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P401 -Retrospective review of acute interstitial nephritis: a 12-year single centre experience

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INTRODUCTION:
While substantial effort and progress have been made in improving the care of glomerular disorders, the clinical significance of tubulointerstitial diseases, acute interstitial nephritis (AIN) in particular, should not be underestimated, given that it accounts for about 15-25% of acute kidney injury. Through early recognition and initiation of effective treatment strategies including cessation of offending agents and commencement of corticosteroid therapy, the clinical outcome of AIN has improved considerably. However, a delay in treatment may result in progression to established interstitial fibrosis and subsequent development of chronic renal impairment.

AIMS:
- To identify the incidence of AIN in our cohort of patients undergoing native renal biopsy
- To determine the frequency of granulomatous interstitial nephritis (GIN) within this group
- To examine extra-renal clinical features (fever, rash, eosinophilia) and their utility in helping establish a diagnosis of AIN
- To evaluate whether corticosteroid therapy facilitates renal recovery

METHODS:
We conducted a retrospective analysis of all cases of histologically-proven AIN by reviewing 937 native renal biopsies performed at our institution over a 12-year period from 2007 to 2018.

RESULTS:
From a total of 937 native renal biopsies, AIN was present in 77 (8.2% of all biopsies), and 8 showed GIN (10% of AIN patients). Clinical presentation in all cases had been with acute kidney injury and urinary abnormalities (haematuria and/or proteinuria). Systemic features (such as fever, rash, arthralgia) were present in 9 patients (12%) and peripheral blood eosinophilia in 11 (14%).

The cause of AIN was attributed to drugs (mainly non-steroidal anti-inflammatory drugs, proton pump inhibitors, antibiotics) in 40 patients (52%), infection in 10 (14%), systemic disease (including sarcoidosis and tubulointerstitial nephritis with uveitis syndrome – TINU) in 8 (10%). In the remaining 19 patients (24%) no cause was identified.

53 patients (69%) were treated with corticosteroids – 43 of these showed a significant improvement in renal function (creatinine fall >50%), with 20 returning to their baseline level of renal function. Of the 24 patients (31%) not given corticosteroids, only 7 returned to their baseline level of renal function. Overall, median serum creatinine of AIN patients at the time of biopsy was 620 µmol/L (range 132-1100), and at the time of recovery was 115 µmol/L (range 60-234).

CONCLUSIONS:
In summary, biopsy-proven AIN was present in a significant proportion of our study population. All presented with acute renal impairment and urinary abnormalities, but extra-renal manifestations (including peripheral blood eosinophilia) were seen only in a minority of patients. Only 10% of AIN biopsies showed

GIN, and most cases were associated with sarcoidosis or TINU. Good renal outcomes were observed in our cohort irrespective of the underlying cause of AIN, particularly in patients treated with corticosteroids; nearly two-thirds showed stabilisation or recovery of renal function.

AIN is a potentially reversible cause of acute kidney injury. Early diagnosis is important to allow appropriate intervention such as cessation of causative drugs or identification of other underlying causes. Anecdotally in our cohort, renal outcomes were better in patients treated with corticosteroids.