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Atheroembolization and potential air embolization during aortic declamping in open repair of a pararenal aortic aneurysm: A case report

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ABSTRACT

INTRODUCTION: When ischemic events ascribable to microembolization occur during open repair of proximal abdominal aortic aneurysms, a likely origin of atheroembolism is not always found.
PRESENTATION OF CASE: A 78-year old man with enlargement of the entire aorta underwent open repair for a pararenal abdominal aortic aneurysm using supraceliac aortic clamping for 20 min. Then the graft was clamped, the supraceliac clamp was removed, and the distal and right renal anastomoses were also completed. The patient was stable throughout the operation with only transient drop in blood pressure on reperfusion. Postoperatively the patient developed ischemia, attributable to microembolization, in legs, small intestine, gall bladder and kidneys. He underwent fasciotomy, small bowel and gall bladder resections. Intestinal absorptive function did not recover adequately and he died after 4 months. Microscopic examination of hundreds of intestinal, juxtaintestinal mesenteric, and gall bladder arteries showed a few ones containing cholesterol emboli.
DISCUSSION: It is unsure whether a few occluded small arteries out of several hundred could have caused the ischemic injury alone. There had been only moderate backbleeding from aortic branches above the proximal anastomosis while it was sutured. Inadvertently, remaining air in the graft, aorta, and aortic branches may have been whipped into the pulsating blood, resulting in air microbubbles, when the aortic clamp was removed.
CONCLUSION: Although both atheromatous particles and air microbubbles are well-known causes of iatrogenic microembolization, the importance of air microembolization in open repair of pararenal aortic aneurysms is not known and need to be studied.
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1. Introduction

The incidence of postoperative renal failure and other ischemic complications after paravisceral aortic clamping is significant [1–4]. Iatrogenic atheroembolization is a well-known cause of ischemic complications after aortic clamping [5–7]. Embolization may occur immediately, at an interval, or repeatedly after interaction between the applied surgical technique and an atheromatous lesion. But often the etiology of ischemic events cannot be ascertained [2,8,9]. A likely origin of atheroembolism is not always found in patients with events compatible with microembolization. During open aneurysm repair with the supraceliac aorta clamped, air gets access to the aorta and its branches below the clamp. Any remaining air may be whipped to microbubbles when the clamp is moved down to the vascular prosthesis. The clinical importance of air embolism during aortic declamping in aortic surgery is unknown.

We present a patient who suffered from atheroembolization, and possibly from air embolization, after an otherwise uneventful open repair of a pararenal aortic aneurysm.

2. Presentation of case

A 78-year old man who had been a smoker since his teenage was admitted in August 2007 with an asymptomatic but slightly tender abdominal aortic aneurysm. On the day of admittance he had an episode of dizziness, felt weak and had fallen down but had recovered completely after a few minutes. There was a history of transitory ischemic attack, he was being treated for hypertension and used aspirin, simvastatin, *N*-acetylcystein, and salbutamol

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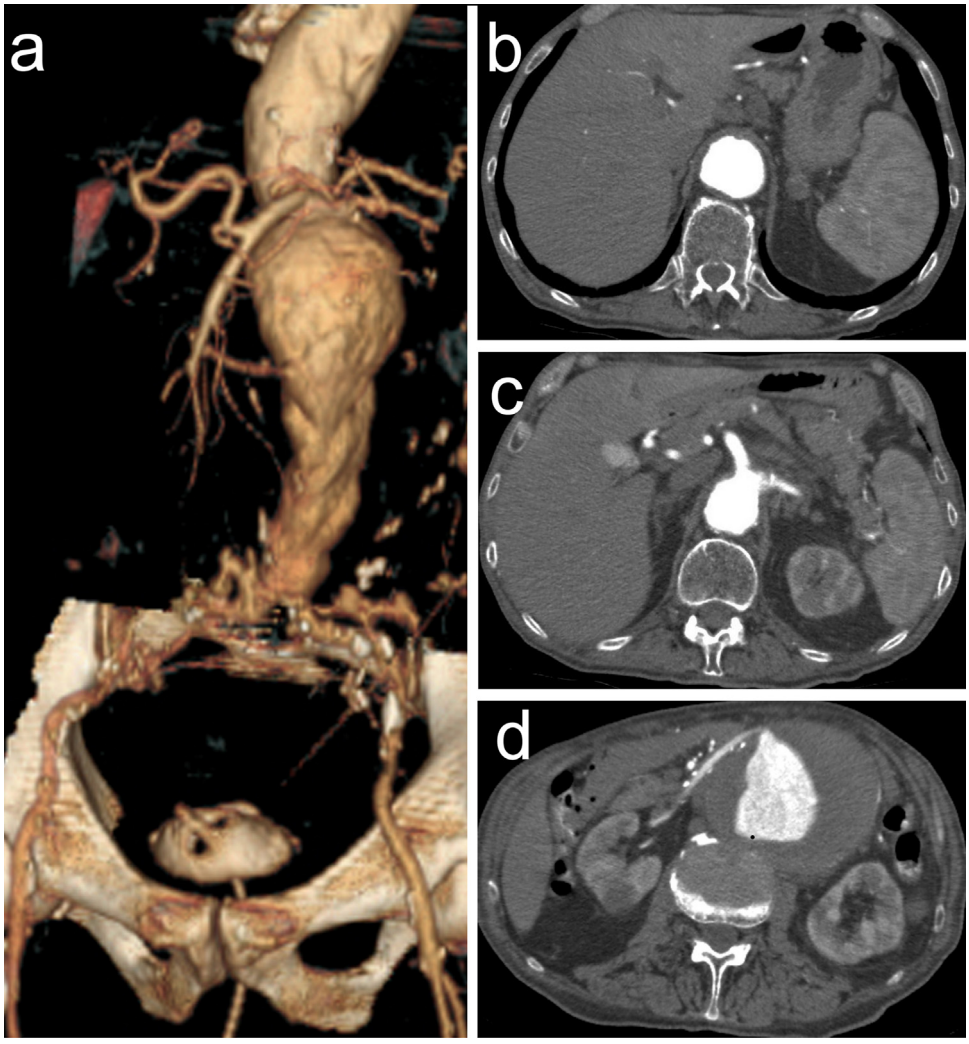

Fig. 1. Computed tomography angiography of the aorta and iliac arteries after admission with (a) outline of the blood-filled lumen, transverse sections through: (b) the descending aorta, (c) the superior mesenteric artery and left renal artery ostia, and (d) the right renal ostium.

inhalation therapy. He performed garden work regularly without feeling restricted in any way.

A computed tomography (CT) angiography showed that the entire aorta was enlarged, measuring 4.5 cm in the supraceliac segment. A pararenal abdominal aneurysm measured 8.4 cm in maximal diameter (Fig. 1). The origin of the left renal artery was from the proximal end of the aneurysm and the right renal artery originated from the aneurysm 5 cm distal to the lower margin of the aneurysm neck. He had normal sinus rhytm. Echocardiography showed slight left ventricular hypertrophy, normal left ventricular ejection fraction, and no signs of any previous myocardial infarction. Ankle pulses were present bilaterally. Blood pressure, at admittance 153/101 mmHg, was lowered to about 120 mmHg systolic pressure, using labetalol.

On day 0, three days after admittance, he underwent open aneurysm repair using a midline incision. After exposing the aneurysm he was given 5000 IU of heparin intravenously. The supraceliac aorta was clamped for 20 min while the proximal aneurysm neck, including the lower rim of the left renal artery, was anastomosed to a straight impregnated 20 mm polyester graft (Unigraft, B. Braun Melsungen AG, Melsungen, Germany). Then the graft was clamped and the supraceliac clamp was removed. After another 25 min the distal anastomosis was also completed. During most of this time 1000 ml of Ringer's acetate at 4 °C was infused into the right renal ostium. The left renal and visceral arteries were not touched. The ostium of the right renal artery was then anastomosed to an opening in the vascular prosthesis using a side-biting clamp. The patient was stable throughout the operation with only transient drop in blood pressure on reperfusion.

In the morning after the operation (day 1), his hands and feet were warm. He complained of pain in the anterolateral compartment of the right leg, and to a minor degree in his thighs. Motor and sensory functions were normal. Tension was increased in the anterolateral compartment of the right leg. A fasciotomy was therefore performed. Tension was normal in other leg compartments. He subsequently developed signs of hypovolemia and poor peripheral circulation. Serum myoglobin was 2829 μg/L. On day 2, he was tender in the abdomen where no peristalsis could be heard, and a re-laparotomy was performed. Despite normal arterial pulses in the small intestinal mesentery, the whole small intestine was dilated, showed no spontaneous peristalsis, and had a bluish colour signifying markedly reduced microcirculation. Along 90 cm of the distal ileum, except for the most distal 20 cm, several greenish-yellowish spots, signifying necrosis could be seen. This segment was resected. One additional spot more cephalad was removed using partial wall resection. The gall bladder, which had a yellowish-greenish appearance, and was tense and of questionable viability, was also removed, resulting in iatrogenic bleeding from the liver. The porta hepatis

Table 1
Tissues at risk for embolization, (Tissues at risk), approximate ischemia time in minutes (AIT), approximate time from aortic de-clamping to reperfusion in minutes (ADR), reversibility of ischemia (RI) and the possibility of air microembolization as etiology (AM).

Tissues at risk	AIT	ADR	RI	AM[a]
Intestines and gall bladder	20	0	Partial	Yes
Left kidney	20	0	Yes[b]	Yes
Spinal medulla	20	0	Yes[c]	Yes[c]
Lower extremities	45	25[d]	Yes	Yes[d]
Right kidney	70[e]	50[f]	Yes	Unlikely

[a] In addition to atheroembolization.
[b] It is not known whether a permanent minor loss of renal function was in either or both kidneys.
[c] Buoyant air bubbles may be less prone than atheromatous particles to enter the dorsal lumbar arterial ostia in a supine patient, but neither caused spinal injury.
[d] Air bubbles may have persisted in the aorta and graft cephalad to the clamp on the graft, see paragraph 2 in Section 3.
[e] Protected by infusion of cold Ringer's acetate.
[f] ca. 25 min after graft de-clamping.

was clamped for 15–20 min while haemostatic sutures were placed. The abdomen could not be closed without tension and was kept open until day 6. It was then closed with some tension. On day 2, there was a slightly increased tension in the anterolateral compartment of the left leg, and a fasciotomy was performed there as well.

The patient was intubated and had respiratory support while his abdomen was open and further until day 11. Despite normal diuresis he developed kidney failure, and needed haemodialysis for approximately 2 weeks, starting on day 12, whereafter renal function almost normalized. On day 15 he was transferred to the local hospital. He was weak, had bronchial secretions, febrile episodes ascribed to pneumonias, and a relapsing pleural effusion. A tracheostomy was performed on day 32. Resorptive intestinal function was poor and did not recover adequately. On day 124, he died from acute melena. Autopsy was not performed.

The distal mesentery that was attached to the resected small intestine was cut and examined for thrombi in the vascular lumina. Forty-seven microscopic sections were cut from the intestine and adjacent mesentery in those segments where the ischemic changes were most marked. The gall bladder was similarly examined. Sections were prepared routinely and stained with haematoxylin and eosin.

On cutting the distal mesentery that was attached to the resected small intestine, no thrombi could be observed in the vascular lumina at macroscopical examination. Among hundreds of intestinal and juxta-intestinal mesenteric vessels that were examined microscopically, three arteries with a luminal diameter of approximately 0.5 mm contained cholesterol emboli (Fig. 2). Cholesterol emboli were also found in the wall of the gall bladder.

This work has been reported in line with the CARE criteria [10].

3. Discussion

Our patient had severe ischemia attributable to microembolization although only a few arteries out of hundreds were blocked by atheroemboli. It is unsure whether a few small arteries, occluded by atheroemboli, among several hundred could have caused the ischemic injury alone. We have therefore considered whether air embolization could have had an additional etiological role (Table 1).

In our patient there had been only moderate backbleeding from aortic branches above the anastomosis while it was sutured. Inadvertently, not all air may have become displaced by blood prior to clamping the graft, and remaining air in the graft, aorta, and its branches proximally to the clamp may have been whipped into the pulsating blood, resulting in the formation of air microbubbles,

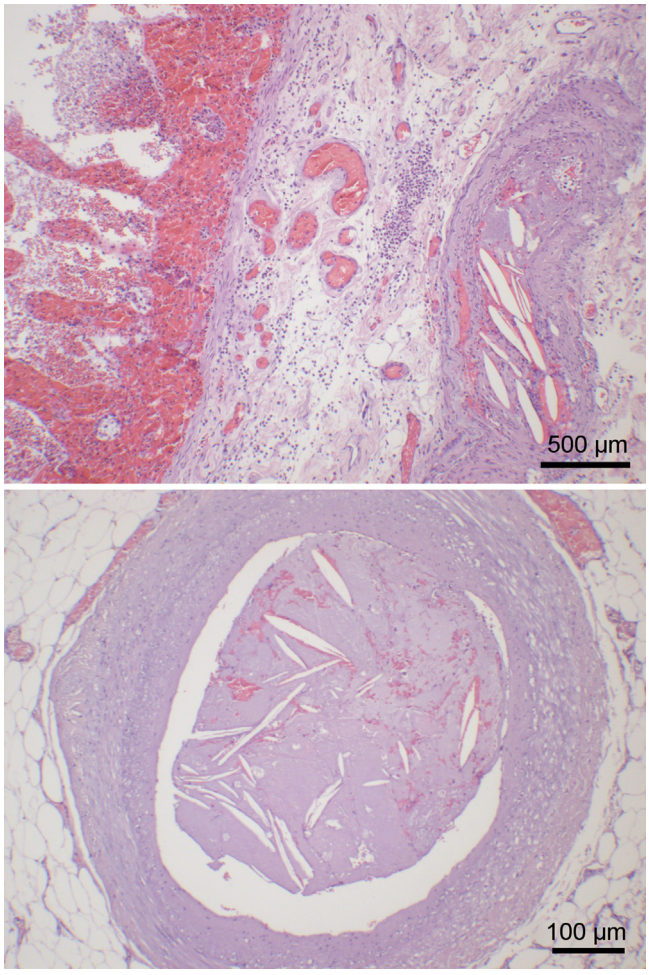

Fig. 2. Microscopic sections of resected small intestine (upper panel) and of juxta-intestinal tissue (lower panel) show atheroemboli with cholesterol crystals. Sections were stained with haematoxylin and eosin.

when the aortic clamp was removed. After heart surgery, when the left ventricle has been opened to air, microscopic air bubbles can be seen in, and exiting from, the left ventricle for nearly half an hour after the heart resumes beating, despite thorough removal of air [11]. Such air bubbles can cause ischemic injury in heart and brain. Analogously, microbubbles and atheromatous particles may have persisted in the distal aorta and graft cranially to the clamp on the graft until the lower body was reperfused.

Air emboli in microvessels damage endothelial cells and may interrupt blood flow long enough to allow thrombus formation [12,13], but leave no specific diagnostic signs.

After a period of aortic clamping that alone will not cause significant ischemic injury, if air embolism extends the ischemic insult, it may be the decisive cause of significant ischemic injury in tissues supplied by embolized vessels. The clinical importance of air emboli in open repair of pararenal aortic aneurysms has not been studied.

Atheroembolization may have occurred just after, during a time interval, or repeatedly after aortic declamping. Most likely, since the right kidney, which was reperfused only ca. 25 min after graft declamping, was also affected, there had been repetitive atheroembolization (Table 1). Alternatively, clamp ischemia and insufficient renal cooling, may have been responsible for the temporary function loss in the right kidney.

4. Conclusion

Both atheromatous particles and air microbubbles are well-known causes of iatrogenic microembolization, but there is a lack of knowledge and no literature on the importance of air embolism as a cause of ischemic complications in open repair of pararenal aortic aneurysms. Our patient had severe ischemic injury attributable to microembolization, and atheroembolization was demonstrated in the gall bladder and intestines. Because only a few small arteries out of several hundred were obstructed by atheroemboli and there may inadvertently have been incomplete de-airing, air microembolization may have had an additional etiological role. Air emboli in microvessels leave no specific diagnostic signs, but cause endothelial cell injury and may interrupt blood flow long enough to allow thrombus formation. Studies on the clinical importance of air microembolization in open repair of pararenal aortic aneurysms are needed.

Consent

Written informed consent was obtained from the patient's next of kin for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Conflict of interest

None.

Sources of funding

None.

Ethical approval

This is a case report on one patient. The patient did not undergo research and ethical approval therefore falls outside the mandate of the regional ethics committee, reference: REC west, no. 2015/2033-3.

Author contribution

EBD: (1) the conception and design of the study, and acquisition of data, and analysis and interpretation of data, (2) drafting the article, (3) final approval of the version to be submitted.

PKL: (1) acquisition of data, and analysis and interpretation of data, (2) revising the article critically for important intellectual content, (3) final approval of the version to be submitted.

Guarantor

Einar Børre Dregelid.

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