Case reports

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Left atrial tamponade
Report of a case after right heart catheterization

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Left atrial tamponade after right heart catheterization is described. It was associated with a low cardiac output but without signs of generalized cardiac tamponade. Failure to remove the cause of obstruction resulted in irreversible cerebral damage.

Left atrial tamponade after heart operations has been described by Yacoub, Cleland, and Deal (1966). They reported 2 patients whose condition deteriorated suddenly some hours after operation, with signs of a low cardiac output but without evidence of generalized cardiac tamponade. After thoracotomy a clot was evacuated from behind the left atrium with rapid recovery in one patient but without effect in the other.

In the patient to be described, myocardial perforation occurred during cardiac catheterization and resulted in left atrial tamponade. Though full myocardial recovery was achieved, cerebral damage was irreversible.

Clinical history

An obese man aged 58, who was known to be hypertensive and in atrial fibrillation before operation, had a left meniscectomy performed under general anaesthesia in November 1970. Six days after operation he developed a painful, swollen, and tender left calf. Ascending venography showed a deep vein thrombosis extending into the left popliteal vein. Twenty-four hours later the patient complained of left-sided pleuritic chest pain associated with diminished breath sounds on that side, but without radiological changes. The patient was ill, but his blood pressure remained at 180/100 mmHg without gallop rhythm or an increase of the jugular venous pressure. A tentative diagnosis of pulmonary embolism was made.

At right heart catheterization for pulmonary angiography, a No. 8 NIH angiocatheter was passed into the heart via a left antebrachial vein with difficulty due to venospasm. While attempting to pass the catheter into the main pulmonary artery, the right ventricular trace changed to a lower pressure. Injection of 0·5 ml 65 per cent Hypaque showed contrast in the pericardial sac. The catheter was withdrawn. Five minutes later, the patient complained of severe pain between the shoulder blades after which he vomited. He became shocked, with profuse sweating. The radial pulses were absent and the left carotid and subclavian pulses were reduced but both femoral pulses were palpable. The cardiac silhouette was not enlarged by comparison with chest *x*-rays taken at the beginning of the procedure, nor had the electrocardiogram changed. After 15 minutes the jugular venous pressure rose, at which time only 50 ml blood-stained fluid could be aspirated by sub-xiphisternal pericardial tap. The patient continued to deteriorate and asystole occurred. External cardiac massage did not restore an adequate circulation and therefore the chest was opened through the left fourth interspace. Intracardiac isoprenaline resulted in ventricular tachycardia but intrathoracic cardiac massage failed to restore an adequate cardiac output. The pericardium was opened but no blood was apparent in the pericardial sac. On further exploration about 200 ml clotted blood was found behind the left atrium. This was removed with an improvement in left ventricular filling, a reversion to the precatheter rhythm, and a return of the peripheral pulses. Normal right atrial and ventricular pressure traces were now noted. Though cardiac output remained satisfactory without supportive therapy for the next 20 hours, the patient did not resume consciousness and required artificial ventilation. At that time the heart stopped in asystole from which the patient could not be resuscitated.

At necropsy there was fibrinous pericarditis. No sign of the puncture made by the catheter could be found. The heart weighed 578 g due to hypertrophy of the left ventricular wall. There was an old fibrous infarction in the posterior upper part of the septum and the myocardium generally was pale and mottled with small haemorrhages. The right coronary artery was occluded

by a fibrinous red thrombus adhering to the wall of the artery within 5 cm of its origin, the remainder showing slight atheroma. There was a similar red thrombus in the anterior descending branch of the left coronary artery near the ventricular apex. The lungs were partially collapsed and both were deeply congested and oedematous. The pulmonary artery to the left lower lobe was occluded by embolus.

Discussion

In retrospect it is probable that the catheter had entered the coronary sinus and not the outflow tract of the right ventricle. It is well known (Oram, 1971) that a right ventricular pressure trace can be obtained from a catheter in the coronary sinus. If this is not appreciated manipulation and attempted passage into the main pulmonary artery in this situation may lead to perforation of the coronary sinus. In our case, at necropsy, the myocardium was so haemorrhagic that it was not possible to find evidence of perforation.

The heart is fixed posteriorly by the pericardial attachments forming the oblique sinus. This *cul-de-sac* is formed by parietal and visceral pericardium meeting at the entry of the pulmonary veins superiorly and laterally where it reflects onto the posterior wall of the left atrium. A localized collection of blood in this situation may embarrass left atrial filling, with a subsequent fall in cardiac output. In these circumstances, the jugular venous pressure would not rise until later, being associated with right ventricular failure and not generalized tamponade. This occurred in our patient and caused irreversible cerebral damage.

Left atrial tamponade should be suspected if signs of a persistently low cardiac output occur during catheterization, in the absence of a raised jugular venous pressure, and in a situation where myocardial perforation might have occurred. The only effective treatment is surgical removal of the clot.

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References

Oram, S. (1971). *Clinical Heart Disease*. William Heinemann Medical Books, London.

Yacoub, M. H., Cleland, W. P., and Deal, C. W. (1966). Left atrial tamponade. *Thorax*, **21**, 305.

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