Difficult Diagnosis

Giant hydronephrosis masquerading as massive ascites

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Summary: A case of unilateral giant hydronephrosis containing about 20 litres of old haemorrhagic fluid, clinically simulating massive ascites, is reported. The role of preceding abdominal trauma in the pathogenesis and the rapidity of the disease process is discussed.

Introduction

Hydronephrosis sometimes presents as an intra-abdominal mass with the features of a renal swelling. However, as a result of widespread use of ultrasonography, most cases of hydronephrosis are now diagnosed before the kidney is large enough to produce a visible swelling. Rarely, massive distension of the kidney may occur and create diagnostic confusion.¹ The present report relates to such a case, in an adult male, who had a rapidly progressive 'giant hydronephrosis' clinically simulating ascites.

Case report

A 30-year-old male, was admitted with rapidly progressive abdominal distension and mild dull aching diffuse abdominal pain of 2 months duration. A few days prior to this, he had fallen from his bicycle in a road accident and had sustained abrasions and bruises over his chin, knees and abdomen. He denied history of fever, weight loss, urinary trouble, or any abdominal swelling in the past.

Clinical examination revealed mild pallor, mild pitting oedema over both legs and normal blood pressure. The abdomen was huge and symmetrically distended (Figure 1). No abdominal mass was palpable. A fluid thrill could be easily elicited. The swelling was dull on percussion but there was no shifting dullness. Examination of other systems was unremarkable.

Laboratory investigations showed haemoglobin 11 g/dl, normal total and differential leukocyte count, ESR 65 mm in 1 hour. Blood sugar, blood urea, serum creatinine, serum amylase, liver

function tests, serum calcium, phosphate and electrolytes were within normal limits. Urine examination did not reveal any abnormality and urine culture was sterile. Plain X-ray of the abdomen revealed elevated right dome of diaphragm up to the third intercostal space anteriorly. Diagnostic aspiration from abdominal swelling revealed coffee-ground fluid with 107 cells/mm³, the majority being polymorphs, many red blood cells, proteins 4.7 g/dl, but no malignant cells on Papanicolaou examination. The fluid culture was sterile. Ultrasonography revealed a large hypoechoic mass lesion occupying nearly the whole of the abdomen with multiple septate internal echoes (Figure 2). The right kidney was not visualized separately but the left kidney appeared normal. The liver was displaced upwards. Intravenous pyelography revealed normal appearance and function of the left kidney but a non-functioning right kidney. Antegrade pyelography revealed diffuse opacification of the abdominal cavity.

The abdomen was explored via a generous midline incision from xiphoid to pubic symphysis. Ascending colon was reflected medially after dividing parietal peritoneum in right paracolic gutter. A hugely enlarged right kidney was seen with predominantly extrarenal pelvis encroaching across the midline to the left lumbar region. The renal parenchyma was thinned out and pushed superiorly by the renal pelvis. The diameter of the ureter was normal with narrowing at the ureteropelvic junction. Right nephrectomy was performed after dividing the renal artery, vein and ureter. Approximately 20 litres of coffee-ground fluid came out. The cut open section of the specimen revealed a huge sac-like mass with a thickened and fibrosed wall and areas of haemorrhage. The postoperative period was uneventful. Histology revealed hydronephrotic sac lined by organizing fibrocollagenous tissue with haemorrhage and congestion with a few tubular cells seen peripherally.

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Figure 1 Photograph showing huge symmetrical distension of the abdomen.

Figure 2 Ultrasonogram showing a large hypoechoic lesion with multiple septate internal echoes.

Discussion

The present case has several unusual features. A kidney containing more than 1 litre of fluid in its collecting system is generally defined as ‘giant hydronephrosis’.¹ A giant hydronephrosis may

rarely fill the entire abdomen, as in our patient, and differentiation of the condition from ascites may then be difficult on clinical examination alone. Occasionally, a band of resonance may be present in the opposite flank but it was not demonstrable in the present case. In a review¹ it was noted that a

correct preoperative diagnosis of giant hydronephrosis has been made in only 46% of cases. The initial clinical diagnosis of the present case was massive ascites, probably due to malignancy. However, his ultrasonographic appearance raised the possibility of right-sided hydronephrosis. Similar ultrasonographic findings have been described by Hricak *et al.*² in giant hydronephrosis. However, ultrasonographic findings are not as characteristic of the disease in giant hydronephrosis as in the diagnosis of hydronephrosis at an earlier stage.² Intravenous pyelography revealed a non-functioning right kidney which supported the possibility of hydronephrosis. Antegrade pyelography was, however, not successful in visualizing the pelvicalyceal system. This was possibly due to the massive size and insufficient contrast. We feel that ultrasonographically guided injection of a large amount of contrast could help to visualize the pelvicalyceal system and the level of obstruction in giant hydronephrosis.

Another interesting feature of the disease in the present case was the rapidity of the disease process. Within a period of 2 months, the patient developed massive hydronephrosis of this degree. Such rapid progression of the disease could not be traced in the available literature. Most of the patients with giant hydronephrosis reported earlier had a slowly progressive long-standing disease.^{1,3-5} We assume that the preceding abdominal trauma in the present case resulted in internal haemorrhage in the previously undetected hydronephrotic kidney with ischaemic changes due to pressure effect on the renal parenchyma leading to rapid aggravation of the hydronephrotic changes in the affected kidney. This was supported by the old haemorrhagic appearance of the aspirated fluid and areas of haemorrhage in the wall of kidney as observed postoperatively and the histopathological findings. Moreover, in experimental animals, ureteric obstruction in ischaemic kidney has been reported to produce rapidly progressive hydronephrotic change.⁶

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