

Correspondence

Metabolic Acidosis: Treat the Symptom or Cause of the Symptom?

The description of bone decalcification as a consequence of endocrinologic pathologies or "other disturbances," as it is stated in the publication of Miekeley et al¹ seems to be over simplistic. The authors point out that the elevated mineral levels of calcium, magnesium, strontium and phosphorus in the hair tissue are related to the hyperactivity of the parathyroid and the thyroid glands. By extirpation of the parathyroid glands, the mineral levels returned to their normal concentrations. The choice of using surgical intervention to treat the endocrinologic pathologies avoids dealing with the original problem. It would be worthwhile to explore the underlying causes of the hyperactive parathyroid glands.

As was described earlier,² when metabolic acidosis drives the intracellular pH so low that it interferes with homeostasis - alkali minerals, mainly calcium, are transferred from the blood to the inside of the cells to neutralize the acidic state (lactic, phosphoric, sulphuric, hydrochloric acids). When the well-buffered blood concentration of calcium sinks below the 9 mg% level, the parathyroid gland (PTG) is triggered to produce its hormone (PTH). PTH osteoclastic activity restores the blood level of calcium and this process is expressed by elevated calcium, magnesium and in certain cases phosphorus levels in hair and most likely in cells as well. Increased excretion of these elements in the urine can also be observed.³

The detrimental effect of the osteoclastic activity of the parathyroid gland induced by the presence of metabolic acidosis is well described by Wachman et al.⁴ stating that "the increased incidence of osteoporosis with age may present, in part, the result of a lifelong utilisation of the buffering capacity of the basic salts of bone for the constant assault against pH homeostasis. The loss of as little as 2 meq of calcium per day would, over a decade,

assuming a total body content of 1 kg, account for a 15% loss of inorganic bone mass in an average individual".

The solution chosen by Miekeley et al.¹ of eliminating the negative effects of the hyperactivity of the PTG, by extirpation of the parathyroid glands is characteristic. Instead of investigating the reason for the hyperactivity of the PTG, they have chosen to remove the symptom-causing gland. As a consequence, the osteoclastic activity of the PTH was definitely eliminated, but at cost of the other essential metabolic activities related to the PTG, such as:

- (a) control of serum levels of calcium and phosphorus;
- (b) regulation of urinary excretion of phosphate and calcium;
- (c) removal of calcium from bone, particularly if the dietary intake of calcium is inadequate;
- (d) regulation of serum alkaline phosphatase in case changes in bone have been produced; and
- (e) activation of vitamin D in renal tissue by increasing the rate of conversion of 25-hydroxycholecalciferol to 1,25 dihydroxycholecalciferol.⁵

As it was described earlier,² the presence of metabolic acidosis will trigger a metabolic response, whether it is caused by lactic acid production (fermentation) instead of oxidative phosphorylation, or by consuming a diet with elevated protein content (which will produce an acid ash end product- phosphoric acid), will trigger a metabolic response. The transfer of serum calcium to the tissues is attempting to neutralize the acid present there by salt formation (calcium lactate, calcium phosphate) and will trigger the production of PTH in order to maintain the 9-11% serum calcium level by its osteoclastic activity. The result will be expressed in loss of bone and in dental caries, and it will only be a question of time as to when the effect will also be demonstrable by bone densitometry. In the article of Miekeley¹ it is stated

that hair analysis is not furnishing information for osteoporosis.

This evidence provides a clear understanding of how hair mineral analysis can furnish valuable information related to the development of osteoporosis, in contrast to the statement by Miekeley¹

In conclusion, it can be stated that hair tissue mineral analysis is not a diagnostic tool. Rather it is a very sensitive predictive information source about the dynamic expression of metabolic processes. By following up on this information, a valuable biochemical picture can reveal what is happening 'behind the scene'. It could be compared to the analysis of ascorbic acid, which is not a diagnostic tool for detecting the presence of scurvy. But if the deficiency, indicated by the ascorbic acid analysis, is not followed up by proper supplementation, the appearance of scurvy can definitely be expected.

By correcting the nutritionally or environmentally induced acidic state of the body by a proper supplementation program-including calcium, magnesium and vitamin D-the metabolic acidosis could be "neutralized." Inclusion of magnesium is essential, since it triggers the production of calcitonin, produced mainly by the thyroid gland.^{6,7} Calcitonin promotes Ca deposition to bone, inhibiting resorption and counteracting PTH activity. In this case, as the hyperactive PTG returns to normal, this process leads to the normalization of the tissue levels of calcium, magnesium and phosphorus. This kind of normalization has been already reported.⁸

George Tamari, Ph.D
Anamol Laboratories Ltd.
83 Citation Dr.
Concord, ON L4K 2Z6

References

1. Miekeley N, de Carvalho Fortes LM, Porto da Silveira CL, Lima MB: Elemental anomalies in hair as indicators of endocrinologic pathologies and deficiencies in calcium and bone metabo-

lism. *J Trace Elem Med Biol*, 2001; 15: 46-55.

2. GM Tamari: Functional Diagnosis in Nutritional Medicine *J Orthomol Med*, 1998;13:85-88
3. Bland J: Dietary calcium, phosphorus and their relationship to bone formation and parathyroid activity. *J John Bastyr Coll Naturopath Med*, 1979; 1: 3-6.
4. Wachman A, et al: Diet and Osteoporosis. *The Lancet*. 1968; May 4: 958-959.
5. Harper HA, Rodwell VW, Mayes PA: *Review of Physiological Chemistry. 17th Ed.* 1979; p 517
6. Roseblatt M, Kronenberg HM, Potts JT: Parathyroid hormone: Physiology, chemistry, biosynthesis, secretion, metabolism and mode of action. *Endocrinol*, 1988; 2: 848-891.
7. Parfitt AM, Bone and plasma calcium homeostasis. *Bone*, 1987; 8:1-8.
8. Wales PJ: *Private communication.*