

Life-threatening intestinal ischaemia and necrosis in a patient with cardiac arrest and atrial fibrillation

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Abstract

A 68-year old man was successfully resuscitated after sudden cardiac arrest. Following this event, the patient developed intestinal ischaemia and necrosis requiring surgical treatment. Our case report investigates the cause of the intestinal ischaemia and warns physicians to be aware of this rare but life-threatening condition.

Keywords

Cardiac arrest; superior mesenteric artery occlusion; intestinal ischaemia.

Introduction

Sudden cardiac arrest is a life-threatening condition causing death within minutes if not immediately and properly treated. It can be divided into out-of-hospital cardiac arrest and in-hospital cardiac arrest, with survival rates of 9.5% and 23.9% in adults, respectively, which shows that this condition, even if treated immediately and in a hospital with trained medical personnel, has a low survival rate^[1]. Patients who survive this condition are also at risk of neurological and other severe complications.

In our case, the patient was successfully resuscitated and had spontaneous circulation within a few minutes after the cardiac arrest, but he then had unexplained abdominal symptoms, which required an urgent exploratory laparotomy to resolve.

Case presentation

A 68-year old man with known chronic coronary artery disease, a successful coronary bypass operation and femorofemoral crossover grafting 10 years previously, had a sudden cardiac arrest while out hunting, with no sentinel chest pain or other cardiac symptoms immediately before the event. A paramedic at the scene initiated immediate cardiopulmonary resuscitation (CPR) and 2 min later an ambulance with a physician arrived at the scene and took over the CPR. The heart monitor revealed ventricular fibrillation and one shock of 360 J was administered, converting the

patient's heart rhythm from ventricular fibrillation into atrial fibrillation with a ventricular rate of 114 beats/min. The electrocardiogram (ECG) revealed ST segment depression in lead V2-V6.

Following this the patient had a spontaneous pulse and circulation and the time to return of spontaneous circulation was only 15 min. The patient was thereafter awake, fully oriented, scored 15 on the Glasgow Coma Scale and his blood pressure was 161/80 mmHg. The patient's only complaint was chest pain accentuated by respiration, which was considered the result of possible rib fractures following the chest compressions during the CPR. The pain was relieved by low doses of intravenous morphine.

The patient was subsequently transferred to the Department of Cardiology at the Regional University Hospital and underwent coronary arteriography, which was normal without any newly significant coronary artery stenosis, and this result could therefore not explain the patient's sudden cardiac arrest. Primary cardiac dysrhythmia was therefore suspected as the cause of the cardiac arrest.

The patient was subsequently transferred to the cardiology ward, where he was carefully questioned and examined by the ward doctor. The patient only complained of pain from his ribs, and no other symptoms were present at that time. The ECG revealed atrial fibrillation with a ventricular rate of 100 beats/min, no ST segment changes and a normal QT interval. The patient had no previous history of atrial fibrillation and had no cardiac-related symptoms before the cardiac arrest, which made it difficult to estimate when the atrial fibrillation had begun. Bedside echocardiography revealed a near normal ejection fraction, no valvular abnormalities and no pericardial effusion. There was a pre-existing hypokinetic area in the inferolateral segments known from earlier echocardiography 10 years previously after the coronary bypass operation.

The first troponin I measurement taken at 17:30 h was 506 ng/l (reference level <25 ng/l), and the second troponin I measurement taken at 22:00 h was 1124 ng/l. Electrolytes (sodium, potassium and calcium) were all found to be in the normal range; the patient's creatinine measurement and blood sugar levels were also within the normal range. Blood pressure was 128/65 mmHg, pulse 80 beats/min and temperature 37.0°C. A chest radiograph revealed two rib fractures but no signs of pneumothorax or pleural effusion.

The patient complained of upper abdominal pain for the first time at 20:30 h. Physical examination carried out by the ward doctor revealed diffuse tenderness in the abdomen but no guarding, rebound or rigidity. The pain was relieved by low doses of intravenous morphine and was considered to be a result of the chest compressions during the CPR earlier that day.

In the following hours the pain worsened and the patient had abdominal cramps and vomiting. An abdominal surgeon was informed but did not suspect any serious condition and also considered this to be a result of the chest compressions and rib fractures.

The patient was needing intravenous morphine in progressively higher doses to relieve the pain, which was now more localized in the right upper part of the abdomen. At 23:30 h, arterial blood gas analysis revealed mild metabolic acidosis (Table 1). The ward doctor requested a contrast-enhanced computer tomography (CT) of the patient's abdomen. This showed an abdominal aortic aneurysm 4.1 cm in diameter with mural thrombus, but no signs of aortic dissection (Fig. 1). It also revealed ischaemia of the ascending colon, several blood clots inside the superior mesenteric artery (SMA) (Fig. 2), and several atherosclerotic plaques in the proximal part of the SMA and coeliac trunk not causing significant stenosis.

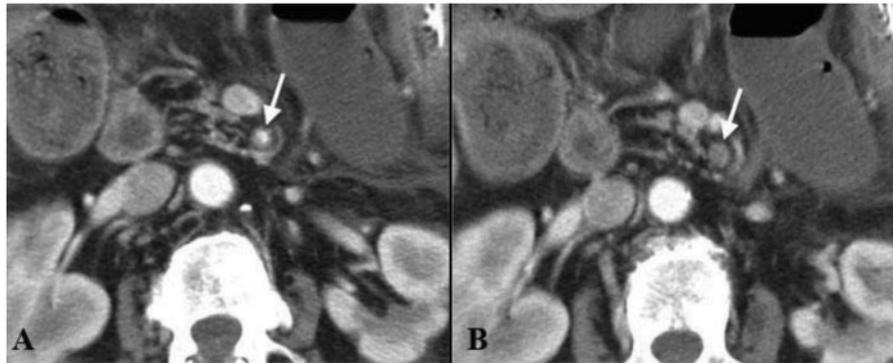
As the scan also suggested intestinal ischaemia consistent with the patient's abdominal symptoms, exploratory laparotomy was performed and subtotal colectomy of the ascending colon was necessary because of ischaemia and necrosis. The transverse and descending colon were both spared and an ileostomy was fashioned.

Following this event, the patient recovered and during hospitalization there were several episodes of non-sustained ventricular tachycardia on continuous heart rate monitoring without concomitant clinical symptoms.

Two weeks later the patient was discharged with an automated implantable cardioverter defibrillator. The ECG on the day of discharge revealed sinus rhythm but the patient was discharged on a treatment dose of the novel oral anticoagulant agent dabigatran and 100 mg metoprolol daily according to the national Danish guidelines^[2]. At follow-up 2 months later, the patient was feeling well and had not experienced any cardiac or gastrointestinal symptoms since discharge. A vascular surgical opinion on the abdominal aortic aneurysm was sought and conservative management with annual surveillance ultrasonography was recommended.

Table 1. The patient's blood gas analysis taken at 23:30h on the day of hospitalization

	Patient's result	Reference interval
pH	7.33	7.35-7.45
P _a O ₂	7.10 kPa	11 - 13 kPa
P _a CO ₂	4.85 kPa	4.7-6.0
Base excess	-6 mmol/L	-2 to +2 mmol/L
Lactic acid	19.6 mEq/L	22-26 mEq/L
	1 mmol/L	0.5-2 mmol/L

**Fig. 1.** Contrast-enhanced abdominal CT of the patient in our case revealing an aortic aneurysm with a mural thrombus. There is no sign of aortic dissection.**Fig. 2.** Contrast-enhanced abdominal CT revealing the superior mesenteric artery (A) and the same almost occluded (B).

Discussion

Intestinal ischaemia may be caused by an acute reduction of the arterial blood supply to the intestines either due to embolism, thrombosis, poor cardiac output, blockage of the venous drainage or by a combination of two or several of these factors. Mesenteric embolism accounts for 25-30% of patients with intestinal ischaemia, and atrial fibrillation represents an important cause of embolism^[3].

The presence of synchronous embolism and/or atrial fibrillation supports this diagnosis as a cause of the arterial occlusion. Thrombotic occlusions are more proximally located than embolic occlusions, result in more extensive intestinal infarction and are related to aortic wall thrombosis

or simply occlusion of the vessel secondary to narrowing^[4]. Patients with thrombosis of the SMA are more likely to have a history of abdominal angina and the onset of symptoms is rarely sudden and dramatic as with an embolism. Embolisms tend to lodge just beyond the origin of the middle colic artery, where the calibre of the SMA narrows rapidly and ischaemia therefore spares the upper jejunum and transverse colon^[5].

There are few case reports in the literature regarding ischaemia of the intestines in patients who survive cardiac arrest^[6,7]. However in these cases, the intestinal ischaemia is a result of low blood flow within the visceral circulation due to hypotension, and not related to thrombotic or embolic occlusion of the SMA as suspected in our case report. Furthermore, these patients received vasoconstricting agents such as noradrenalin or vasopressin; our patient did not receive any vasoconstricting agent.

Iraklis et al.^[6] report a case of an 83-year old woman who had sudden in-hospital cardiac arrest, and following successful CPR was transferred intubated to the intensive care unit, where she was treated with the vasoconstricting agent norepinephrine to support her circulation. Several hours later she developed increasing abdominal distension and severe metabolic acidosis. Multidetector computer tomography (MDCT) revealed intestinal ischaemia and a subsequent exploratory laparotomy revealed ischaemia of the terminal ileum and extensive colonic necrosis. The scan did not show any endoluminal defect of mesenteric arteries or veins. The cause of the ischaemia in this case is thought to be the low cardiac output and hypotension despite the successful CPR.

Stockman et al.^[7] report a 38-year old woman who survived cardiac arrest following prolonged CPR but had ischaemia and necrosis of the colon, requiring subtotal colectomy of the ascending, transverse and descending colon. Pathological examination excluded arterial thrombosis as the cause. In this case, the patient had no spontaneous circulation for 84 min after her collapse, which might have contributed to a long period of hypotension and reduced blood flow in the visceral circulation leading to ischaemia of the intestines. Furthermore, the patient was treated with a vasopressin, which contributed to vasoconstriction of the intestinal arteries, poor tissue perfusion, ischaemia of the colon and necrosis^[8].

A case where the cause of the intestinal ischaemia is related to embolism of the SMA is presented by Han et al.^[9] who report on a 43-year old man admitted to the hospital 4 h after the onset of nausea, vomiting and diarrhoea. Contrast-enhanced CT and angiography revealed occlusion of the SMA.

The patient in our case developed abdominal symptoms almost 4 h after his cardiac arrest, which was faster than the patients in the cases where hypotension was the suspected cause of the intestinal ischaemia^[6,7] and similar to other cases where embolism was the suspected cause^[5,9]. Furthermore, the contrast-enhanced abdominal CT of our patient revealed several endoluminal defects of the SMA suggesting blockage of this artery by either thrombosis or embolism. CPR was initiated immediately and the patient had spontaneous circulation 15 min after collapse when the patient's blood pressure was 161/80 mmHg. Therefore we do not believe that the intestinal ischaemia in our patient was related to hypotension during the cardiac arrest and the period of CPR.

The most likely cause of the intestinal ischaemia in our patient is blockage of the SMA during the first hours of hospitalization, most likely due to an embolism. Atrial fibrillation was the predominant rhythm on the ECG following successful resuscitation, and continued to be so during the first few days of hospitalization. This is the most likely cause of the embolism. According to other studies in the literature^[4], another source of embolism could be an aortic thrombus. Contrast-enhanced CT of the patient in our case confirmed an aortic aneurysm with an aortic thrombus (Fig. 1).

Prior to the cardiac arrest, the patient had never complained of abdominal symptoms and did not have abdominal angina. Furthermore, the patient's symptoms were sudden in onset and dramatic as seen in patients with embolism of the SMA^[5].

The only factor supporting the thrombosis theory is that the contrast-enhanced CT revealed several atherosclerotic plaques in the proximal part of the SMA and coeliac trunk. This, together with a history of chronic coronary artery disease suggests a tendency to generalized atherosclerosis. Plaque rupture in one of these plaques could have resulted in thrombosis of the SMA leading to ischaemia and necrosis of the intestines.

It can be difficult and almost impossible to differentiate between these two causes of occlusion of the SMA as reported by Acosta et al.^[4].

We agree with the authors in the above-mentioned cases^[5-7,9] that once intestinal ischaemia is suspected the patient should undergo immediate medical imaging in the form of contrast-

enhanced CT or mesenteric angiography to determine the cause of the ischaemia and initiate immediate surgical or medical intervention.

Conclusions

In the present case, a 68-year old man with known chronic coronary artery disease survived sudden cardiac arrest following successful CPR, but subsequently had intestinal ischaemia and necrosis requiring surgical intervention. The cause of the ischaemia is most likely an embolism to the SMA. If patients develop atrial fibrillation and abdominal symptoms following successful resuscitation from a cardiac arrest, then intestinal ischaemia should be ruled out. However, atrial fibrillation, whether related to cardiac arrest or other factors, may cause an embolus and possible intestinal ischaemia. These patients must undergo immediate medical imaging, and physicians must initiate immediate treatment in order to reduce the high mortality rates of this condition.

Teaching points

First, our case warns physicians to be aware of this life-threatening condition (intestinal ischaemia and necrosis) as most of these patients may have concomitant medical conditions or diseases that tend to shift the light away from this.

Second, patients with acute intestinal ischaemia are often not hospitalized in surgical wards but more likely in medical wards due to other concomitant internal medical diseases, therefore presenting a challenge to physicians in these wards, as its proven that the diagnosis of this condition in the early stages is difficult and often made too late for surgical intervention^[10].

Anyone who develops abdominal pain following cardiac resuscitation should be suspected of having acute intestinal ischaemia and be investigated as a matter of urgency.

Conflict of interest

The author declares that there is no conflict of interests regarding the publication of this article.

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