Olecranon bursitis in chronic haemodialysis patients

Sir,
Osteoarticular diseases are frequent complications observed in chronic haemodialysis patients. Aetiologies of articular manifestations are numerous, including secondary hyperparathyroidism, crystal arthropathy such as gout and hydroxyapatite deposition, amyloidosis, aluminum overload and infectious processes [1]. We report here swelling of olecranon bursa observed over a 9-month period (during years 1999 and 2000) in six of our 240 chronic haemodialysis patients.

Five men and one woman were affected; their mean age was 65 with a range between 49 and 78 years. They had been on intermittent haemodialysis treatment for an average duration of 18.8 ± 9.8 months. These six patients were all dialysed in chairs. For vascular access, five patients had a native arteriovenous fistula (three brachio-cephalic and two radial) and one a synthetic graft. All accesses were on the left arm since our six patients were right-handed. The causes of underlying renal diseases were diabetic nephropathy (1), nephroangiosclerosis consequent to hypertension (1), rapidly progressive glomerulonephritis (1), Alport disease (1), membranoproliferative glomerulonephritis (1), and unknown chronic glomerulopathy in the last patient. None of the patients had previously presented any articular problems except for one patient who was known for gout.

During the period mentioned above, the six patients complained of at least one episode of swelling of one of their olecranon bursae, usually ipsilateral to their vascular access. Three patients had repeated episodes on the same side and one patient had a second episode on the opposite side (i.e. contralateral to the vascular access).

The swollen olecranon bursae were all punctured at least once but only four effusions were analysed. The removed fluid was usually sero-sanguinous and cloudy; however, no crystals were identified and cultures were negative. The fluids nevertheless, contained inflammatory elements with neutrophils (from $0.10 \times 10^{12}$ to $0.58 \times 10^{12}$), lymphocytes (from $0.06 \times 0.28 \times 10^{12}$), and monocytes (from $0.13 \times 0.84 \times 10^{12}$), as well as erythrocytes (from $0.07 \times 1.96 \times 10^{12}$). Besides avoiding prolonged pressure on the affected joint, the initial treatment was based on anti-inflammatory agents with a poor response; in one case, an intra-articular steroid injection was attempted with success. After some delay (generally more than 2 weeks), the olecranon bursitis subsided with no more specific intervention, except for repeated aspirations in two patients for recurrent swelling.

All patients were immune for hepatitis B and none had evidence of hepatitis C. The values for serum calcium and uric acid were all in the normal range. Only two patients had concomitant serum phosphate concentrations above...
2.0 mmol/L. Serum aluminium was below detectable limits. Protein electrophoresis was also normal. The mean value for serum β2-microglobulin was 3092 nmol/L (with a range from 1390 to 6592).

Such a swelling of the olecranon bursa has been reported previously [2,3]. The precise explanation for this phenomenon remains unknown although a few hypotheses have been made. Since the usual causes for bursitis (namely infection and crystal arthropathy) were excluded in those cases, but because inflammation appeared to be involved, it has been proposed that uraemia per se could be a factor. Others have suggested that a mechanical factor was possibly responsible since the patients were resting on their elbow during haemodialysis treatment. Our present observations would be in agreement with this last view. Indeed, the fact that olecranon bursitis occurred almost exclusively on the side of the vascular access tends to favour a mechanical phenomenon from repeated micro-trauma and prolonged pressure over the elbow. Further studies may provide different explanations; in the meantime, dialysis physicians should be aware of this potential complication in their chronic haemodialysis patients.

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