Biochemical evidence for a deficiency of vitamin B_6 in the carpal tunnel syndrome based on a crossover clinical study

(pyridoxine)

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ABSTRACT In a patient with severe carpal tunnel syndrome and a significant deficiency of vitamin B_6, the evidence for the deficiency was an extraordinarily low basal specific activity of the glutamic-oxaloacetic transaminase of the erythrocytes (EGOT). This enzyme was also deficient in pyridoxal phosphate. The patient was treated with the recommended dietary allowance of pyridoxine, 2 mg/day, for 11 weeks, then 100 mg/day for 12 weeks, a placebo for 9 weeks, and again pyridoxine at 100 mg/day for 11 weeks. Sixty-one monitorial assays of EGOT over 48 weeks supported the following interpretations. (*i*) His diet permitted the development of a debilitating carpal tunnel syndrome. (*ii*) Treatment with pyridoxine at 2 mg/day reduced the deficiency of EGOT activity from about 70% to 50%, maintained a deficiency of pyridoxal phosphate, and relieved but allowed a marginal syndrome. (*iii*) Treatment at 100 mg/day for 12 weeks nearly achieved a "ceiling" level of EGOT and eliminated the deficiency of pyridoxal phosphate. (*iv*) After placebo for 7 weeks, the deficiencies of EGOT activity and pyridoxal phosphate reappeared, and clinical symptoms become worse. (*v*) Retreatment at 100 mg/day reestablished a "ceiling" EGOT, with no deficiency of pyridoxal phosphate, and the patient was asymptomatic. These data also support the concept that a deficiency of vitamin B_6 is significant in the etiology of the carpal tunnel syndrome. Mechanistically, a state of deficiency of the coenzyme seems to lower the level of the apoenzyme; a state of no deficiency of the coenzyme regulates a ceiling level of the transaminase. The latter state is presumably desired for health.