

DIAGNOSIS

First, the possibility of renal tuberculosis was considered. However, the patient had been treated adequately before transplantation and both the Ziehl-Neelsen and auramine staining for acid-fast bacteria were negative. Other causes of granulomatous tubulo-interstitial nephritis (GTIN) that were considered included acute T-cell mediated rejection, drug-related toxicity, infections, sarcoidosis, tubulointerstitial nephritis and uveitis syndrome, paraproteinaemia and Wegener's granulomatosis. Finally, GTIN can be idiopathic.¹

To find out if the histiocytic infiltrate in the biopsy was patient- or donor-derived, we performed fluorescent *in situ* hybridisation with probes directed to centromeres of the X- and Y-chromosome (the transplantation was from a male donor to a female recipient). This experiment showed that in the inflammatory infiltrate, both XX and XY cells were present. A double staining with CD68 indicated that the histiocytic cells were indeed donor-derived (*figure 2*). The diagnosis of donor-derived renal sarcoidosis was made.

The contralateral kidney of this same donor was also transplanted in another centre. In the pre-implantation biopsy a patchy infiltrate in the interstitium with giant cells and formation of granulomas without necrosis was present too.

Sarcoidosis is a chronic, idiopathic multisystem, inflammatory disease characterised by the presence of non-caseating granulomas in one or more organs, most often involving the lungs and hilar lymph nodes.^{1,2} Renal

manifestations of sarcoidosis most commonly include hypercalciuria, nephrocalcinosis and nephrolithiasis as a result of a derangement in calcium homeostasis.^{1,3} Sarcoidosis may also directly involve the kidney in the form of GTIN. GTIN is unusual with a reported incidence ranging between 0.7 and 30%.³

Only ten cases of recurrent renal sarcoidosis in the form of GTIN in a renal allograft have been described previously.³ To the best of our knowledge, donor-derived, sarcoid granulomas in a renal allograft have never been described before.

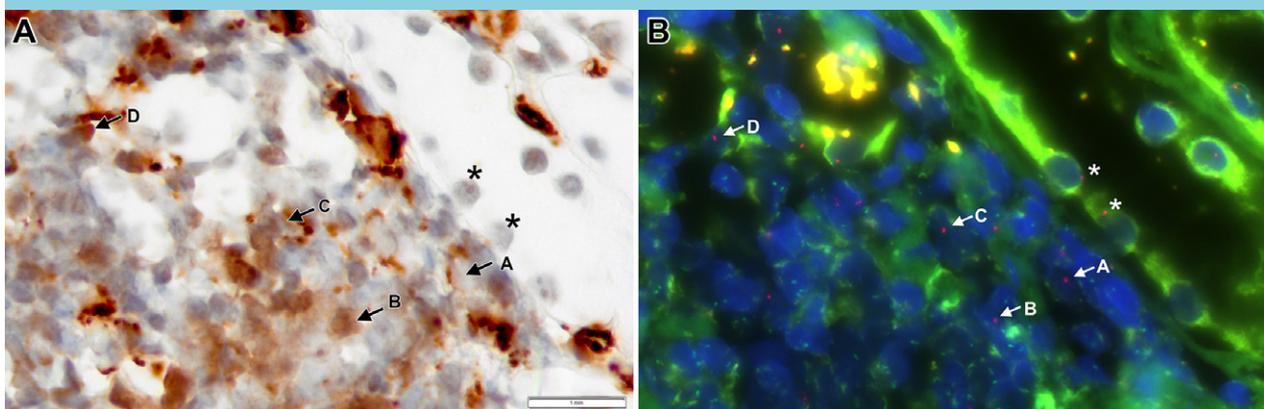
Following the biopsy, her renal function recovered without additional treatment and she could be discharged from hospital in a good clinical condition two weeks after transplantation.

In conclusion, if GTIN is present in a recently transplanted kidney, clinicians should consider the possibility of donor-derived sarcoid granulomas and should not immediately attribute this finding to an acute T-cell mediated rejection.

REFERENCES

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2. Stehlé T, Joly D, Vanhille P, et al. Clinicopathological study of glomerular diseases associated with sarcoidosis: a multicenter study. *Orphanet J Rare Dis.* 2013;8:65.
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Figure 2. Donor-derived histiocytes in the tubulointerstitial inflammatory infiltrate



A (left): CD68 immunostaining. B (right): X- and Y-chromatin fluorescent *in situ* hybridisation (FISH): X = green signal and Y = red signal. Arrows A-D point to histiocytes with cytoplasmic staining for CD68 (left) and with presence of a Y-chromosome (red signal) in the corresponding cells (right), confirming that they are donor-derived. ** = donor-derived tubular epithelial cells, also containing a Y-chromosome.