Redetection and growth of colorectal polyps

Hofstad and colleagues present the results of an endoscopic study in which they observed over a three year period, but did not remove, all colorectal polyps of less than 10 mm in diameter. In their study group of 116 patients, aged 50 to 76, they found that although polyps smaller than 5 mm tended to grow, those measuring between 5 mm and 9 mm tended to regress, with the net result that there was no overall change in size. In the subjects aged between 50 and 60, adenomas increased in size and further polyps were detected at follow up colonoscopy more frequently than in older patients. Patients with multiple adenomatous polyps developed greater numbers of new polyps at follow up than those who originally only had a single polyp. Most of the new polyps tended to arise in the right colon.

The interesting finding that adenomas can regress challenges the conventional view that colorectal adenomas, as neoplasms, must relentlessly increase in size unless interventional therapy is given. The concept of the adenoma-carcinoma sequence assumes that this is so, although it has for long been a mystery why, in familial adenomatous polyposis (FAP), only one or two colorectal cancers develop when there may be many thousands of adenomatous polyps.

That adenomas measuring less than 10 mm may regress suggests that they may be subject to environmental restraints. There is evidence from trials of non-steroidal anti-inflammatory drugs in patients with FAP that in both the rectum and duodenum adenomas can be induced to regress, but only if they are small. To explain this phenomenon, it has been proposed that adenomas may not acquire mutations of their proto-oncogenes until they reach a certain size. Without mutated oncogenes, it is possible that adenomas may remain responsive to environmental influences and, perhaps, in the case of colorectal adenomas, 10 mm represents a point of no return at which such mutations render progressive growth inevitable and beyond which chemotherapeutic intervention is impracticable. Conversely, this provides a rationale for the various trials of chemotherapeutic intervention with such drugs as aspirin or sulindac, in patients who have small, but not large, intestinal adenomatous polyps.

It has for long been recognised that adenomatous polyps measuring less than 10 mm only rarely progress to adenocarcinoma, while adenocarcinoma develop in those that measure 10 mm or more and are seen to be increasing in size. It has been assumed that it is probably perfectly safe to leave diminutive adenomatous polyps, of up to 5 mm, in place, only polyps of 10 mm or larger being regarded as significant pre-cancerous lesions. As a result of the work of Hofstad and colleagues, endoscopists now have direct confirmation that this is indeed the case.

The tendency for adenomatous polyps to develop in the right colon later in life is not a new finding. This was reported by one of the Oslo workers among others. It seems probable that a different pathogenetic mechanism is responsible for right sided adenomas compared with left sided ones. It is clear that cancer prevention for at risk subjects aged between 50 and 60 requires colonoscopy rather than flexible sigmoidoscopy.

The apparent falling off in new polyp formation in patients over the age of 60 is a further important indicator to endoscopists that these people are at reduced risk once their colons have been cleared of polyps and a policy of discharge from surveillance, at that age, of most people with a 'clean' colon after removal of a small adenomatous polyp, provided it was solitary, is probably correct.

I C TALBOT

Academic Department of Pathology, St Mark's Hospital, Harrow, HA1 3UJ