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CASE REPORT

Colon ischaemia and necrosis as a complication of prolonged but successful CPR[☆]

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Summary Survival after prolonged cardiopulmonary resuscitation (CPR) is often associated with neurological and other sequelae. We describe a patient who survived prolonged cardiac arrest due to ventricular fibrillation neurologically intact but suffered colon ischaemia and necrosis in the post-resuscitation period. Subtotal colectomy was performed. We wonder whether this complication was related to the use of vasopressin.

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Introduction

Sudden cardiac arrest occurs with an incidence of 1/100,000 in the general population. Successful treatment depends on the circumstances and the prompt initiation of cardiopulmonary resuscitation (CPR). Recovery after CPR with good neurological outcome is achieved in only 5–20% of patients.¹ We describe the successful neurological outcome after prolonged CPR in a 38 year old woman who suffered sudden cardiac death (SCD) secondary to ventricular fibrillation (VF). The post-resuscitation period

was complicated by colon ischaemia and necrosis, necessitating subtotal colectomy. This complication has not previously been reported. We wonder whether the use of vasopressin, introduced as an option into ACLS algorithms since 2000² might have contributed to this complication.

Case report

A 38 year old woman suddenly collapsed on the dance floor. She had no medical history, was not on any medication and had no allergies. She was a non-smoker and had no history of drug abuse. A nephew had died age 18 of sudden cardiac death while biking. She had no prodromal features. When she collapsed (approximately 01.21 h), she

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was unconscious, apnoeic and pulseless. A cardiology nurse who was present immediately started bystander CPR. The emergency system was activated (01.23 h, recorded times (EMS-System)) and an ambulance and a medical intervention team with an emergency nurse and an anaesthesiologist (S.B.) arrived at the scene at 01.35 h. The presenting rhythm was VF. Advanced life support was initiated according to standard algorithms. Three monophasic shocks (200–200–360 J) were delivered. An IV line was placed and the trachea was intubated. External chest compressions and bag ventilation on 100% oxygen was continued throughout. Drug therapy consisted of adrenaline (epinephrine) (total of 8 mg), amiodarone 300 mg and saline. Twelve more shocks were administered (360 J), but the rhythm remained VF. A further 300 mg of amiodarone was administered. Fifty minutes after the collapse and after a total of 15 shocks, the patient continued to be in refractory VF. The anaesthesiologist on the scene decided to transport the patient to our emergency department (ED) while continuing external chest compressions and ventilation, leaving the scene at 02.09 AM.

At arrival at our ED (02.17 h) she was still in cardiac arrest and the rhythm was VF. External chest compressions and mechanical ventilation were continued. A further 150 mg of amiodarone was administered. 40 IU of vasopressin (Pitressin®) was administered (02.25 h). At 02.45 h, after 4 more shocks, spontaneous circulation resumed, i.e. 84 min after collapse. The patient was in sinus rhythm with a blood pressure of 100/60 mmHg. A continuous infusion of dopamine (3 mcg/kg/min) and noradrenaline (0.1 mcg/kg/min) was started to support blood pressure. The ECG was not suggestive of myocardial infarction, nor were cardiac enzymes elevated. C-reactive protein level was not elevated. On transthoracic echocardiography (T.T.E.), marked dilatation of the right ventricle was noted. Because arterial PaO₂ was 50 mmHg on 100% oxygen, a CAT-scan of the thorax was performed. The scan did not show lung contusion or pulmonary embolism, but showed a large subcapsular liver haematoma, probably a complication of prolonged external chest compressions.

Intensive therapy was continued on the Coronary Care Unit (CCU). The patient remained stable during the first hours. In the morning, she was clearly awake and fully responded to commands. However, in the next 24 h, she developed oliguria and acute renal insufficiency. Good left and right ventricular function, no regional wall motion abnormalities and normal heart valves were seen on T.T.E. She developed marked distension and tenderness of the abdomen, with no drop in

haemoglobin concentration. Doses of dopamine and noradrenaline had to be increased. An abdominal CAT scan 30 h after admission suggested colon ischaemia and an exploratory laparotomy was performed. Subtotal colectomy of the ascending, transverse and descending colon was necessary because of ischaemia and necrosis. The sigmoid and rectum were intact and spared. An ileostomy was placed and the subcapsular liver haematoma drained. Pathological examination revealed extensive ischaemic-hemorrhagic necrosis, mainly of the inner layers from the ileo-caecal valve up to the last 30 cm of the resected specimen. Signs of focal peritonitis were noted. There was no evidence of arterial thrombosis, vasculitis or other pre-existing disorders.

The patient remained on the CCU for 6 weeks. After 3 weeks of renal replacement therapy, renal function recovered completely. She remained on ventilator therapy for 30 days after tracheotomy on day 20, and had severe gastroparesis for 4 weeks.

She was investigated for underlying cardiac disease (day 30). Trans oesophageal echocardiography and coronary angiogram were normal. During the electrophysiological study (performed under the effect of amiodarone) only non-sustained multiform ventricular tachycardia was inducible. No specific cardiac aetiology for the SCD syndrome could be identified. An automated implantable cardioverter defibrillator (AICD) was implanted.

On day 45, she left the CCU, neurologically fully intact, except for very mild neuro-cognitive disturbances, mainly affecting short term memory. She left the hospital on day 80, and resumed all daily household activities. She returned for bowel re-anastomosis and remains well at home.

Discussion

We report a case of colon ischaemia and necrosis after prolonged CPR with good neurological recovery. An extensive Medline search did not reveal previous reports of colonic ischaemia and necrosis after prolonged and successful CPR, and to our knowledge this is the first such report. Cases of colon ischaemia after haemorrhagic shock have been reported,³ and a case of ischaemic colitis after the use of glypressin for bleeding oesophageal varices has been reported.⁴

The success of CPR depends on the timely initiation of four actions, commonly described in the chain of survival. This consist of: the early recognition of cardiac arrest and the early activation of the emergency medical systems, the early initiation of basic life support, early defibrillation and

the timely initiation of advanced life support.² In general good outcome after sudden cardiac arrest is only achieved in 5–20% of cases. In cases of prolonged CPR, outcome is even worse.

In this case, all four interventions were very efficiently achieved, contributing to the good outcome. ACLS was administered according to the Guidelines 2000,² but VF could not be reversed before the administration of vasopressin. The interaction of vasopressin with V1 receptors causes intense vasoconstriction of the vessels to the skin, skeletal muscle, intestine and the fat with relatively less constriction of the coronary and renal vascular beds and vasodilatation of the cerebral vasculature.⁵ In a swine model, vasopressin has been shown to increase left anterior descending coronary blood flow during extremely low flow conditions on coronary bypass without affecting coronary vascular resistance.⁶ The use of vasopressin, 64 min after the start of CPR, might have increased perfusion pressure to the coronary arteries leading to successful defibrillation and increased oxygen delivery to the brain, preserving neurological function.

Since vasopressin is a potent vasoconstrictor, vasoconstriction in the intestinal arteries may result in poor tissue perfusion and colon ischaemia and necrosis.⁷ Splanchnic blood flow was reduced after the use of vasopressin during CPR in one porcine model⁸ although in another model splanchnic blood flow in the epinephrine resuscitated and the vasopressin resuscitated group was similar.⁹

The possibility that colon ischaemia was present prior to the onset of cardiac arrest seems very unlikely since this patient had no medical history and was asymptomatic until the moment of collapse. Also, C-reactive protein on admission was not elevated. If low flow and hypoperfusion during CPR in itself is the cause of this complication, we would expect this complication to have been reported before. Another explanation could be the occurrence of an arterial embolus, related to atrial fibrillation or the in situ formation of a thrombus during CPR. However, there is no evidence that this patient had atrial fibrillation before the collapse and she never had it afterwards.

Another factor leading to the complication might be the concomitant use of adrenaline and vasopressin, leading to an exaggerated vasoconstrictive response. Although current ACLS guidelines 2000 do not recommend the combined use of these agents, we used vasopressin after previous therapy, including the recommended doses of adrenaline, remained unsuccessful. On the other hand, this patient could only be successfully defibrillated *after* the administration of vasopressin, leading to

the possibility that she would not have survived without vasopressin, as has been suggested in previously published case reports of prolonged CPR.¹⁰

Since the doses of noradrenaline administered subsequently on the CCU were rather low, we suspect that the colon ischaemia and necrosis was not a complication of the use of noradrenaline.

Thus, it remains suggestive but speculative that the colon ischaemia and necrosis was a complication of the administration of vasopressin itself.

Conclusion

In conclusion, we report the occurrence of colon ischaemia and necrosis requiring subtotal colectomy after prolonged CPR with good neurological outcome in a patient with cardiac arrest. We speculate that the complication might be associated with the use of vasopressin.

Conflict of interest

None.

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