Association of Bartter’s syndrome with vasculitis

Sir,

Deliska et al. describe a 25-year-old woman who was given a diagnosis of Bartter’s syndrome. However, the patient had a number of features that have not been described in Bartter’s syndrome: she was hyponatraemic, her serum potassium concentration was only mildly reduced to 2.8 mM, and her spontaneous urinary chloride concentration was unusually low (67 mEq/day) for classical Bartter’s syndrome. The authors did not report on a long history of known hypokalaemia in their patient—as would be typical for Bartter’s syndrome—nor did they report measurements of urinary prostaglandins, the renal distal fractional chloride excretion rate, the results of platelet studies, the results of genetic analyses (concerning the chloride and potassium transporters of the TALH) or a trial using indomethacin and its results.

Therefore, it is by no means clear that the case presented truly has classical Bartter’s syndrome. We propose that the diagnosis of ‘Bartter’s syndrome’ be used in a more specific manner and on the basis of established literature.

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Reply

Sir,

We thank very much Prof. Dr P. Gross for his letter and comment concerning our ‘Case report’. The case we presented was a combination of hypokalaemia and metabolic alkalosis with vasculitis. We reviewed the literature because we had to make a differential diagnosis between Bartter’s syndrome, Gitelman syndrome, Bartter-like syndrome and Milk-Alkali syndrome. The patient satisfied most of the criteria of Bartter’s syndrome with hypokalaemia, metabolic alkalosis, hyperreninemic hyperaldosteronism with normal serum magnesium and disturbed concentrating urine capacity. We agree that it would be suitable for the diagnosis to measure urinary prostaglandins and to make some additional examinations such as genetic analyses but unfortunately we have not the possibility to perform them. A short course with indomethacin has been applied but the patient was unresponsive to that treatment. We supervised our patient subsequently. Nearly 2 years after the clinical manifestation of Bartter’s syndrome she is in a stable condition, with remission of the vasculitis, without clinical manifestation of Bartter’s syndrome. The serum potassium is in the normal range, with a mild alkalosis. She receives only potassium supplements and reduced dairy products in her diet. We hypothesize that the onset of vasculitis is the reason for the clinical manifestation of mild Bartter’s syndrome.

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