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Showdown on Ownership Disclosure Requirement for Imaging Equipment

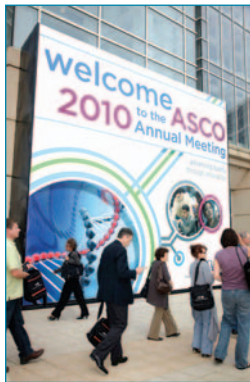
BY LOLA BUTCHER

A new self-disclosure mandate for oncologists who own high-tech imaging equipment is unlikely to affect their imaging-related revenues for now. But the law is a reminder that Congress is casting a skeptical eye toward physician-owned imaging equipment. Although there is widespread consensus that too many unnecessary high-tech images are driving up America's health care tab, there is no agreement on who's to blame. Some point to physicians who own imaging equipment, saying they overuse their scanners to make money. But some equipment-owning physicians say the overutilization comes from primary care docs not knowing which images to request and radiologists not knowing what they're looking for.

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Research Showing More Links Between Infection/Inflammation & Cancer

BY ROBERT H. CARLSON



“Not everyone gets sick from *H. pylori* infection, and there is a legitimate concern about overusing antibiotics to treat it. But the majority of the time these polyps will become cancerous if not removed, so we need to screen for the bacteria and treat it as a possible cancer-prevention strategy.”
 —Duane T. Smooth, MD

WASHINGTON, DC—New research showing links between infection-related disease and the risk of cancer was presented here at the American Association for Cancer Research Annual Meeting.

Included in the program were studies on AIDS-related and Epstein Barr virus (EBV)-related lymphomas, *H. pylori*-related colorectal polyps, and serum C-reactive protein levels associated with colon cancer risk.

“Chronic or recurrent inflammatory conditions appear to contribute to the development of a diverse array of cancers,” said William G. Nelson, V, MD, PhD, Professor and Director of Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins University, who moderated a news conference highlighting infection and inflammation research presented at this meeting. “I have hope that if we can figure [these associations] out, tackling these conditions early could be an avenue for prevention.”

Lymphoma-Specific HIV

Recent research suggests there is a lymphoma-specific form of HIV evolving within individuals who develop AIDS-related lymphoma. Now, researchers from the University of California, San Francisco have identified a subset of AIDS-related lymphomas containing HIV-infected macrophages that is immunophenotypically distinct from EBV-positive AIDS-related lymphomas.

Leanne C. Huysentruyt, PhD, Assistant Research Scientist, said the risk of developing non-Hodgkin lymphoma is 60 times greater in HIV-infected individuals com-



LEANNE C. HUYSENTRUYT, PhD: “Therefore, targeting HIV-infected macrophages for drug development may be an effective adjunct to current anti-lymphoma therapies.”

pared with non-infected individuals, despite the initiation of highly active anti-retroviral therapy (HAART). The primary difference between HIV-infected and non-infected lymphoma is that AIDS-related lymphoma is high-grade and metastatic, she said.

The subset of AIDS-related lymphomas Dr. Huysentruyt and colleagues identified are the EBV-/p24+ tumors. She said a comparison of pre- and post-HAART AIDS-related lymphomas found that only 13% of tumors in the pre-HAART era were EBV-/p24+, compared with 30% in the post-HAART era.

“This suggests that the incidence of this tumor type is increasing,” she said. “And with the increase in life expectancy with HAART, the overall number of AIDS-related lymphomas is expected to rise in the future.”

“In the case of cells already infected, such as macrophage viral reservoirs, HAART has little or no effect, suggesting that HIV-infected macrophages in AIDS-related lymphomas must be relatively long-lived and unaffected by antiretroviral therapy. Therefore, targeting HIV-infected macrophages for drug development may be an effective adjunct to current anti-lymphoma therapies.”

AIDS-related NHL

Excessive cytokine signaling may contribute to AIDS-related non-Hodgkin lymphoma (NHL), according to researchers at the National Cancer Institute.

It is known that HIV infection can lead to cytokine dysregulation and decontrol of EBV latency, both potential mechanisms for AIDS-related NHL, said Charles S. Rabkin, MD, a senior investigator there.

To explore this association, he and colleagues examined pre-NHL-diagnosis blood samples from 66 AIDS-related NHL cases in three prospective cohort studies of HIV infection, as well as samples from 186 HIV-positive lymphoma-free matched controls.

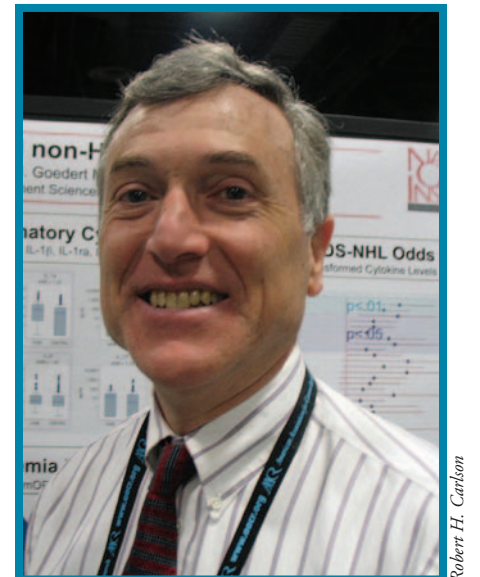
Mean CD4 counts were 172 mm³ for cases and 174 mm³ for controls.

“The cases [who subsequently developed lymphoma] had significantly higher cytokine levels for nine of 30 measured cytokines,” Dr. Rabkin said. Of the nine, the researchers identified GM-CSF, IL-12p70, and IL-15 as the most predictive markers.

“We found elevations of T-helper type-1, T-helper type-2, pro-inflammatory cytokines, and growth factors up to two years before AIDS-NHL,” he said.

EBV viremia was not significantly predictive, but the study’s statistical power was limited, he added.

“If these findings are confirmed, future research should determine if modifying cy-



CHARLES S. RABKIN, MD, said that if the results of his AIDS-related NHL study are confirmed, then future research should determine if modifying cytokine levels in some way could prevent lymphoma in this high-risk population.

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Colorectal Polyp Size

Besides an association with stomach and peptic ulcers and stomach carcinoma and lymphoma, *Helicobacter pylori* infections are also linked to colorectal polyps, mainly through hypergastrinemia and its trophic effect on colon mucosa. A report at the AACR meeting suggested that infection may also increase the chances of larger polyp size in African Americans.

Duane T. Smoot, MD, Chief of the Gastrointestinal Division at Howard University, reported a retrospective study there included data on 1,262 African Americans older than age 40 who had undergone both a colonoscopy and upper GI endoscopy on the same day, with pre-procedure indication and post-procedure diagnosis recorded. Multivariate logistic regression was used to assess the independent risk factors for polyp occurrence.

Colorectal polyps, regardless of polyp pathology, were more prevalent in *H. pylori*-infected subjects, 43% (160/368), compared with 34% (302/894) of uninfected subjects, he said, equating this to a 50% higher risk of having a polyp if the person was *H. pylori* infected.

Furthermore, there was a trend toward a larger polyp size, 1 cm or greater, in *H. pylori*-positive patients.

“Not everyone gets sick from *H. pylori* infection, and there is a legitimate concern about overusing antibiotics to treat it,”

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Dr. Smoot said. "But the majority of the time these polyps will become cancerous if not removed, so we need to screen for the bacteria and treat it as a possible cancer-prevention strategy."

Circulating C-reactive Protein

C-reactive protein, a sensitive marker of low-grade systemic inflammation, has been associated with an increased risk of colon cancer in some studies, but not all. Researchers at Vanderbilt University conducted a prospective study to evaluate the association and the potential effect of time on C-reactive protein measures.

Using data from the Shanghai Women's Health Study, Gong Yang, MD, MPH, Vanderbilt Research Associate Professor, and colleagues compared 338 cases of colorectal cancer with 451 matched controls. Follow-up was as long as 10 years.

"Chronic or recurrent inflammatory conditions appear to contribute to the development of a diverse array of cancers. I have hope that if we can figure out these associations, then tackling these conditions early could be an avenue for prevention."

**—William G. Nelson, V,
MD, PhD**

Women in the highest quartile of C-reactive protein had a 2.5-fold risk of colon cancer compared with those in the lowest quartile. But the risk was much greater for women with higher levels of C-reactive protein measured in blood samples collected close in time to disease diagnosis, he said.

"The findings support the hypothesis that low-grade systemic inflammation is associated with an increased risk of colon cancer," and cancer-induced inflammation may partly explain the observed association. ■