averaged 22 days, it is likely that most VRE-colonized patients were identified through the surveillance system. Six control-patients were excluded, because their length of hospitalization was shorter than the median duration of hospitalization before VRE BSI of case-patients. The inclusion of these short-stay patients likely would have skewed results, as they would not have had the same opportunity to develop VRE BSI; most likely, these patients would have been even less severely ill and less likely to have mucositis than the controls who were included.

Degree of neutropenia was not found to be a significant risk factor on either univariate or multivariate analysis. Since the median absolute neutrophil count in both case- and control-patients was zero on the hospital day of study (emphasizing the profundity of immunosuppression in our population), this factor likely did not optimally reflect differences in immune system function.

Our study found that increased severity of mucositis is independently associated with VRE BSI in hospitalized cancer patients, most of whom were known to be VRE-colonized. The association of mucositis with VRE BSI may indicate diffuse gastrointestinal mucosal breakdown, which promotes bloodstream invasion by gut-colonizing VRE. Gastrointestinal complications may further increase the risk of VRE BSI. Recently, the presence of *C difficile* has been identified as a risk factor for VRE BSI in patients with acute leukemia.[12]

Interventions to ameliorate the severity of mucositis or to alter VRE colonization status may help to prevent VRE BSI in VRE-colonized cancer patients. Antimicrobial therapy, such as mupirocin, has been shown to be ineffective in eradicating VRE carriage.[13] Other interventions, such as alteration of gastrointestinal flora through addition of *Lactobacillus,* deserve investigation.[14] Ultimately, prevention of VRE colonization is the most effective way to prevent VRE infection. Active surveillance and cohorting of high-risk patients, such as those with hematologic malignancy and mucositis, may be the most effective intervention to prevent VRE infection.[15]

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HCV in HIV-Infected Hemophiliacs: An Opportunistic Infection

Gina Pugliese, RN, MS
Martin S. Favero, PhD

Lesens and colleagues from the Montreal General Hospital, Montreal, Quebec, Canada, investigated the hypothesis that hepatitis C virus (HCV) infection behaves like an opportunistic infection in which progressive liver disease (PLD) is the principal manifestation.

PLD in 81 hemophiliacs coinfected with HCV and HIV was compared with 53 HIV-seronegative HCV-infected hemophiliacs. Progression to AIDS and death in 22 HCV- and HIV-coinfected hemophiliacs with PLD was also compared with 59 coinfected hemophiliacs who did not develop PLD. The risk of PLD occurrence associated with an HIV-positive status was 7.4. In the coinfected group, the risk of PLD occurrence was higher in subjects with severe AIDS-defining immunodeficiency than in those without. Persons with PLD also had a faster progression to AIDS than those without PLD.

The researchers concluded that, as with other chronic resident human viruses, HCV should be considered another opportunistic pathogen in HIV disease.

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