Clinical Digest

GERONTOLOGY

Considering Cobalamin Deficiency in Elders

An estimated 15% of people over age 60 have an undiagnosed cobalamin (vitamin B_{12}) deficiency, and there's evidence that this isn't just a normal function of aging. Very often, say researchers from Hôpitaux Universitaires de Strasbourg, Strasbourg, France, the deficiency results from malabsorption, the effects are devastating, and the condition can be treated.

The investigators studied 92 elderly patients, all of whom had well established food-cobalamin malabsorption (FCM) and serum cobalamin levels of less than 200 pg/mL. The most common clinical manifestations were neurologic or psychological, such as mild sensory polyneuropathy, confusion or impaired mental functioning, and debilitation. In fact, reduced or absent reflexes (such as knee jerk) were noted in 36 patients. At least one third of the patients had hematologic abnormalities, including anemia, leukopenia, thrombopenia, and pancytopenia.

Although 10 patients were considered to have idiopathic FCM, all the others had at least one associated condition, such as nonspecific atrophic gastritis, *Helicobacter pylori* infection, diabetes treated with metformin, or esophageal reflux.

Cyanocobalamin PO was administered to 28 of the patients. The rest were treated with cyanocobalamin IM. In all cases, the serum cobalamin levels rose to above 200 pg/mL within one month. Within three months, blood count parameters improved in all patients and were corrected in 58. Eighteen patients with peripheral neuropathy and 14 with asthenia recovered, as did two with jaundice and one with combined medullary sclerosis. Even the patients with dementia showed mild improvement. Long-term data (based on a mean follow-up of one year) were available for 26 patients treated with either cobalamin IM (13) or PO (13). At follow-up, all of these patients had normal cobalamin levels and improved total homocysteine levels and blood counts. No hematologic abnormalities were found in 11 of the 13 patients given cobalamin PO.

Source: *Am J Med.* 2005;118:1154–1159.

NEUROLOGY

Cognitive Decline After CABG

Studies have indicated that coronary artery bypass grafting (CABG) surgery carries a risk of postoperative cognitive impairment, with a reported incidence of up to 33% at 12 months. But, according to researchers from Johns Hopkins University, Baltimore, MD, most of those studies—especially the long-term ones—have not compared patients who've undergone CABG to control patients with similar degrees of cardiovascular and cerebrovascular disease. It's not clear, therefore, whether the postoperative decline is due to the CABG surgery, to surgery in general, or to vascular pathologies of the aging brain.

To investigate these matters, the researchers randomly selected 380 patients from four groups: patients who'd had CABG for coronary artery disease (CAD), patients with CAD who'd undergone off-pump coronary surgery without cardiopulmonary bypass, control patients under medical management for CAD who'd had no surgery, and heart-healthy control patients.

The researchers found no evidence that the cognitive test performance of patients after CABG differed significantly from that of the other two groups of patients with CAD over a one-year period. Three months after baseline measurements, all groups had improved, though the three groups with CAD remained proportionately lower compared with the heart-healthy group. From three to 12 months, all groups showed minimal intrasubject changes, and the researchers saw no consistent differences between the CABG and off-pump groups.

Nevertheless, when the patients themselves reported on cognitive symptoms, those in both surgery groups were more likely to talk about change for the worse in personality, memory, and reading. These adverse changes (particularly memory changes in the CABG group) persisted at 12 months. Reports of memory problems were more common among patients with higher depression scores.

All three groups with CAD performed significantly lower at baseline than the heart-healthy control group in executive function, psychomotor speed, and motor speed. The researchers suggest that some degree of cognitive dysfunction demonstrated after CABG may be secondary to cerebrovascular disease present before surgery.

The lack of cognitive decline over 12 months after CABG is striking, the researchers say, though they acknowledge that their investigation does not address the issue of "late decline"—which, according to some reports, occurs between six months and five years after surgery.

Source: *Neurology.* 2005;65:991–999.