Vitamin B₆ Deficiency on Hemodialysis Causing Sideroblastic Anemia

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Dear Sir,

Vitamin B₆ deficiency is prevalent within the dialysis population and is known to cause sideroblastic anemia in experimental animals [1]. We encountered an instance of severe sideroblastic anemia associated with a selective vitamin B₆ deficiency, after commencement of maintenance hemodialysis.

A 62-year-old female with analgesic nephropathy became symptomatically anemic 9 months after commencement of hemodialysis with a fall in hemoglobin to 4 g/dl (MCV 98 fl) and required frequent regular blood transfusions. Anorexia, lethargy and finger dysesthesia were also noted. Bone marrow aspiration showed reduced but predominantly normoblastic erythropoiesis, abnormal numbers of sideroblasts (8% ring sideroblasts) and increased size of siderotic granules. Granulopoiesis, other cell lines and posterior iliac crest trephine were normal and iron stores were increased. Vitamin B₁₂ and folate deficiency were excluded. Blood films showed some markedly hypochromic red cells, consistent with secondary acquired sideroblastic anemia. None of her routine medications are known to cause sideroblastic anemia and she was a nondrinker. Vitamin B₆ markers were depressed, with a serum vitamin B₆ level of 19 nmol/l (NR = 50–100) (4.8 ng/ml), erythrocyte aspartate amino-transaminase (EAST) of 122 U/g Hb (NR > 330), EAST and pyridoxal-5-phosphate of 280 U/g Hb (NR > 600). The calculated stimulated index was 2.23 (normal < 1.25). Her multivitamin capsules, containing 2 mg pyridoxine, were taken 4 times per week and she had a low dietary

vitamin B₆ intake. Pyridoxine 25 mg q.i.d. was commenced. Repeat vitamin B₆ estimations 1 month later were borderline and had normalized at 4 months. The reticulocyte index increased from 0.19 to 1.85 and her mean trough Hb also increased from 4.8 to 6.2 g/dl with pyridoxine repletion. Transfusion requirements ceased, finger dysesthesia disappeared and a marked improvement of the patient's sense of well-being occurred.

Sideroblastic anemia is a heterogeneous group of anemias characterized by ineffective erythropoiesis and large numbers of ringed sideroblasts in the bone marrow [1]. Although in some cases large pharmacological doses of pyridoxine can correct the hematological abnormality, associated pyridoxine deficiency is unusual in man. Pyridoxal-5-phosphate, which is enzymatically synthesized from pyridoxine, is a coenzyme that is required for proto-porphyrin synthesis. Iron is then inserted into the porphyrin ring within the mitochondria to form heme. Impairment of this biosynthesis results in passive mitochondrial iron accumulation, formation of pathological sideroblasts, cellular death and sideroblastic anemia. In this case, vitamin B₆ deficiency may have resulted in reduced protoporphyrin synthesis and subsequent sideroblastic anemia.

Vitamin B₆ (pyridoxine, pyridoxamine and pyridoxal) are water-soluble vitamins, in which deficiencies are common in end-stage renal disease, hemodialysis and peritoneal dialysis populations [2, 3]. Requirements vary in the dialysis population from 2.5 to 10 mg/day of pyridoxine hydrochloride, assessed by normalization of erythrocyte glutamic pyruvic transaminase index [2].

Pyridoxine-Deficient Anemia on Dialysis 675

Kopple JD, Mercurio K, Blumencrantz JJ, Jones MR, Tallos J, Roberts C, Card B, Saltzman R, Casciato D, Swendseid M: Daily requirements for pyridoxine hydrochloride in chronic renal failure. *Kidney Int* 1981;19:694–704.

Stone WJ, Warnock LJ, Wagner C: Vitamin B₆ deficiency in uraemia. *Am J Clin Nutr* 1965;28:950–957.

Supplements are recommended to compensate for excessive losses on dialysis and the often inadequate diet of these patients. Hence, in dialysis patients with an unexplained anemia or pathological sideroblasts on bone marrow examination, evaluation of vitamin B₆ status should be performed and pyridoxine therapy considered.