CASE PRESENTATION

A 51-year-old man presented with an altered mental state and intermittent fever. A known intravenous drug abuser, the patient had a previous hospital admission for left hand abscess and right calf necrotising fasciitis. Laboratory results at the present admission showed a haemoglobin level of 10.1 g/dL, haematocrit level at 30%, and a total white cell count of 24.6 x 10^9/L. Non-contrast computed tomography (CT) of the head was performed, which showed a large right frontal lobe haematoma with intraventricular extension and midline shift. The haematoma was surgically evacuated.

During post-surgical recovery, distension of the abdomen was noted, and the haemoglobin and haematocrit levels dropped to 4.9 g/dL and 14.6%, respectively. Contrast-enhanced CT imaging of the abdomen was performed in the arterial and portal venous phases (Fig. 1a-c). What do these images show?
IMAGE INTERPRETATION
These CT images of the abdomen at the level of the spleen in the arterial phase (Fig. 1a) and portal venous phase (Fig. 1b), and coronal reconstruction of the abdomen and pelvis in the arterial phase (Fig. 1c) show two aneurysms (white block arrows) arising from the branches of the splenic artery with a perisplenic haematoma (white open arrow) with active extravasation (curved arrow in Fig. 1b) and high density fluid in the abdomen and pelvis (white arrow), in keeping with haemoperitoneum. In addition, there was a subcapsular haematoma in the right kidney (black arrow in Fig. 1a).

DIAGNOSIS
Ruptured mycotic aneurysms of the spleen with haemoperitoneum; mycotic aneurysms of the kidney with perinephric haematoma.

Fig. 2 (a) Splenic angiogram shows two aneurysms (arrows). (b) Post-embolisation angiogram with coils (block arrow) shows no filling of the aneurysms.

Fig. 3 Right renal angiogram shows (a) multiple aneurysms (arrow heads) in the branches of the renal artery. (b) Note the small leak from a peripheral aneurysm in a delayed image (curved arrow).

Fig. 4 CT image of the head at presentation shows a large right frontal haematoma (white arrow) with intraventricular extension (black arrow).
CLINICAL COURSE

The patient underwent abdominal angiography. Selective splenic angiogram revealed two small aneurysms arising from the superior pole branch of the splenic artery with active contrast extravasation (Fig. 2a). The artery supplying the superior pole of the spleen was selectively catheterised and embolised with metallic coils. Post-embolisation check angiogram showed no active contrast extravasation (Fig. 2b). Right renal angiogram showed multiple small intra-parenchymal mycotic aneurysms (Fig. 3) with a small leak from one of the peripheral aneurysms. The aneurysms were deemed to be too small and too numerous for endovascular embolisation management. Hepatic, left renal and superior mesenteric angiograms did not reveal any aneurysms.

The patient tolerated the splenic embolisation procedure well without any immediate complication. Subsequent haematological tests showed a stabilisation of the haemoglobin level. Blood cultures were negative. The intracranial haematoma (Fig. 4) was thought to be secondary to a ruptured intracranial mycotic aneurysm. A final clinical diagnosis of multiple mycotic aneurysms from infective endocarditis secondary to intravenous drug abuse was made. The patient received intravenous gentamycin and ceftriazone. He recovered well with no further episodes of haemorrhage and was discharged to a community hospital for further rehabilitation care. There were no episodes of recurrence of intra-abdominal haemorrhage during the next two years of follow-up.

DISCUSSION

The term “mycotic aneurysm” was first used by Sir William Osler to denote aneurysms resulting from the lodgement of emboli which originate from infective endocarditis. Mycotic aneurysm (MA) can result from the following: (1) lodgement of septic emboli from infective endocarditis (Fig. 5), most commonly at points of branching, sites of tapering or sharp bends in a normal vascular tree; (2) lodgement of bacteria in the vasa vasorum or in the diseased intima such as atherosclerotic plaques (Fig. 6); (3) contiguous spread from a localised area of inflammation with destruction of the arterial wall and formation of a pseudoaneurysm (Fig. 7); and (4) injury to an artery with concomitant contamination and
subsequent formation of an infected pseudoaneurysm, which is commonly seen in drug abusers (Fig. 8), vascular surgery or gunshot wounds involving an artery. (2) MAs occur in 2%–10% of infective endocarditis cases, (2) and usually involve the aorta, as the primary branches of the aorta, the femoral, cerebral and visceral arteries are the next most common sites. (3) Most of the reported visceral MAs are in the form of a single or a few aneurysms involving one of the visceral territories. The appearance of multiple visceral MAs in the same patient is rare. (4-7)

MAs account for 10% of all splenic artery aneurysms, (8) and are associated with a 37% risk of rupture as opposed to a 2%–3% risk in a true splenic aneurysm. (9) Most true splenic aneurysms are asymptomatic and present as incidental findings on imaging (Fig. 9) carried out for unrelated indications. They are four times more common in women, and mostly involve the distal segment of the splenic artery (Fig. 10). (9) On the other hand, splenic MAs are frequently symptomatic, and mostly present with symptoms of abdominal pain and bleeding from the gastrointestinal tract. Other causes include splenic aneurysms secondary to increased flow from the splenomegaly (Fig. 11), erosion of the pseudocyst of the pancreas into a branch of the splenic artery (Fig. 12)
or into the main splenic artery, thus resulting in a pseudoaneurysm. Contrast blush seen in severe splenic injury may also mimic an aneurysm (Fig. 13), but this represents a contained leak in the injured spleen and is a strong indicator for immediate endovascular or surgical management.

Renal MAs are also uncommonly reported, and this is probably due to a lack of significant signs and symptoms directly referable to the aneurysm. Unilateral and bilateral renal artery involvements have been reported in the literature. A case of bilateral renal artery involvement, along with hepatic and mesenteric arteries MAs, has recently been reported. Most of the reported cases of renal MAs were diagnosed on angiography performed for haematuria, or nephrographic defects found on intravenous urography.

MAs are associated with life-threatening haemorrhages. The treatment options include antibiotic therapy, transcatheter embolisation and surgical resection with vascular reconstruction. Most of the visceral and intracranial MAs are initially treated with antibiotics, followed by either percutaneous embolisation or surgery. Antibiotic therapy may reduce haemorrhagic complications and may even resolve some MAs; however, the outcome is not predictable. Unfortunately, there are no certain predictive factors to evaluate the risk of a rupture. Life-threatening haemorrhages caused by ruptured MAs may require urgent embolisation or surgery without the use of antibiotic therapy, as illustrated in our case. Although transcatheter embolisation may be successful in preventing further haemorrhages, the risk of residual infection in the MA after embolisation is a known complication. Surgical resection of the aneurysm with vascular reconstruction is a suitable approach for large artery MAs and after failed embolisation. However, the management of multiple visceral MAs is still controversial. Tihansky et al reported successful embolisation of multiple splenic artery MAs. Kul et al managed multiple MAs of the hepatic renal and mesenteric arteries with only antibiotics, and the follow-up angiography showed a resolution of most of the aneurysms with some persistent MAs, which were clinically followed up. Mourad et al managed bilateral renal MAs with only antibiotic therapy and followed up with angiography, which showed a resolution of the MAs. In our case, the splenic artery MAs were managed with embolisation as they were causing life-threatening haemorrhage, and the renal MAs were managed with antibiotics and clinical follow-up. Splenic artery embolisation for MAs has a higher chance of preservation of the spleen and thus avoids the complications associated with splenectomy.

In summary, we have presented a rare case of multiple visceral MAs affecting the spleen and right kidney secondary to infective endocarditis in an intravenous drug abuser who presented with rupture and haemoperitoneum. The MAs were managed with a combination of transcatheter embolisation and antibiotic therapy. Although multiple visceral MAs are rare, clinicians and radiologists need to be aware of this entity and the various methods of treatment available.

ABSTRACT

Multiple visceral arterial mycotic aneurysms are very uncommon. We present a case of a 51-year-old male intravenous drug abuser who initially presented with intracranial haemorrhage secondary to a ruptured intracranial mycotic aneurysm. The haematoma was surgically evacuated. The postoperative recovery period was complicated by distension of the abdomen and a drop in the haematocrit and haemoglobin levels. Computed tomography of the abdomen demonstrated mycotic aneurysms in the spleen and right kidney. The splenic aneurysm had ruptured, resulting in haemoperitoneum. The aneurysm was successfully treated with embolisation, and the multiple renal artery aneurysms were managed with antibiotics and clinical follow-up. During the two-year follow-up period, no further episodes of bleeding were encountered. Intravenous drug abuse is associated with a high risk of infective endocarditis and the development of mycotic aneurysms, which can rupture and result in life-threatening bleeding. Multiple visceral mycotic aneurysms can be managed with a combination of endovascular treatment and antibiotic therapy.

Keywords: embolisation, intravenous drug abuse, mycotic aneurysm, renal, splenic

REFERENCES


True  False
☐  ☐
☐  ☐
☐  ☐
☐  ☐

Question 2. Regarding mycotic aneurysms:
(a) They occur in up to 10% of cases with infective endocarditis.
(b) The splenic artery is the most commonly affected artery in the body.
(c) They can involve the intracranial arteries.
(d) Rupture of the aneurysm can be life-threatening.

Question 3. Comparing mycotic aneurysms and true aneurysms of the splenic artery:
(a) Mycotic aneurysms are more common than true aneurysms.
(b) Mycotic aneurysms are less likely to rupture.
(c) True aneurysms are more common in women.
(d) Mycotic aneurysms are more likely to be symptomatic.

Question 4. Regarding renal mycotic aneurysms:
(a) They are uncommon.
(b) They involve only one kidney.
(c) They are frequently asymptomatic.
(d) They are more common than splenic mycotic aneurysms.

Question 5. Regarding the treatment of mycotic aneurysms:
(a) Transcatheter embolisation is an established method of treatment.
(b) The size of the mycotic aneurysm can predict rupture.
(c) Most of life-threatening ruptures of mycotic aneurysms can be treated by embolisation, and surgery if embolisation fails.
(d) Multiple small unruptured mycotic aneurysms may respond to antibiotic therapy alone.

Doctor’s particulars:
Name in full: __________________________________________________________________________________
MCR number: _____________________________________ Specialty: ___________________________________
Email address: _________________________________________________________________________________

SUBMISSION INSTRUCTIONS:
(1) Log on at the SMJ website: http://www.sma.org.sg/cme/smj and select the appropriate set of questions. (2) Select your answers and provide your name, email address and MCR number. Click on “Submit answers” to submit.

RESULTS:
(1) Answers will be published in the SMJ December 2010 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 15 December 2010. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (October 2010 SMJ 3B CME programme): 12 noon, 8 December 2010.