Determination of 4,4'-Methylenebis(2-chloroaniline) in Urine

To the Editor:

We read with interest the paper by Okayama et al. (1) describing a liquid-chromatographic method for measuring 4,4'-methylenebis(2-chloroaniline) (MBOCA) in urine by use of solid-phase extraction and electrochemical detection. For over 10 years we have been involved in the routine monitoring of workers who are exposed to MBOCA. We do this by measuring MBOCA in the urine (2). Initially, we used a gas chromatography–electron capture method but for the past five years we have used HPLC with electrochemical detection. In their publication, Okayama et al. do not appear to have taken account of labile conjugates of MBOCA that are present in the exposed worker's urine. The presence of these conjugates was first suspected when we observed that if freshly collected urine samples were analyzed immediately, then left at room temperature for 24 h and reanalyzed, there was a two- to fivefold increase in the apparent MBOCA concentration (3). Investigations showed that after mild hydrolysis of the urine sample by heating at 50 °C for 30 min, similar increases were found. Our recent studies have produced strong evidence that these labile conjugates are N-glucuronides of MBOCA (4). We have also confirmed the presence of a minor metabolite, N-acetyl MBOCA (5), reported by Ducos et al. (6). We feel that it is important to establish that N-acetyl MBOCA does not interfere in the HPLC analysis described by Okayama et al. (1).

We recommend to our occupational physicians that a biological monitoring strategy for workers who are exposed to MBOCA should include the measurement of urinary MBOCA after hydrolysis. This procedure will overcome changes in the "free" MBOCA concentration caused by temperature fluctuations during transport of the unfrozen urine samples from the workplace to the laboratory or due to thawing of frozen specimens.

References

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Serum Magnesium as Affected by Drugs

To the Editor:

I read with interest the Technical Brief of Zemb-Palko and Lachar (Clin Chem 1988;34:1913) concerning the effect of drugs on serum magnesium concentration. The occurrence of hypomagnesaemia in their patients was ascribed to the effects of aminoglycoside antibiotics, diuretics, or cyclosporine. Although it is well documented that these drugs can precipitate hypomagnesaemia, it is also possible that some of the disease states noted by the authors could have been responsible for the low magnesium concentrations found.

Of the patients they studied, 18 had chronic renal failure, 16 unspecified cardiac disease, eight trauma, and eight renal transplants. Others have documented that hypomagnesaemia may occur in chronic renal failure (1, 2); after acute myocardial infarction (3); after surgery, trauma, and burns (4–6); and after renal transplantation (7). I agree with the conclusion of Zemb-Palko and Lachar that serum magnesium should be monitored in patients receiving medication that affects its metabolism, and I would add to their list of drugs the anti-mitotic cisplatin, which can cause serious renal magnesium wasting in up to 50% of those patients receiving it (8). I would, however, disagree with the implication that the hypomagnesaemia they observed was solely the result of drug treatment, and would further suggest that, in some cases at least, it was a result of the disease process itself.

References

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