Case Report

Xanthogranulomatous pyelonephritis in the native kidney of a renal transplant recipient

V. Boulanger¹, P. Merville¹, D. Morel¹, J. L. Pariente², C. Deminière³ and L. Potaux¹

¹Department of Nephrology and Renal Transplantation, ²Department of Urology, ³Department of Pathology, Hôpital Pellegrin-Tripode, Bordeaux, France

Key words: xanthogranulomatous pyelonephritis, renal transplantation, xanthoma cells

Introduction

Xanthogranulomatous pyelonephritis [1] is a rare condition, which is rarer still in an allograft recipient. We had recently the opportunity to observe this pathology in such a recipient.

Case Report

A 55-year-old renal allograft recipient was admitted with a 3-month history of chronic abdominal pain, malaise, and weight loss (2 kg) without fever. He had no record of urinary tract obstruction or calculi. In 1987 he had received a cadaveric renal allograft for renal failure which had appeared in 1979. The primary nephropathy was unknown, and a renal biopsy was not performed at that time because of the atrophic nature of his kidneys. Renal function recovered promptly after transplantation with an immunosuppressive regimen comprising cyclosporin A, azathioprine, and steroids. During the following years, he presented numerous urinary tract infections, with isolation of various organisms (mainly Escherichia coli and Staphylococcus aureus).

Physical examination was normal. Laboratory results showed an important inflammatory syndrome with elevated levels of CRP (320 mg/l), fibrinogen (7.8 g/l), and accelerated erythrocyte sedimentation rate at 110 mm. White blood cell count was 10 000/mm³. Values of haemoglobin, liver enzymes, serum creatinine, and blood urea nitrogen were normal. Urine examination revealed leukocyturia without bacteriuria, and a protein level at 1.0 g per day. Haematuria was also present and morphological study showed that 90% of the erythrocytes were distorted, without any additional abnormality.

Abdominal radiography did not show any calcifications. Ultrasound examination discovered an enlarged left native kidney with a dilated collecting system, whereas vascularization and echogenicity of the allograft were unremarkable. As indicated in Figure 1, an abdominal CT scan confirmed mild hydronephrosis of the left native kidney, which appeared enlarged and hyperdense with regular limits and a substantial perirenal hypodense infiltration. These findings suggested lymphoma or urothelial tumour.

A left-sided nephroureterectomy was performed. The kidney was macroscopically pale and surrounded with indurated fat tissue. Cross-sections of the organ displayed multiple yellowish foci and a markedly dilated pyelum, filled with a purulent material. Histological examination diagnosed xanthogranulomatous pyelonephritis with a typical pattern of histiocyte infiltration. The histiocytic nature of the infiltrative cells was confirmed by immunostaining using specific monoclonal antibody against CD68. The patient had an uncomplicated recovery after surgery and left the hospital with long-term antimicrobial therapy. Kidney

Correspondence and offprint requests to: Dr Pierre Merville, Service de Néphrologie et Transplantation Rénale, Hôpital Pellegrin-Tripode, Place Aimée Raba-Léon, 33076, Bordeaux Cedex, France.

© 1997 European Renal Association–European Dialysis and Transplant Association
graft function was unaffected by this episode and we did not modify the immunosuppressive regimen.

**Comment**

The incidence of xanthogranulomatous pyelonephritis is not known, a fact that probably reflects its rarity. In one series it was found in six of 1000 surgically proven cases of chronic pyelonephritis [2], and a review of the Mayo Clinic’s experience from 1918 to 1975 found only 26 cases of this condition [3]. The depicted case represents the unique patient we have observed out of more than 1400 renal recipients over the last 20 years in our own centre.

The originality of this observation lies in the onset of xanthogranulomatous pyelonephritis in a renal transplant recipient. In this context, two situations can be clearly identified.

First, xanthogranulomatous pyelonephritis may affect the native kidney, as in our case. The disease was first described in 1984 in a single native kidney following a living-donor kidney transplantation, and was surgically treated [4]. Bilateral native localization has been described and associated lesions, particularly renal carcinoma, are not infrequent [5]. Consequently we recommend surgical treatment as the optimal management of any solid mass of a native end-stage kidney in a transplanted patient.

Second, xanthogranulomatous pyelonephritis may affect the kidney allograft itself. In this particular localization, differential diagnosis with acute rejection can be problematic [6]. Non-contributive biopsy must be repeated, since the lesions can be focal. Massive unusual infiltration of CD68+ histiocytes, such as that demonstrated by the immunohistological staining we performed, may be considered as a reliable indication of the diagnosis. Surgical treatment is not mandatory and long-term antibiotic therapy has been successful in preserving graft function [7].

In conclusion, xanthogranulomatous pyelonephritis is a rare, but perhaps underestimated renal pathology which can affect kidney allograft recipients. Curative treatment consists in surgery, when the native kidney is affected, or antibiotic therapy, when the localization is in the allograft.

**References**


Received for publication: 4.11.96
Accepted: 14.11.96