By Dee Rapposelli

Highlights From Digestive Disease Week

Normalization of Vitamin D Status May Reduce Risk of Surgery and Hospitalization in Patients with Inflammatory Bowel Disease

Low plasma 25-hydroxy vitamin D (25[OH]D) levels, defined as less than 20 ng/mL, were associated with higher C-reactive protein levels and increased risk of surgery and hospitalizations in patients with inflammatory bowel disease (IBD). The findings were presented by Ashwin N. Ananthakrishnan, MD, MPH, of Harvard Medical School in Boston, Massachusetts, during the American Gastroenterological Association/American Society for Gastrointestinal Endoscopy Presidential Plenary Session at Digestive Disease Week, held May 19–21, 2013 in Orlando, Florida. Dr. Ananthakrishnan explained that most studies that examine the immunologic role of vitamin D in IBD have been retrospective and show a weak correlation between vitamin D status and disease activity. Dr. Ananthakrishnan and his team, using a prospective study design, sought to capture the occurrence of first-time IBD-related surgery and hospitalization in patients who had vitamin D values on record and to record median C-reactive protein levels.

The study included 3,217 patients. During a median follow-up of 8 years, 16% of these patients underwent IBD-related surgery, and 40% had IBD-associated hospitalizations. One third of patients were vitamin D–deficient, and vitamin D levels were insufficient (25[OH]D <30 ng/mL) in an additional 27% of patients. Low plasma 25(OH)D levels were associated with an increased risk of surgery and IBD-related hospitalizations compared with plasma 25(OH)D levels of greater than 30 ng/mL.

Of patients with vitamin D insufficiency or deficiency, normalization of plasma 25(OH)D levels occurred in 43%. Patients whose 25(OH)D levels normalized were less likely to require IBD-associated surgery (odds ratio [OR], 0.48; 95% confidence interval [CI], 0.32–0.70) or hospitalization (OR, 0.51; 95% CI, 0.38–0.69) than patients whose 25(OH)D levels remained below 30 ng/mL. Median C-reactive protein levels were also lower in these patients than in patients who remained vitamin D–deficient.

Predictors of normalization included older age, use of anti–tumor necrosis factor biologic agents, and vitamin D supplementation.

Could Vitamin D Prevent Liver Cancer?

In another presentation at Digestive Disease Week, Lior H. Katz, MD, a visiting researcher at the University of Texas MD Anderson Cancer Center in Houston, reported that vitamin D suppresses proliferation of several hepatocellular cancer (HCC) cell lines and that high dietary vitamin D in experimental models suppresses hepatocyte proliferation and steatosis.

Dr. Katz and his team measured the effect of calcitriol on cell proliferation in HCC cell lines to evaluate in vivo effects of vitamin D in b2SP± murine models predisposed to development of liver cancer. The mice were fed diets containing either 200 or 10,000 IU vitamin D/kg/day for 9 weeks, after which the animals were euthanized and hepatocyte proliferation and the fat content of the liver were analyzed.

A significant increase in CYP24A1 expression was seen, and growth of HCC cell lines was suppressed. Further, steatosis and hepatocyte proliferation was higher in b2SP± mice receiving low-dose vitamin D than in those fed high-dose vitamin D, in which the incidence of steatosis and hepatocyte proliferation was significantly reduced.

Prophylactic NSAIDs Confirmed to Be of Value in Preventing Post–Endoscopic Retrograde Cholangiopancreatography Pancreatitis

A systematic literature review aimed at clarifying the role of nonsteroidal anti-inflammatory drugs (NSAIDs) in the prevention of post–endoscopic retrograde cholangiopancreatography pancreatitis (PEP) found that prophylactic use of NSAIDs decreased the overall incidence of moderate-to-severe PEP. The study was conducted by Saurabh Sethi, MD, and colleagues from the Beth Israel Deaconess Medical Center at Harvard Medical School in Boston, Massachusetts and reported during a poster presentation at Digestive Disease Week. Using EMBASE, PubMed, the Cochrane Central Register of Controlled Trials, and Digestive Disease Week and American College of Gastroenterology databases, the researchers examined placebo-controlled randomized clinical trials (RCTs) that examined the efficacy and safety of prophylactic rectal NSAIDs (ie, diclofenac and indomethacin) in patients (age ≥18 years) with post-PEP.

Of 2,293 RCTs identified and screened for eligibility, 7, inclusive of 2,236 patients, were included in the meta-analysis. The researchers were able to confirm that prophylactic rectal NSAIDs do have an influence on lowering the overall incidence of post-PEP. The relative risk for PEP after prophylactic administration of NSAIDs was determined to be 0.51 (95% CI, 0.40–0.65; $P<.01$). No difference in efficacy or safety was seen between diclofenac and indomethacin, and no deaths or NSAID-related complications were reported in any of the RCTs.