The increasing burden of mortality from viral hepatitis in the United States

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IN RESPONSE: We agree with Dr. Goff that further study of the discriminatory accuracy of positive and negative family history in cardiovascular disease risk algorithms is needed.

The comments from Dr. Robson and colleagues and other trials (1) provide relevant information. However, our research had a clinical rather than an epidemiologic aim and thus addressed different questions. We were concerned with clinical utility. We assessed the feasibility and impact of systematically collecting family history in comparison with its usual ad hoc collection and subsequent use in cardiovascular risk assessment in family practice (2). Our study offers the highest level of evidence for this sort of clinical intervention by use of a cluster randomized trial.

Dr. Robson and colleagues provide helpful contextual information about the QRISK2 tool used in the United Kingdom. On commencing our trial in 2007, the most widely adopted cardiovascular risk assessment tool in English family practice was the Joint British Societies 2 risk calculator as described and used in our study. It is listed in the current British National Formulary and is issued for use by all primary or secondary care physicians in the United Kingdom. Because the tool uses the Framingham algorithm, which is employed in other developed countries, we hope our study will have wider international resonance, including in the United States where it originated.

As noted midway through our study, the potential value of family history became more prominent in the United Kingdom, and we cite guidelines to which Dr. Robson and colleagues also refer (3). In addition to the Joint British Societies tool, this guidance (reissued with further information in 2010) alerts clinicians in the United Kingdom that they have an increasing number of options for risk assessment tools, including QRISK2, and notes the considerable debate about their relative merits (3). One major challenge in actual practice remains the same regardless of the assessment tool used: capturing and using authentic family history data. As Berg suggests in his editorial (4), our trial highlights the promise of a potentially low-cost and feasible intervention to realize this goal in primary care practice.

The continued interest in the role of family history in cardiovascular risk assessment is encouraging. We look forward to further research in this field.

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References

The Increasing Burden of Mortality From Viral Hepatitis in the United States

TO THE EDITOR: Although the data on mortality associated with hepatitis C virus (HCV) are certainly important to public health, Ly and colleagues (1) present them in a way that may lead to overestimation of HCV-related mortality. First, the authors define HCV death as having a death certificate mentioning HCV as either a contributing or an underlying cause. They do not present a breakdown of cause-specific mortality among these “HCV deaths.” In our own analysis of HCV mortality, liver-related deaths represented only 20% of deaths in a large cohort of HCV-seropositive former blood donors (2). Most mortality in our HCV cohort was related to trauma or suicide (23%), drug or alcohol use (14%), cardiovascular causes (13%), and cancer excluding hepatocellular carcinoma (12%). Ascertainment of HCV serostatus from blood donor records is probably more complete than from death certificates and therefore provides a more accurate estimate of attributable mortality than studies based on the latter method.

Second, in considering secular trends in HCV mortality, the authors do not mention the possible contribution of a birth cohort effect, which has been well-described for HCV infection (3, 4). Because most persons infected with HCV in the United States probably acquired their infection from injection drug–related activity in the 1960s and 1970s, they constitute a cohort that is now aged 45 to 64 years. Therefore, it is reasonable that all-cause mortality will increase as this cohort ages, independent of a possible increase in HCV-related liver mortality. Ly and colleagues could perform age-adjusted analyses to address this issue in their data.

Finally, the abstract states that “By 2007, HCV has superseded HIV as the cause of death in the United States . . . .,” which gives the misleading impression that there is an epidemic of deaths from HCV. In fact, even with the loose definition of deaths from HCV that Ly and colleagues report, the rate is increasing gradually and the rate of deaths from HIV is decreasing, presumably owing to successful treatment of HIV disease, as the authors note. Although they are correct that deaths from HCV may be underestimated because of a lack of recording HCV status on the death certificate, that many HCV-infected persons die of causes unrelated to HCV may counterbalance this possible underestimation. Physicians treating these persons should be aware of potentially preventable, non–liver-related mortality due to trauma, suicide, and substance abuse.

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References


IN RESPONSE: Our analysis of approximately 22 million multiple-cause death certificates from 1999 to 2007 showed increasing mortality among persons noted to have HCV infection; by 2007, there were more recorded deaths among HCV-infected persons than among HIV-infected persons. We have since received data on deaths in 2008 and have recorded an extension of the same trends as shown in that article (Figure).

Dr. Murphy and colleagues analyzed a small sample of 453 HCV-infected blood donors who died between 1991 and 2002. This analysis was of only a single cause of death and found a very high rate of trauma and suicide in persons who died at an average age of 50 years (1). This does not seem to be a representative sample (2), and we think that comparing that study with ours is inappropriate. In any case, such deaths among HCV-infected blood donors would be subsumed in our analysis of all deaths of U.S. residents for the years of study. As indicated in Table 1 of our article, national death certificates indicate that 57% of decedents who had hepatitis C noted as an underlying or contributing cause of death also had a diagnosis of “chronic liver disease.”

In answer to Dr. Murphy’s question about a cohort effect, our analyses clearly indicated age-adjusted mortality rates in the text, figure, and tables.

We respectfully assert that there is indeed “an epidemic of deaths from HCV” in the United States, especially among baby boomers now aged 47 to 66 years. Rate increases only appear gradual because they are flattened by using a 100,000 person population as a denominator for the rates. More important, as we discussed, death certificates actually underenumerate HCV-related (or HBV-related) deaths because various studies now show that hepatitis C is diagnosed in half or fewer of patients before death. Even when patients are diagnosed, physicians and others filling out death certificates are often not the primary clinicians and may not be aware of the decedent’s HCV infection.

We do agree that nonhepatic mortality among persons with HCV infection—including those with trauma, suicide, and substance abuse, as well as from effects of the virus on other organ systems—may not be adequately appreciated.

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References


Observation

Chiropractic Manipulation of the Neck and Cervical Artery Dissection

Background: Chiropractic manipulation of the neck can cause cervical artery dissection and stroke, although the incidence of these complications is unknown (1–4). Patients younger than 45 years with vertebral artery dissection and stroke are 5 times more likely to have visited a chiropractor in the previous 30 days than an age-matched control group (1).

Case Report: In mid-March 2012, a 37-year-old registered nurse with a history of chronic neck pain went to her chiropractor. She had seen the same chiropractor for 12 to 15 years, usually going once a month for cervical spine manipulation. Because of a new symptom (pain when turning her head up and to the right), the current visit had been the fourth in a week. From the patient’s perspective, the manipulation done during the current visit was similar to past procedures.

During the manipulation, when the patient’s head was turned rapidly, she heard a loud pop and immediately had the sensation that the room was spinning. Over the next few minutes, the vertigo intensified and she began sweating profusely. She also noted a “blind spot” in her left eye, along with other visual field disturbances. The