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Images in Nephrology
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A mysterious 'homesickness'

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An 82-year-old female patient presented with fever and chills of two weeks' duration. Because of pyuria on dipstick, sulfamethoxazole and trimethoprim had been empirically initiated. On admission, temperature was 38.5°C. Blood tests showed increased C-reactive protein and creatinine serum levels (Figure A). White blood cell count was normal. Urinalysis confirmed leucocyturia (350/hpf) but urine culture was negative. Kidney ultrasonography was normal. Intravenous ciprofloxacin and rehydration led to full clinical and biological recovery. Surprisingly, fever quickly relapsed at home, in association with acute renal failure, systemic inflammation and sterile pyuria with eosinophiluria (Figure A). Ciprofloxacin was stopped, and both clinical and biological parameters were improved. The day after discharge fever and chills recurred with similar laboratory abnormalities (Figure A). A positron emission tomography (PET)-CT was then performed (Figure B), which showed bilateral kidney enlargement and significant renal uptake of 18-fluorodeoxyglucose compatible with acute interstitial nephritis (AIN). The pelvis area was contrastingly silent.

The most common cause of AIN in native kidneys is drug therapy [1]. Although the classic triad of rash, fever and eosinophilia was incomplete in our patient, the clinical picture with recurrent full recovery while in hospital and rapid relapse at home pointed out to a hidden offending agent. Well-targeted history-taking finally disclosed that the patient took omeprazole on her own at home for dyspepsia. Here, the clinical history with inadvertent rechallenge with a drug known to be a common cause of AIN in the elderly [2], as well as the finding of PET-CT, made a kidney biopsy hardly ethical. The subsequent clinical and biological evolution after omeprazole removal was favourable without corticosteroid treatment.

The pathophysiology of PPI-associated AIN remains unclear [3], but drug withdrawal permits a rapid improvement of renal function in most cases. The use of corticosteroids remains debated [4]. Such a hypermetabolic PET-CT pattern of both renal parenchyma has never been previously reported in drug-induced AIN. This supports the view that PET-CT might prove useful in inflammatory tubulointerstitial diseases.

Conflict of interest statement. None declared.

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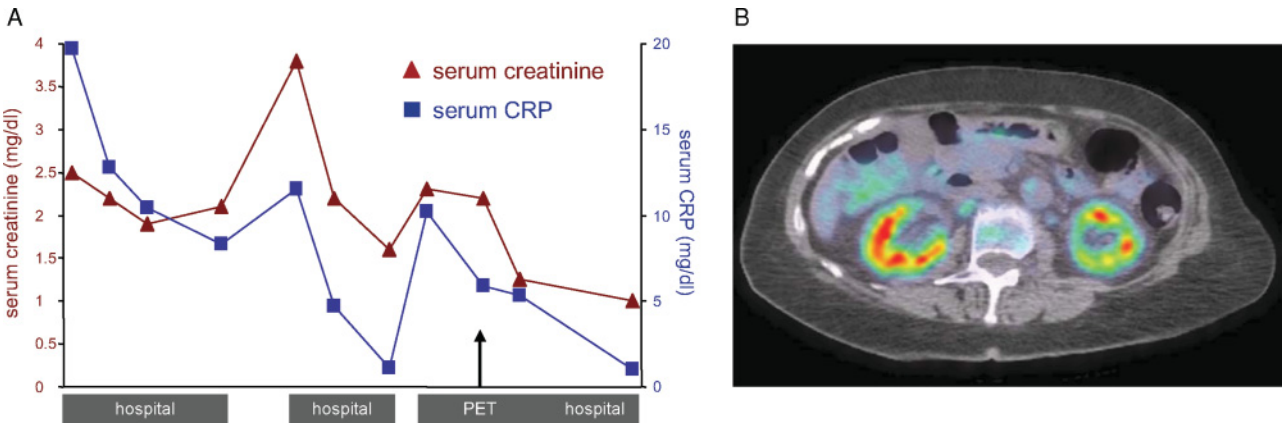

Fig. (**A**) Evolution of C-reactive protein and creatinine serum levels. (**B**) PET-CT performed 102 minutes after intravenous injection of 18-fluorodeoxyglucose showing significant tracer uptake in enlarged kidneys.