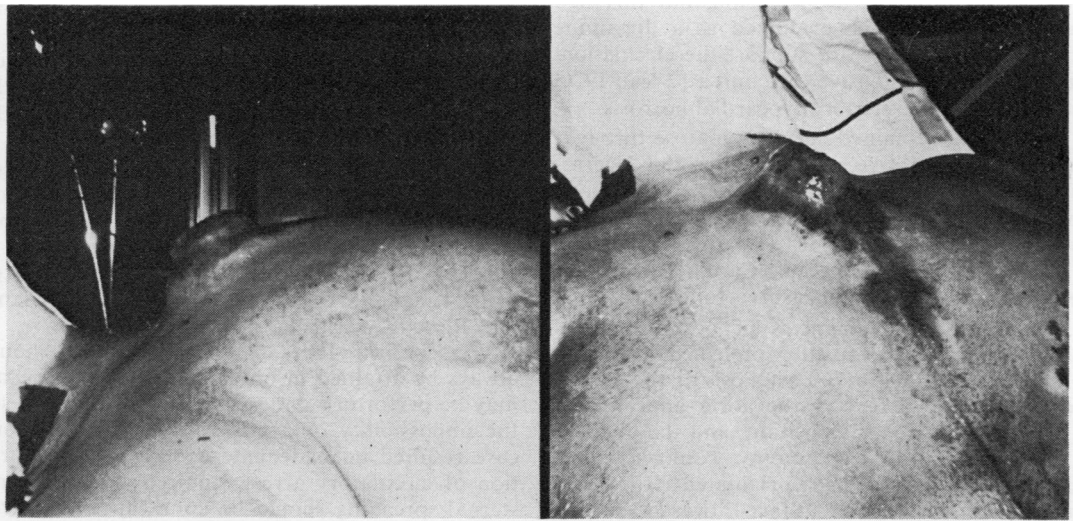

FIG. 2. *External appearance of the aneurysm.*

Under general anaesthesia, the femoral artery and vein were cannulated in the right groin, and the lower two-thirds of the sternotomy scar were reopened. At 25°C the pump was turned off as the aneurysm was entered: owing to firm adhesions fixing the right ventricle to the sternum, the wound edges could be spread only for about 6 cm, and a tear was produced in the right coronary artery stretched between the sternal edges.

Removal of the clots in the upper half of the wound allowed visualization of a defect of the anterior wall of the aorta about 2·5×3·5 cm in size. Adhesions and inadequate exposure precluded aortic cross-clamping above the defect, which was far too large to permit direct closure. A woven Dacron patch graft was therefore inserted by means of a continuous suture buttressed over Teflon felt; blood was occasionally administered from the pump to maintain a level in the aorta and prevent air embolism. Cardiopulmonary bypass was then reinstituted after 33 minutes of circulatory arrest, venting the aorta through the suture line first, then through an air vent needle connected to suction while the patient was kept head-down. The heart resumed a good spontaneous contraction during the rewarming phase, and the tear in the right coronary artery was repaired with multiple 6/0 Prolene sutures. Cardiopulmonary bypass was discontinued at 34°C, and the sternal edges were freshened and closed over a single drain following heparin neutralization.

The pupils were noted to be dilated shortly after termination of the perfusion, but this sign could not properly be evaluated as a dopamine drip was being infused to support the circulation. On return to the intensive care unit a 12-lead ECG showed signs of posterior myocardial necrosis.

The patient remained deeply comatose throughout the postoperative period, and serial electroencephalographic studies confirmed diffuse and severe brain damage. Some 40 hours after the end of the operation the ECG showed extension of the myocardial infarction. Cardiac arrest unresponsive to standard measures followed.

At necropsy the diagnosis of posterior myocardial infarction was readily confirmed. The right coronary artery had a 70% stenosis at the site of the repair. The defect in the aorta appeared to be soundly repaired; its position, and the absence of any other sutured aortotomy, confirmed that the false aneurysm had originated from the cannulation site. The Starr-Edwards mitral prosthesis was correctly placed.

An extensive subarachnoid haemorrhage was also found, but there were no signs of a focal brain lesion.

Histological studies failed to reveal any cause of the poor quality tissue encountered at the first operation.

COMMENT

Cannulation of the ascending aorta instead of the peripheral arteries has increased the safety of cardiopulmonary bypass (Flick *et al.*, 1971). Nevertheless recent reports indicate that fatal complications, both immediate and late, may occur. Immediate complications include carotid hypoperfusion (Parker, 1969; Magilligan *et al.*, 1972) or hyperperfusion (Krous, Mansfield, and Sauvage, 1973), acute aortic dissection (Salama and Blesovsky, 1970; Reinke *et al.*, 1974; Williams, Suwansirikul, and Engelman, 1974), and entrance of the cannula into one of the arch branches or the left ventricle (Magner, 1971).

A false aneurysm may develop from the aortic cannulation site following mediastinal infection (Lillehei *et al.*, 1969; Salama and Blesovsky, 1970). In the absence of infection, this seems to be most uncommon, and only one more case was found in the literature (Flick *et al.*, 1971) in which the aetiology of the false aneurysm remained unknown. A sound explanation is missing for the friability of the tissues and the difficulty of haemostasis during the previous operation in our patient.

The situation that the surgeon is faced with is a most challenging one, whatever the cause of the aneurysm. Success depends on several factors, two of which were not adequately considered in our case; these were the impossibility of dissecting the aorta in order to clamp it, and the injudicious sternal spreading which damaged the right coronary artery.

The technique to correct this complicated pathology, employing limited cardiopulmonary bypass and circulatory arrest under deep hypothermia, has proved to be of value according to Lillehei and associates (1969), who first reported using it, and to Flick *et al.* (1971) and Salama and Blesovsky (1970).

We confirm that deep hypothermia should always be attained in order that the whole repair may be performed under arrest; failure to foresee the impossibility of aortic cross-clamping in our case resulted in inadequate cooling for the duration of circulatory arrest required. Furthermore, sternal spreading should be done cautiously *after* turning off the pump and exsanguinating the

patient into the oxygenator: only a bloodless field can prevent damage to the underlying cardiac structures which may be, as they were in our case, soundly adherent to the posterior aspect of the sternum.

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SURGICAL EDITOR

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