Letters and Replies

Two underlying diseases causing hypercalcaemia?

Sir,
In your recent December issue of NDT, Schmaldienst et al. [1] presented an interesting patient with hypercalcaemia secondary to primary hyperparathyroidism (HPT) with co-existing Paget’s disease of bone (instead of two underlying diseases causing hypercalcaemia, as stated in the title). From the presentation and the outcome, it is safe to assume that HPT was the only pathology contributing to the increased serum calcium levels. The presence of hypophosphataemia and the correction of hypercalcaemia by removing the adenoma without treating the Paget’s disease support our argument. Thus, there was a single rather than ‘two underlying diseases’ that resulted in a hypercalcaemic state. We would also like to correct the authors’ suggestion that ‘alkaline phosphatase is a very reliable marker of increased osteoclast activity’. Bone specific alkaline phosphatase, or—in the absence of liver disease—total serum alkaline phosphatase, signify enhanced osteoblastic rather than osteoclastic activity. Furthermore, we should add that cytokines, such as IL-6 and RANK-L, have been recognized as contributing factors in the abnormal bone metabolism in Paget’s disease [2] and the actions of parathyroid hormone (PTH) [3,4] on bone. Finally, the authors did not comment on why they did not consider the use of bisphosphonates after establishing the diagnosis of Paget’s disease. Indeed, characteristic findings on the pelvic radiographs, together with a positive isotope bone scan and the elevated alkaline phosphatase signify active disease and constitute strong grounds in considering initiation of appropriate treatment. Also, at this point we should stress that alkaline phosphatase is probably the most useful marker in Paget’s disease, but is unreliable as a marker for HPT, especially in this case where PTH was reported as normal (although we believe that PTH is probably the most useful marker in Paget’s disease, but is unreliable as a marker for HPT, especially in this case where PTH was reported as normal (although we believe that PTH was inappropriately elevated taking in to account the high serum calcium concentration).

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Reply

Sir,
We appreciate Drs Pazianas and Zaidi’s interest in our case report and their valuable comments. We agree that in our case hypercalcaemia was due to primary hyperparathyroidism and not to Paget’s disease of bone. Thus, the title ‘A patient with evidence of two underlying diseases able to cause hypercalcaemia’ would have been more appropriate. We regret the typographic error regarding alkaline phosphatase as a marker of osteoblastic activity. The comment of Drs Pazianas and Zaidi on IL-6 and RANK-ligand is indeed a very interesting point. However, the determination of these cytokines is not yet established in clinical practice and usually not available for routine purpose, thus we did not mention these aspects in our teaching point. As discussed in our paper, the main diagnosis in our patient was primary hyperparathyroidism, which could be treated successfully by parathyroidectomy [1]. At the time of diagnosis Paget’s disease was clinically silent in our patient. We agree that the serum parathyroid hormone levels in the presence of hypercalcaemia were inappropriately high, and tried to stress this fact in the discussion section of our teaching point. As the patient was clinically asymptomatic and pre-operatively elevated levels of alkaline phosphatase returned to normal, indicating a low disease activity, we felt no urgent indication for bisphosphonate treatment [2]. Furthermore, the effect of bisphosphonate (in particular with high doses as recommended for Paget’s disease) on bone alterations after surgical correction of primary hyperparathyroidism is unknown.

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