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BRIEF REPORT

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Transient post-renal obstruction may protect the kidney against nephrotoxic damage – a case report

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Abstract In critically ill patients, acute renal failure is mostly multifactorial in origin. In general, the simultaneous presence of several deleterious factors tends to aggravate the renal damage. The present case report describes a patient with multifactorial acute renal failure, in whom one of the factors contributing to the renal failure, i.e. transient unilateral post-renal obstruction, apparently protected the obstructed kidney against damage from other causes.

Key words Kidney failure · Acute Ureteral obstruction · Hydronephrosis

Introduction

In critically ill patients acute renal failure is usually due to acute tubular necrosis, which is often multifactorial in origin. The simultaneous presence of several deleterious factors potentiates the renal damage [1, 2]. We report on a patient in whom acute unilateral ureteral obstruction appeared to protect the obstructed kidney from nephrotoxic damage.

Case report

A 50-year old woman was admitted to another hospital. She had been well until two days earlier, when her general practitioner prescribed amoxycillin, and diclofenac suppositories for right flank pain and fever. On admission she was very ill and oliguric. Blood pressure was 130/100 mmHg; it decreased to 55/30 mmHg over the next hours. Serum creatinine, previously normal, was 201 umol/l, urea 9.9 mmol/l. Complete blood count showed 24.10^9/l leukocytes and 40.10^9/l thrombocytes. Fragmented red blood cells were present. Coagulation studies revealed prolonged prothrombin time (29 s, normal 11–16 s), prolonged activated partial thromboplastin time (60 s, normal 30–39 s) elevated fibrin split products (>320 µg/ml; normal <8 µg/ml), and decreased antithrombin III (45%, normal 80–120%) and fibrinogen (0.5 g/l, normal 1.7–3.5 g/l), all consistent with disseminated intravascular coagulation. Gentamicin (100 mg once daily) and piperacillin (2 mg four times daily) were administered. A plain abdominal X-ray displayed two normal-sized (12 cm) kidneys and a radio-opaque calculus in the right pelvic region. As ultrasonography was inconclusive, intravenous urography (with Conray 60, containing 600 mg meglumine iothalamate) was performed. It showed delayed and persistent nephrography of the left kidney without excretion of contrast into the pyelum; size and contour of the kidney were normal: no nephrography occurred at the right side. Repeated ultrasonography disclosed a right kidney with a dilated pyelum and ureter and a stone obstructing the distal ureter. A nephrostomy catheter was inserted into the right pyelum. The patient was then referred to our hospital. She had a full-blown septic shock and developed multi-organ failure. She was treated by hemodynamic support with volume substitution and inotrope, positive pressure ventilation, continuous arterio-venous hemofiltration and imipenem/cilastatin. The nephrostomy catheter allowed assessment of separate kidney function; bilateral anuria was present for eight days. After improvement of hemodynamic stability the right kidney became transiently polyuric, with a diuresis of 4 l daily. Thereafter, creatinine clearance gradually increased and stabilized at 25 ml/min. The left kidney remained anuric throughout the period the nephrostomy catheter was in situ, i.e. 15 weeks. Then a uretero-lithotomy was performed and the nephrostomy catheter was removed. At discharge the patient was well, with a creatinine clearance of 25 ml/min.

Comment

Multiple causes contributed to the renal failure in our patient. Pre-renal factors were the hypotension and the use of the non-steroid antiphlogistic diclofenac; renal factors were the use of aminoglycoside and the intravenous

radiocontrast agent, and the presence of disseminated intravascular coagulation; finally post-renal obstruction of the right kidney was present. Remarkably, recovery of renal function was confined to the temporarily obstructed right kidney. This suggests that obstruction provided protection against the combined nephrotoxicity of hypoperfusion and tubulotoxic substances. This is surprising, as obstruction in itself can induce renal damage. It has been observed however, that obstruction can affect the distribution of diffuse renal disease, as suggested by the association of unilateral obstruction with the rare condition of unilateral glomerulonephritis [3], where glomerulonephritis is only present in the non-obstructed kidney.

It can be argued that we do not have conclusive evidence that the left kidney was functionally normal prior to the acute events. The normal size and countour of this kidney on urography, however, argue against chronic unilateral left kidney disease; the delayed and persistent nephrography of that kidney is fully compatible with the state of pre-renal oliguria present at that time. Moreover, the nephrography of the left kidney with an absent nephrography of the obstructed right kidney implies that, at that time, the function of the left kidney was less severely impaired, as it was still able to filtrate the radiocontrast agent.

By what mechanism could obstruction have provided renal protection? First, both aminoglycosides and radiocontrast agents exert their nephrotoxic effect after having entered the tubules by glomerular filtration [4, 5]. The complete cessation of filtration in the obstructed kidney at the time of administration of these agents (as evident from the urography), therefore, may have provided protection against their toxic effects. Second, as renal function in the non-obstructed kidney did not recover, intravascular coagulation may have been a major factor in the pathogenesis of renal failure in this patient, as this can induce permanent, ischemic, damage. Unilateral ureteral obstuction reduces renal perfusion due to predominantly preglomerular vasoconstriction [6]. Reduced renal perfusion, beyond the degreee already present by the prerenal factors, may therefore have limited glomerular endothelial injury in the obstructed kidney.

In the diagnostic work-up of acute renal failure exclusion of post-renal obstruction is mandatory. Our observation underlines the importance of cautiously ruling out obstruction, even when other factors affecting renal function are obviously present, as obstruction not only represents a reversible cause in itself, but may also increase the reversibility of the renal functional impairment by nephrotoxic substances.

References

1. Rasmussen HH, Ibels LS (1982) Acute renal failure. Multivariate analysis of causes and risk factors. Am J Med 73: 211–218
2. Zager RA, Sharma HM, Johannes GA (1983) Gentamycin increases renal susceptibility to an acute ischemic insult. J Lab Clin Med 101:670–678
3. Editorial (1975) Unilateral glomerulonephritis. Lancet I:206–207
4. Mudge GM (1980) Nephrotoxicity of urographic radiocontrast drugs. Kidney Int 18:540–552
5. Humes HD (1988) Aminoglycoside nephrotoxicity. Kidney Int 33:900–911
6. Dal Canton A, Corradi A, Stanziale R, Maruccio G, Migone L (1979) Effects of 24-hour unilateral ureteral obstruction on glomerular hemodynamics in rat kidney. Kidney Int 15:457–462