Ruptured abdominal aortic aneurysm and horseshoe kidney

A case report

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Summary

A patient with a ruptured abdominal aortic aneurysm associated with a horseshoe kidney is reported on. The treatment included aneurysmectomy and insertion of an aortic Dacron prosthesis without division of the isthmus of the kidney.

The postoperative course was complicated by a stroke and mild renal failure, but the patient made excellent progress and was discharged from hospital 1 month after admission.

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The anomaly of a horseshoe kidney is reported to occur in 1/500 - 1/1 000 autopsies.[1] These malformed kidneys usually function normally and are not more predisposed to renal disease than normal.[1]

The association of an abdominal aortic aneurysm (AAA) and a horseshoe kidney is uncommon.[2,3] This combination is extremely difficult to diagnose and is often unsuspected. The incidence of isolated ruptured abdominal aneurysms in association with a horseshoe kidney is very low.[4-6] The coexistence of a horseshoe kidney and an AAA presents multiple challenges to the surgeon which include correct pre-operative diagnosis, difficult dissection of the aneurysm, preservation of renal vasculature, inadequate exposure, and difficulty with insertion of the prosthesis.

We report the successful treatment of a patient who presented with a ruptured AAA and an unsuspected horseshoe kidney.

Case report

A 59-year-old White man was referred from a peripheral hospital to Tygerberg Hospital because of an acute abdomen. Apart from mild hypertension, for which he had been receiving treatment for 2 months, he had had no previous illnesses of note.

The present history included a sudden onset of excruciating abdominal pain localized to the left iliac fossa, pain in the lumbar region and perineum and syncope, which had been present for 12 hours before admission.

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On examination the blood pressure was 170/85 mmHg, the pulse rate 96/min and the haemoglobin value 8 g/dl. A palpable, pulsatile abdominal mass was noted to the left of the midline, associated with rebound tenderness. All peripheral pulses were palpable. Examination of the respiratory and cardiovascular systems and gastro-intestinal tract was negative.

A clinical diagnosis of ruptured abdominal aneurysm was made and an emergency laparotomy, without aortography or ultrasonography of the abdomen, was carried out.

At laparotomy a ruptured infrarenal AAA 8 cm in diameter was found; the aneurysm was associated with a horseshoe kidney and was situated anterior to the bifurcation of the aorta. Excessive haemorrhage together with the overlying kidney made dissection and control of bleeding from the aneurysm extremely difficult. The left renal artery was normal; the right renal artery originated from the right common iliac artery and entered the isthmus of the kidney. Both renal veins emerged from the upper poles of the kidney and joined the inferior vena cava below the diaphragm. Two ureters crossing the anterior surface of the kidney were identified. The isthmus of the kidney was 1,0 - 1,5 cm thick and consisted of functioning parenchymal tissue. After proximal control of the aneurysm had been achieved the aneurysm was resected and replaced with a 22 x 10 cm woven Dacron aortic bifurcation graft, without division of the isthmus of the kidney. The graft was placed behind the kidney. The aneurysm had eroded the lumbar vertebra and had ruptured on the left posterolateral aspect.

Intra-operative blood loss was in excess of 8 litres. The patient was anuric for 5 hours but started excreting after an infusion of 20% mannitol. He sustained a right-sided hemiparesis because of a cerebrovascular accident on the 5th postoperative day but recovered partially and was discharged 1 month after resection of the aneurysm with residual paresis of the right leg. Mild renal failure was managed without need for dialysis.

Discussion

The contemplated resection of an AAA in association with a horseshoe kidney poses special problems to the surgeon which include preservation of renal blood supply and renal function in addition to problems related to the fused kidney. In elective cases the resectability of the aneurysm is determined entirely by the blood supply of the kidney. Besides aortography, intravenous pyelography is of vital importance in planning the operative approach. The vessels supplying a horseshoe kidney may arise at any point along the aorta, occasionally from the iliac arteries, as in our case, or from the mesenteric vessels.[2] In many cases the aneurysm is irresectable if the major renal vessels arise from it. In addition, if multiple small vessels enter the aneurysm the latter is considered inoperable.

The management of the isthmus of the kidney is controversial. Some workers[2,3] have advocated simple division of the isthmus with oversewing of the cut surfaces, which facilitates resection of the aneurysm and placement of the prosthesis. Unfortunately, a complicated vascular pattern and a functioning isthmus are

likely to coexist. The outcome of division of the isthmus in these cases is unpredictable. Under these circumstances resection of the aneurysm may be possible if the graft is placed posteriorly, as in our patient. A second alternative is autotransplantation of the kidney, but there is no recorded instance in the literature of successful autotransplantation of a horseshoe kidney following resection of a ruptured AAA.

Division of the isthmus of the kidney is not without danger. Firstly, damage to the renal pelvis as a result of surgical manipulation may produce spillage of infected urine which exposes the patient to the serious sequelae of an infected vascular prosthesis. Secondly, division of the isthmus or an associated polar artery may result in infarction of renal tissue.[5] Cayten *et al.*[4] have suggested that the isthmus should only be divided when extended exposure is mandatory. Perhaps division of the isthmus should be strongly considered in ruptured aneurysms so as to facilitate control of bleeding from the aneurysm. They indicate that in elective cases it is frequently possible to mobilize the kidney and place the graft on the posterior aspect of the isthmus. They also point out that anomalous venous drainage is common in the presence of a horseshoe kidney, necessitating great care before unidentified vessels are ligated.

Rupture or threatened rupture of an AAA in the presence of a horseshoe kidney is a life-threatening situation and the survival of the patient is at stake. Fortunately the entity is rare, but heroic surgical manoeuvres may be required to save the patient.

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REFERENCES

1. Robbins SL, Cotran RS. *Pathologic Basis of Disease*. 2nd ed. Philadelphia: WB Saunders, 1979: 1123.
2. Bietz DS, Merendino KA. Abdominal aneurysm and horseshoe kidney: a review. *Ann Surg* 1975; **181:** 333-341.
3. Brown OW, Dosick SM, Blakemore WS. Abdominal aortic aneurysm and horseshoe kidney: a different perspective. *Arch Surg* 1979; **114:** 860-861.
4. Cayten CG, Davis AV, Berkowitz HD, Roberts B. Ruptured abdominal aortic aneurysms in the presence of horseshoe kidneys. *Surg Gynecol Obstet* 1972; **135:** 945-949.
5. Landes RG, Trumbell HR, Nicoleff DM. Abdominal aortic aneurysm with rupture into the inferior vena cava associated with horseshoe kidney. *Ann Surg* 1978; **187:** 329-331.
6. Mannick JA, Brooks JW, Bosher LH, Hume DM. Ruptured aneurysm of the abdominal aorta. *N Engl J Med* 1964; **271:** 915-920.

Carbon monoxide poisoning

Report of a case with 1-year computed tomographic follow-up

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Summary

A case of acute carbon monoxide poisoning with 1-year computed tomographic follow-up is presented. The typical initial bilateral symmetrical low-density areas in the basal ganglia were found to have decreased markedly in size in the latter scan. These appearances coincided with the initial early oedematous phase of infarction ending in the late permanent necrotic stage.

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Hypoxia resulting from carbon monoxide (CO) poisoning is due to the displacement of oxygen (O_2) from haemoglobin. CO competes with O_2 by diffusing across alveolar membranes and binding to haemoglobin. Haemoglobin has approximately 250 times the affinity for CO that O_2 has, and exposure to small concentrations of CO may therefore be clinically significant. Although all organs are affected by CO poisoning, the brain is the most important of these. Pathologically there is marked venous and capillary dilation. Petechial haemorrhages and frequently early necrosis of the basal ganglia, most often the globus pallidus as well as the reticular zone of the substantia nigra, have been observed. The Purkinje cells of the cerebellar cortex and dentate nucleus are commonly involved, as are cortical cells.[1]

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Case report

A 26-year-old man who attempted suicide by gassing himself in a motor vehicle was admitted to an outlying hospital in a confused stuporous state. He regained consciousness a day later and was transferred to our hospital. On examination he was disorientated for time and place with marked memory impairment and a mask-like facies. There were no extrapyramidal signs. Generalized brisk reflexes were present. The neurological examination was otherwise negative. Computed tomography (CT) showed bilateral symmetrical low-density areas in the globus pallidus. There was no change following intravenous contrast administration (Fig. 1). The patient was discharged shortly thereafter and followed up as an outpatient. One year later he again underwent scanning owing to persistent poor memory and inability to concentrate. CT showed a marked diminution in the