Obesity Not a Factor in Colorectal Screening

BY ALICIA AULT
Associate Editor, Practice Trends

WASHINGTON — People who are overweight or obese appear to take advantage of colorectal cancer screening opportunities at the same rate as normal-weight Americans.

Several studies have indicated that people with a higher body mass index (BMI) do not seek out screening for breast and colon cancer.

But Dr. Deborah A. Fisher, of Duke University, Durham, N.C., and Durham Veterans Affairs Medical Center, and her colleagues determined that overweight and obese residents of North Carolina access fecal occult blood tests, flexible sigmoidoscopy, and colonoscopy at the same rate as those who are normal weight.

At the annual Digestive Disease Week, she presented an analysis of the North Carolina Colon Cancer Study, a case-control population-based study. The study used height and weight measurements to calculate BMI, but information about colon cancer screening was self-reported by patients.

The primary outcome was whether the patient was current for any colon cancer screening test, which included a fecal occult blood test in the past year, a colonoscopy within the past 10 years, a flexible sigmoidoscopy within the past 5 years, or a barium enema within the past 5 years.

Among the 928 patients, the average age was 67 years; 29% were normal weight (BMI 18-24.9 kg/m^2), 39% were overweight (BMI 25-29.9), 19% were obese category I (BMI 30-34.9), 9% were obese category II (BMI 35-39.9), and 4% were obese category III (BMI 40 and up).

Across all the BMI categories, the percentage of those who had undergone screening ranged from 54% to 67%, the authors said.

The overall screening rate of 61% was comparable to other populations that have been studied, she said. Thus, the differences in screening behavior between obese and normal-weight people seen with other cancers may not be true of colorectal cancers, she said.

Dr. Fisher suggested that the increased risk of colorectal cancer in obese people that has been documented in several studies "may be due to biology and not lower screening rates in this group."

Dr. Fisher reported no disclosures. The study was supported by a National Institutes of Health grant. ■

NBI Colonoscopy Had No Advantage Over White Light

BY JOHN R. BELL
Associate Editor

WASHINGTON — Narrow-band imaging colonoscopy appears to offer no advantage over white-light colonoscopy in detecting colorectal neoplasia, according to clinical trial data presented at the annual Digestive Disease Week.

Modern colonoscopy can limit the miss rate for detection of adenomas to 13%, compared with the often-cited rate of 24%. But the use of narrow-band imaging (NBI) colonoscopy, which uses only blue (415-nm) and green (540-nm) wavelengths with the intent of making blood vessels and neoplasia stand out, does not lead to a lower miss rate, reported Dr. Tonya R. Kaltenbach and her colleagues at Stanford (Calif.) University.

They enrolled 284 patients over a 13-month period and performed two consecutive same-day colonoscopies in 240 of the patients. Patients were randomly assigned to undergo first either standard white-light colonoscopy (121 patients) or NBI colonoscopy (119 patients). Each patient then underwent a second white-light colonoscopy, to determine how many lesions had been missed on the first colonoscopy. All polyps were removed upon detection.

The investigators found a total of 259 neoplasias in 130 patients. The 12% rate of undetected adenomas with white-light colonoscopy did not differ significantly from the 13% rate with NBI colonoscopy.

Those rates stand in stark contrast to the 24% miss rate reported for colonoscopy by Dr. Douglas Rex and colleagues in 1997 (Gastroenterology 1997;112:24-8).

Coinvestigator Dr. Roy Soetikno noted at a press briefing that it is erroneous to compare results obtained using modern colonoscopes—which have a wider field of vision, improved resolution, and more flexibility—with results obtained with prior-generation scopes, and pointed out that there is now a better understanding of the morphology of colorectal lesions.

The investigators reported no potential conflicts of interest. ■

THE EFFECTIVE PHYSICIAN

Chronic Hepatitis B

BY WILLIAM E. GOLDEN, M.D., AND ROBERT H. HOPKINS, M.D.

Background

There are an estimated 1.25 million chronic hepatitis B carriers in the United States. Up to 40% of these will develop significant sequelae during their lifetimes. The American Association for the Study of Liver Disease recently published guidelines for the diagnosis and management of chronic hepatitis B.

Conclusions

Eight genotypes of hepatitis B virus (HBV) have been identified, with variable worldwide prevalence. All have been found in the United States. Recent data have suggested that genotype may be useful in predicting the progression of HBV-related liver disease and response to treatment, but more data are needed.

Chronic HBV is defined as the presence of hepatitis B surface antigen (HBsAg) for longer than 6 months, more than 2,000 IU/mL of serum HBV DNA, elevated aspartate transaminase (AST)/alanine transaminase (ALT) levels, and a liver biopsy showing chronic hepatitis with at least moderate necrosis/inflammation. Hepatitis B e antigen (HBeAg) may be present or absent in chronic HBV; its presence predicts a higher likelihood of response to available treatments.

Most HBV carriers will clear HBeAg, produce anti-HBe (antibodies to hepatitis B e antigen), and progress to the inactive carrier state. Unfortunately, up to one-fifth of these patients will spontaneously revert to positive HBeAg status.

The inactive HBV carrier state is defined as the presence of HBsAg, serum levels of HBV DNA lower than 20,000 IU/mL, the absence of HBeAg, the presence of anti-HBe, persistently normal liver transaminases, and no significant hepatitis on liver biopsy.

Older age, longer duration of HBV infection, HBV genotype C, and coinfection with hepatitis C virus (HCV) are all risk factors for progression to cirrhosis and for the development of hepatocellular carcinoma. One-third to one-half of patients who develop hepatocellular carcinoma in association with HBV do not have antecedent cirrhosis.

Implementation

Persons born in endemic areas for HBV, men who have sex with men, persons who ever used injection drugs, dialysis patients, pregnant women, those with HIV infection, and household or sexual contacts of HBV-infected individuals should be tested for HBV infection. Testing for HBsAg and anti-HBs is recommended. Seronegative persons should be vaccinated against HBV. Household and sexual contacts of persons with HBV should receive HBV vaccine.

It is important to obtain the family history of hepatocellular carcinoma and/or liver disease when evaluating patients with chronic HBV. Initial laboratory testing should assess for liver dysfunction and HBV replication, and evaluate for coinfection with HCV and HIV. Hepatitis D coinfection should also be considered in injection drug users and persons from the Mediterranean or parts of South America. Ultrasound screening for hepatocellular carcinoma is recommended every 6-12 months in high-risk patients. Hepatitis A vaccine should be administered to patients not immune to this virus.

Persons with chronic hepatitis, ALT levels two times normal, moderate to severe hepatitis on biopsy, and HBV DNA greater than 20,000 IU/mL should be considered for treatment.

Persons with ALT levels that are persistently less than two times normal should not generally be treated for chronic HBV.

Liver biopsy is most useful in patients who do not clearly meet guidelines for treatment of HBV, such as those over age 40 years with fluctuating or minimally elevated ALT levels, or those with HBeAg-negative disease and mildly elevated ALT levels or serum HBV DNA less than 20,000 IU/mL. Treatment may be indicated if moderate to severe inflammation or fibrosis is evident on biopsy.

Initial treatment regimens for chronic HBV consist of monotherapy with interferon-α, pegylated interferon-α, or nucleoside analogues.

Patients who fail to respond to standard interferon (16 weeks for HBeAg-positive or 48 weeks for HBeAg-negative disease) or pegylated interferon (48 weeks regardless of HBeAg status) may be treated with nucleoside analogues if further treatment is indicated.

Nucleoside analogues are usually administered for at least 6 months after the patient has developed anti-HBe, in HBeAg-positive patients, and until clearance of HBsAg in HBeAg-negative patients.

Resistance is a potential difficulty with nucleoside analogue treatment in HBV. Patients who fail to have a primary response to nucleoside analogue monotherapy (a decrease in HBV DNA of less than $\log_2$ after at least 6 months) should be switched to an alternative treatment regimen. Noncompliance or resistance can lead to breakthrough infection in patients treated with nucleoside analogues.

Patients with HIV and HBV coinfection who need treatment for both should be treated with highly active antiretroviral therapy regimens that contain drugs active against both viruses.

HBsAg testing is recommended in patients at high risk of chronic HBV infection prior to initiation of immunosuppressive therapy. Prophylactic antiviral treatment of chronic HBV carriers is recommended at the onset of chemotherapy or immunosuppressive therapy.

Only patients with severe acute HBV infection warrant nucleoside analogue treatment, as more than 95% of immunocompetent patients with acute HBV recover. Interferon therapy is contraindicated in this clinical setting.

Reference

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