# Aplastic anemia, membranous nephropathy and mercury

J. Rooney

Sir,

I read with interest the recent case study by Priya *et al*.[1] The authors state that reports of the effects of mercury on the bone marrow are rare, citing only two previous cases, and state also that membranous nephropathy has not previously been reported for injected elemental mercury.[1] However, I note that both aplastic anemia,[2] and a range of nephropathies with varied histological findings,[3] have been reported as side-effects of penicillamine, the chelation agent used in the current case.[1] Therefore, it seems worth considering whether penicillamine may have played a contributory or even a casual role in the development of aplastic anemia and membranous nephropathy in this case.

In fact, it is partly due to the risk of such serious reactions to penicillamine, as well as greater efficacy that the chelators sodium 2,3 dimercaptopropanesulfate (DMPS) or meso-2,3-dimercaptosuccinic acid (DMSA) are more frequently used to treat mercury toxicity.[4] However, it is worth pointing out that DMPS and DMSA must be used with caution in the presence of renal disease as they both undergo renal excretion. Inappropriate use in the presence of renal failure can lead to a paradoxical increase in blood mercury levels–particularly in the situation where there are deposits of mercury within the body.[5,6] Such renal failure was not reported by Priya *et al*., and in fact, considering that the vast majority of blood-borne mercury is protein bound,[7] we can speculate that the presence of a proteinuria in their patient, whilst obviously a pathology with negative consequences in its own right, may have indirectly led to a more rapid urinary mercury excretion.

Finally, Priya *et al*., correctly point out that after acute mercury exposure, urinary levels only remain elevated for a period of weeks, thus posing a diagnostic challenge.[1] At least for cases of chronic exposure, analysis of urinary porphyrins has shown some promise in adults in detecting mercury exposure (particularly where the patient carries the coproporphyrinogen oxidase 4 (CPOX4) polymorphism).[8]

## Article information

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