Severe hypotension due to potassium-induced pericardial injury

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The occurrence of iatrogenic pericarditis after the administration of drugs such as procaine amide, hydralazine, and phenylbutazone and traumatic pericarditis has been previously reported.^{1,2} We report a case in which potassium chloride was accidentally injected intrapericardially, leading to acute pericardial irritation.

Case report

A 59-year-old man underwent coronary artery bypass grafting for disabling angina pectoris. The operation was uneventful. Before the pericardium was closed a direct right atrial line was introduced. After operation blood loss from the drains was minimal and the patient's clinical condition was stable. An electrocardiogram (ECG) showed minor non-specific ST-T changes. There were no signs of infarction. The laboratory results and postoperative chest radiograph were normal. The right atrial line was used intermittently for potassium administration (20 mmol potassium chloride in 60 ml 5% dextrose in water) to control occasional ventricular extrasystoles. Two hours after operation the patient complained of a sudden constricting and severe retrosternal pain and breathlessness. An ECG monitor strip showed ST segment elevation. The pain gradually subsided and a full 12-lead ECG then showed slight ST elevation in leads II, III, aVF and aVL without reciprocal ST depression. One and a half hours later the patient had a further attack of pain, this time more incapacitating. He was now having nitroglycerine sublingually and intravenously. Again the ECG strip showed ST-segment elevation, now associated with horizontal depression of the PR-(PTa) segment (fig A). The pain continued to increase and the patient developed both pronounced tachypnoea and bradycardia. The blood pressure fell rapidly to a mean of 25 mm Hg. At this time the mean right atrial and mean pulmonary artery wedge pressures were both 20 mm Hg. Frequent ventricular extrasystoles appeared. The patient was paced via the epicardial leads with an external pacemaker and was intubated and ventilated with 100% oxygen. Ephedrine chloride, calcium chloride and dopamine were given via the right atrial line and the right atrial opening of a Swan-Ganz catheter. There was no real improvement. At this time it was noted that no blood could be aspirated from the right atrial line, but that a few millilitres of serohaemorrhagic fluid were

aspirated. The fluid being given through this line was immediately stopped. After a few minutes the mean arterial pressure rose to 110 mm Hg and the ECG improved (fig B). After an hour the patient was stable and symptom free. The rest of the postoperative course was uncomplicated. No abnormal serum enzyme pattern was seen. The patient was discharged fit and well on the 12th postoperative day.

Discussion

Retrospectively we established that the two episodes of pain and cardiovascular disturbance coincided with two periods of administration of concentrated potassium chloride via the right atrial line. Potassium had been given half an hour after the patient arrived in the intensive care unit through the same line. The patient then had shown no abnormal reaction, presumably because the line was still properly positioned at that time. It must have been displaced into the pericardial sac between half an hour and two hours after operation when potassium was again administered, this time with the symptoms and signs described. During the administration of potassium chloride the ECG showed ST-segment elevation. It returned rapidly to normal at the end of the first infusion and when it was stopped the second time.

In the differential diagnosis of the episodes acute cardiac tamponade, angina pectoris, and myocardial infarction had been considered. Cardiac tamponade could be ruled out because the drains were functioning well and the evidence of the subsequent clinical course, the ECG, and the serum enzymes were against myocardial infarction. Potassium chloride is known to be extremely irritant even in fairly dilute concentrations. Probably the potassium chloride here had a strongly irritant effect on the pericardium and epicardium. The clinical picture was indeed one of acute pericardial and epicardial irritation. Pain, dyspnoea, hypotension, and disturbances of rhythm are prominent in acute irritation of the pericardium and epicardium. Pain is one of the most important symptoms of acute pericardial irritation. The pattern of pain is very variable.^{3,4}

The rapid development of hypotension, bradycardia, and ventricular extrasystoles was attributed to vagal stimulation resulting from the pain, with a possible further element due to direct effects of the potassium chloride on the epicardium and subepicardial layers of the myocardium.^{1,3-5} Acute ischaemic posterior wall injury cannot, however, be excluded with certainty and if sufficiently extensive could also account for all the symptoms and signs

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Bipolar monitoring lead showing the ECG during the second episode of severe chest pain and hypotension (A) and after disappearance of symptoms and signs (B). In A the arrows point to typical horizontal depression of the PR-(PTa) segment, which is associated with maintenance of the intrinsicoid deflection of the QRS complex and upward concave elevation of the ST segment. The discordant displacement of PR and ST segments creates the impression of pronounced ST segment elevation. In B the PR and ST segments have returned to normal.

observed in this patient. Such an ischaemic injury could have been due to spasm of the right or circumflex coronary artery, secondary to the irritating effects of the local administration of potassium chloride. In our opinion, however, the discordant horizontal depression of the PR-(PTa) segment is a strong argument in favour of non-ischaemic, inflammatory irritation of the subepicardial layers of the myocardium. This finding has been reported as a specific sign of pericarditis,^{6,7} and it is not usually seen in otherwise uncomplicated ischaemic myocardial injury.

When a 12-lead ECG is available the ST changes are seen in most of the leads. There is no reciprocal ST depression except in lead aVR. The intrinsicoid deflection of the QRS complex mainly remains. The ST elevation is ascribed to myocardial changes under the affected pericardium. These changes disappear as soon as the inflammation disappears.^{1,4,6-8}

A practical point from this case is that use of a right atrial line for intravenous treatment is unsafe if there is no regular check that the line is properly in position (easy withdrawal of blood with a three-way tap and syringe). An apparently adequate fixation of the line at surgery and an apparent right atrial curve on the pressure trace are not

adequate insurance that the catheter is still in the atrium.

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