Surgery: A Last Resort

In severe or advanced cases of Crohn's disease, abscesses can develop in chronically inflamed tissues. These abscesses can grow and tunnel through tissue barriers to produce fistulas, or channels between organs. More than one third of patients who have Crohn's disease develop perianal disease involving anal fissures and perianal abscesses and fistulas. These symptoms seldom respond well to conventional therapies (Braunwald E 2001; McNamara DA et al 2004). Surgery may be required to drain abscesses or remove and close fistulas (Danelli P et al 2003). Surgery on inflamed tissue is itself potentially dangerous, and complications are frequent.

Surgery may also be recommended to remove severely inflamed portions of the intestinal tract. The goal of surgery is to preserve as much of the intestine as possible. Surgery commonly involves the colon or small intestine. Occasionally, the end of the intestine that has been left in place will need to be brought to the skin's surface. When this procedure involves the small intestine, it is called an ileostomy. If the procedure involves the colon, it is called a colostomy. Although Crohn's disease may recur after surgery, the symptoms are likely to be less severe and less debilitating than they were previously. However, when the disease does recur, it usually does so at the site of the last surgery.

In patients with ulcerative colitis, surgery is indicated for up to half of patients in the first decade of their illness. At one time, the surgery of choice was removal of the anus and a portion of the lower colon, which resulted in lifelong incontinence and an ileostomy. Newer surgeries, however, have been developed that can preserve fecal continence by using part of the ileum to create a pouch that is connected to the intact rectal sphincter.

The Protective Effect of Folate on Colon Cancer in Ulcerative Colitis

Evidence suggests that people with ulcerative colitis are at increased risk of colon cancer (Mitamura T et al 2002). About 5 percent of people with ulcerative colitis develop colon cancer. The risk of cancer increases with the duration and the extent of involvement of the colon. For people with ulcerative colitis, there are two factors affecting the risk of developing colon cancer. The first factor is that risk increases after 8 to 10 years of having ulcerative colitis. The second is the extent of the disease in the colon. Patients who have ulcerative colitis only in the rectum have the lowest risk. Having the disease in only part of the colon carries an intermediate risk. The greatest risk is for people whose entire colon is diseased (called pancolitis) (Itzkowitz SH et al 2004). It is assumed that chronic inflammation is what causes cancer in ulcerative colitis. This is supported by the fact that colon cancer risk increases with longer duration of colitis, greater anatomic extent of colitis, and the concomitant presence of other inflammatory manifestations (Itzkowitz SH et al 2004).

Two case-control studies have shown that folate may protect against the development of colon cancer caused by ulcerative colitis. The most recent study showed that folate use for at least 6 months reduced the risk of colon cancer by 28 percent in 98 patients who had ulcerative colitis for at least 8 years. Of the patients with ulcerative colitis, 29.6 percent developed cancerous lesions. The greater the dose of supplemental folate consumed, the lower the rate of colon cancer. Scientists concluded that “daily folate supplementation may protect against the development of neoplasia in ulcerative colitis” (Lashner BA et al 1997). Supplementing the diet with vitamin B12 enables the body to metabolize folate better and avoids masking a vitamin B12 deficiency. Vitamin B12 supplementation is important, particularly for older people (when it is less effectively absorbed) and for vegetarians (because vitamin B12 is found only in red meat).
Inflammatory Bowel Disease Raises Homocysteine Levels

A number of studies have shown that patients with inflammatory bowel disease are more likely to have elevated homocysteine levels. In one study, more than 55 percent of patients with inflammatory bowel disease had elevated homocysteine levels (Roblin X et al 2006). The greatest risk factor for elevated homocysteine in patients with inflammatory bowel disease is reduced folate levels (Zezos P et al 2005). Vitamin B12 deficiencies are also frequently encountered (Mahmood A et al 2005). Certain drugs used to treat inflammatory bowel disease, such as methotrexate, are antimetabolites for folic acid, which may help explain why so many patients are deficient in this vital nutrient.

The elevated homocysteine level that is typical in patients with inflammatory bowel disease accounts for a 3-fold higher risk of blood clots and vascular disease (Fernandez-Miranda C et al 2005; Srirajaskanthan R et al 2005). It also helps explain why patients with inflammatory bowel disease are more likely to have early atherosclerosis (Papa A et al 2005). Based on these findings, it is logical that patients with inflammatory bowel disease should take a prophylactic B complex vitamin, with adequate folic acid and vitamin B12.

Inflammatory Bowel Disease and Bone Loss

Osteoporosis is a serious complication of inflammatory bowel disease that has not received adequate recognition despite its high prevalence and potentially devastating clinical effects (Compston JE 1995; Harpavat M et al 2004; Scharla SH et al 1994). Osteoporosis can be caused by inflammatory bowel disease itself or it can be an adverse effect of corticosteroid treatment. Data derived from a retrospective survey of 245 patients with inflammatory bowel disease suggest that the prevalence of bone fractures in people with ulcerative colitis and Crohn’s disease is unexpectedly high, particularly in patients who have a long duration of disease, frequent active phases, and high cumulative doses of corticosteroid intake (Bischoff SC et al 1997; Gassull MA 2003; Vanis N et al 2003). Recent advances in the diagnosis and management of osteoporosis have facilitated early detection of bone loss and identified means by which it may be prevented. Bone-density measurements to predict fracture risk and define thresholds for prevention and treatment should be performed routinely in patients with inflammatory bowel disease (Rogler G et al 2004). For more information, see the chapter Osteoporosis.

Corticosteroids can also contribute to the risk of osteoporosis because of their effects on calcium and bone metabolism. Corticosteroids suppress calcium absorption in the small intestine, increase calcium excretion by the kidneys, and alter protein metabolism. Patients with inflammatory bowel disease who are taking corticosteroids experience a 6.2 percent annual loss of total bone mass compared with only a 0.9 percent annual loss of total bone mass in patients who are not taking corticosteroids. Nutrients that can help protect bone loss include calcium, vitamin D, and vitamin K.