Gout, Renal Failure, and Lead Accumulation

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Dear Sir,

We certainly agree with Colleoni and D'Amico [1] that lead accumulation should be considered as a cause of progressive renal damage in patients with gout and renal failure. Nevertheless, their paper deserves several comments.

The first point to discuss is doubtless the diagnosis of primary or secondary gout. In lead nephropathy (LN), gout usually follows renal disease [2]. Among German patients with renal failure and gout, lead accumulation was found in those with renal disease prior to the first episode of gout, but seldom in those who experienced gout first [3]. Sex is also worth considering, since both sexes are equally affected in LN as opposed to primary gout. The patients' sex was not reported in the paper under discussion, but the patients developed gout on average 5 years before the first sign of renal failure. Although subclinical LN might have decreased uric acid clearance several years before increasing serum creatinine, we think that there is no firm support for the statement of these authors that 'this disorder [gout] was also due to excessive lead absorption'.

The second comment relates to the parathyroid hormone (PTH) data. A low-calcium diet has been shown to enhance retention of lead [4]. It might explain the low serum calcium, high PTH and the positive correlation between PTH and magnitude of lead accumulation found by the authors in their gouty patients.

Finally, the handling of lead in the kidney damaged by other diseases is still not well known. Lead is eliminated by filtration and tubular secretion [5]. The finding by Colleoni and D'Amico of no lead accumulation in patients with chronic glomerulonephritis does not exclude the occurrence of an impaired ability to eliminate the environmental lead in other nephropathies associated with tubular abnormalities. Patients with interstitial nephritis or essential hypertension might therefore be more suitable as controls.

In conclusion, if we agree that gouty patients with renal failure should be tested for lead overload, it remains to be established whether this reflects primary lead nephropathy, or whether the renal abnormalities associated with hyperuricemia and hypertension predispose to lead accumulation.

References

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