Acute focal bacterial nephritis

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SUMMARY In the kidney, acute focal parenchymal infection without liquefaction can produce a 'mass lesion'¹ that may mimic an abscess or tumour, both clinically and radiologically. Ultrasound and computed tomography can differentiate between these lesions and allow appropriate antibiotic treatment to be used safely, avoiding unnecessary surgical intervention.

Case report

A previously well 19 month old boy presented with four days of irritability and fever and two days of vomiting. On hospital admission he was still feverish, but no focus of infection could be found; in particular he had no abdominal or loin tenderness. Mild hypospadias was also noted. A white cell count was grossly raised at $41.7 \times 10^9/l$ (86% neutrophils). Initially his urine was sterile and free from pus cells, but pyuria was evident in a subsequent specimen which grew klebsiella. Several blood cultures were sterile.

A diagnosis of urinary tract infection was made and co-trimoxazole prescribed, but after two doses had been vomited, amoxycillin was substituted. Fever continued, however, and the child remained unwell. On the third day the antibiotic sensitivities of the klebsiella showed resistance to amoxycillin but sensitivity to co-trimoxazole and cefuroxime. After 48 hours, intravenous cefuroxime produced a sustained clinical improvement and was continued for a total of five days. Oral co-trimoxazole was then tolerated for a further three weeks, with complete recovery.

Because of the slow clinical resolution during the initial stages of this illness, intravenous urography was performed on the fourth day. This showed expansion and a poor nephrogram at the upper pole of the right kidney with calyceal compression suggesting a space occupying lesion. An ultrasound scan confirmed a mass lesion which, although relatively anechoic, was not of fluid consistency and did not have the features of an abscess (Fig. 1). The possibility of a tumour was therefore raised and computed tomography was done on the fifth day. This showed an expanded, wedge shaped area at the

Fig. 1 Longitudinal ultrasound scan of right kidney (RK) showing a 3 cm solid mass (M) lesion at the upper pole.

right upper pole posteriorly, affecting both cortex and medulla, with poor uptake of intravenous contrast (Fig. 2) but without the distortion of the normal renal parenchyma or the pathological circulation associated with a neoplasm. The computed tomogram, however, was consistent with a diagnosis of acute focal bacterial nephritis as were the ultrasound findings when viewed in retrospect.

Follow up ultrasound scan at 14 days showed complete resolution of the upper pole mass while a computed tomogram after 31 days showed a cortical scar at the site of the previous abnormality. Measurements of renal function and blood pressure were normal throughout the illness and repeated urine specimens were sterile. A micturating cystogram is planned for the near future.

Fig. 2 Computed tomogram with intravenous contrast showing an expanded, wedge shaped area at the right upper pole, affecting both cortex and medulla, with poor excretion of contrast.

M=mass; RK=right kidney; LK=left kidney; L=liver; S=spleen; P=pancreas.

Discussion

Renal parenchymal infection is a spectrum of disease that begins with acute focal bacterial nephritis in which no liquefaction occurs.² Later micro-abscesses form which coalesce to produce a single abscess. Renal infection usually presents with fever, loin pain, and tenderness but symptoms and signs may be non-specific, particularly in children under 5 years of age. Infection is usually with Gram negative organisms introduced along a presumed ascending route,¹⁻³ with vesicoureteric reflux being found,⁴ or at least implied, in the pathogenesis. Predisposing factors²⁻³ include urinary stasis caused by an anatomical anomaly or obstruction; an abnormal host response to infection such as in states of immunosuppression or diabetes; or, rarely, a disease in another system, for example congenital heart disease with subacute bacterial endocarditis.

Urgent investigation of renal tract infection becomes necessary when signs of focal sepsis occur or if treatment does not produce a clinical improvement, as drainage or surgery may be required.⁵ Acute focal bacterial nephritis, however, is best managed conservatively with parenteral antibiotics.^{2 4 6} If a mass lesion is found in the kidney the differential diagnosis principally lies between focal bacterial nephritis, renal abscess, and renal tumour, the treatment of each being quite different. The choice of appropriate investigations is therefore important.

Excretory urography shows focal renal enlargement (presumably due to local oedema), a faint nephrogram, and poor opacification of the collect-

ing system.^{4 6} These are relatively non-specific changes which may be found in any of the three disease states. Arteriography or venography in acute focal bacterial nephritis show more specific signs of stretching of the vessels with occlusion of some small venules,² but both procedures are extremely invasive. Radioisotope scanning will show the focal nature of the lesion but is not specific and often gives a false negative result.³ The results are available only 24 hours after injection, although ^{99m}Tc DMSA scanning is completed within three to four hours and can be more sensitive than conventional intravenous urography.

Ultrasound can detect focal abnormalities of 2 cm or more in diameter⁵ and is highly selective in differentiating acute focal bacterial nephritis from an abscess.³ The former is seen as a poorly defined, relatively anechoic solid mass with disrupted corticomedullary differentiation and an ill defined distal wall.^{1 4 6} In contrast an abscess has well defined borders, including its distal wall, and may be seen to contain debris or even fluid levels in an area of central liquefaction.⁴ A tumour produces distortion of the renal anatomy and is usually more echogenic than acute focal bacterial nephritis.⁴

Acute focal bacterial nephritis is similarly shown on computed tomography to be a solid mass, with poor contrast enhancement in single or multiple wedge shaped defects extending from the medulla to the renal cortex in lobar distribution.⁵ In contrast, an abscess contains central fluid and is sharply demarcated from the normal adjacent enhancing parenchyma, while a tumour distorts renal anatomy and will usually show abnormal heterogeneous enhancement with contrast due to the pathological circulation.

Ultrasonography is comfortable, safe, non-invasive, and particularly suited for use with children. The findings of a relatively anechoic solid renal mass in the clinical setting of urinary tract infection should enable the diagnosis of acute focal bacterial nephritis to be made. After appropriate antibiotic treatment, clinical improvement and resolution of the mass on ultrasound scan will confirm the correct diagnosis.⁶ Computed tomography should be necessary only when doubt exists after ultrasound, or in the evaluation of the extent of severe renal or perirenal infection, although some find it more reliable than ultrasound.⁵ In retrospect, ultrasound should have been our initial investigation as its correct interpretation would have obviated the necessity for excretory urography or computed tomography. Micturating cystography is irrelevant in immediate management, but should be performed later to identify those patients predisposed to future renal infections.⁴

All previous reports on acute focal bacterial nephritis have been published in urological or radiological journals¹⁻⁶ and it has yet to receive individual coding in the international classification of diseases. It is probable, therefore, that this disorder is often not recognised due to lack of awareness of the diagnosis. In addition, early treatment with antibiotics can no doubt lead to resolution before progression to abscess formation, and only the use of ultrasound or computed tomography in the acute stage can accurately identify this disease entity. Nevertheless, when encountered, precise diagnosis is important so that acute focal bacterial nephritis can be appropriately and conservatively managed with parenteral antibiotics, avoiding unnecessary laparotomy or attempted percutaneous drainage.

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Remission of progressive renal failure in familial Mediterranean fever during colchicine treatment

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SUMMARY Colchicine was administered to a 12 year old girl with familial Mediterranean fever and progressive renal insufficiency. There was immediate resolution of abdominal attacks together with a dramatic fall in the serum creatinine concentration and the degree of proteinuria. At the same time her severely impaired growth was stimulated.

Familial Mediterranean fever is an inherited disease with recurrent inflammation of the joints and the pleural and peritoneal cavities. Development of amyloidosis in this disease is fairly common and is seen in one third of patients during the first two decades of life.¹ Onset of familial Mediterranean fever amyloidosis in childhood indicates a poor prognosis, with only 20% survival 5 years after proteinuria has developed,² and no spontaneous regression of renal failure has been reported.¹ Colchicine has been shown to reduce the severity and frequency of attacks.³ We describe the course of progressive renal failure in a 12 year old girl with familial Mediterranean fever which remitted during colchicine treatment.

Case report

This girl was the first of three children of Turkish parents. From 5 years of age she had suffered recurrent attacks of abdominal pain. She was admitted to our department when 11 years old, shortly after arriving in Denmark as an immigrant. She was chronically disabled with oedema of hands and feet, anaemia (haemoglobin 9.6 g/dl), hepatomegaly, height 107 cm (less than -3 SD), weight 18 kg (less than -3 SD), and erythrocyte sedimentation rate 97 mm in the first hour. Proteinuria was present (10 g/day), but her renal function was normal (creatinine clearance 94 ml/minute, serum creatinine value 33 µmol/l). The fibrinogen value was considerably raised at 10.3 g/l. Blood pressure and liver function were normal. At first, nephrotic syndrome was suspected but a trial with prednisone produced no effect. Rectal biopsy showed amyloid infiltration and the clinical picture was consistent with familial Mediterranean fever (recurrent polyserositis). This disorder has subsequently been diagnosed in a cousin. Her abdominal symptoms partly subsided and she was treated with diuretics only. During autumn 1983 the abdominal attacks