tion for most Quebeckers (by Internet only) and the short period allowed (consultations are now closed) excluded those who do not have a computer and limited the options for those who do. I believe that the only ethical way for the Quebec government to implement its recommendations would be a province-wide referendum specifically addressing its proposals for private health insurance for services currently covered by publicly funded health care. Such a referendum would permit citizens to decide on the sort of health care system they want and would be congruent with practices in other democracies, such as Switzerland, which holds referenda on important issues like this one.

Joseph Erban
Member
Clinical Ethics Committee
Sir Mortimer B. Davis — Jewish General Hospital
Montréal, Que.

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Hepatitis C: reviewing the options

Tom Wong and Samuel Lee[1] mention some extrahepatic manifestations of infection with hepatitis C virus (HCV), but they do not discuss the urticarias. Internists and primary care physicians need to be aware that several forms of urticaria can be associated with asymptomatic HCV infection.

The link between HCV and urticaria is controversial,[2] because various studies have failed to differentiate between acute urticaria, chronic urticaria and urticarial vasculitis, all of which have been proposed as being associated with HCV infection. The estimated prevalence of urticaria varies from 1.8% to 24%, and one case–control study disputed the association altogether.[2] The association with other hepatitis viruses is more certain. For example, electron microscopy was used to identify hepatitis B surface antigen-antibody complexes in cryoprecipitates taken from patients during the acute urticarial episode.[3]

Immune-complex deposits of viral hepatitis can activate the complement system, which results in a serum-sickness-like syndrome, with arthritis, excruciating headache and urticaria (known as Caroli's triad).[4] Urticaria resolves on treatment with interferon, and more benefit is seen when urticarial vasculitis is associated with mixed essential cryoglobulinemia.[5]

HCV testing should not be a routine screening test for all urticarias, but it is good clinical practice to consider viral marker studies in a patient with urticaria who presents with icterus or elevated transaminase levels (or both). The awareness that urticaria or urticarial vasculitis may be caused by hepatitis C is important, as early antiviral treatment can reduce significant morbidity and mortality.

Sujoy Khan
Specialist Registrar Immunology
PathLinks Immunology
Scunthorpe General Hospital
Scunthorpe, North Lincolnshire, UK
W.A.C. Sewell
PathLinks Immunology
Scunthorpe General Hospital
Scunthorpe, North Lincolnshire, UK
University of Lincoln
Brayford Pool, Lincoln, UK

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As Tom Wong and Samuel Lee mention in their article about hepatitis C,[1] patients with genotype 2 or 3 "are expected to have a high likelihood of treatment success"; however, the conclusion that these patients "may not require a liver biopsy and do not require a baseline viral load measurement" seems premature.

First, several clinical trials[2,3] have demonstrated a lower likelihood of sustained virological response in patients with genotype 2 or 3 who also have advanced fibrosis. Other researchers were unable to reproduce these findings, probably because such patients are often underrepresented in clinical trials.[4] Additional studies are now showing that steatosis is another independent predictor of sustained virological response in these patients.[4,5] Interestingly, findings in patients with genotype 3 indicate that only metabolic (but not viral) steatosis is associated with lower sustained virological response.[6]

Second, current evidence suggests that among patients with genotype 3, viral load is an important predictor of both sustained virological response[3,4,7,8] and early virological response.[3] Moreover, for patients with genotype 2 or 3 and early virological response at week 4, shorter courses of therapy (12–16 weeks) were as effective as the recommended course of 24 weeks.[3,7,8] Whether patients with genotype 2 or 3 who have a high viral load and/or absence of early virological response (with or without advanced liver fibrosis) will benefit from longer treatment should be investigated in further clinical trials.

It therefore appears that both baseline histologic findings and viral load may be useful for tailoring treatment in certain subgroups of patients with genotype 2 or 3 in whom the standard duration of therapy might constitute overtreatment.

Michal R. Pijak
Consultant in Internal Medicine, Rheumatology, Allergy and Clinical Immunology
Department of Internal Medicine
University Hospital
Bratislava, Slovakia

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[The authors respond:]

We agree with Drs. Khan and Sewell.

Dr. Khan raises several important issues about the treatment of patients with HCV genotypes 2 and 3, but we stand by our general conclusions. First, the largest randomized study of 24 versus 48 weeks of peginterferon and ribavirin treatment showed similar sustained virological response (SVR) rates for genotype 2/3 patients regardless of baseline viral load and histological stage.[1] Although not all studies find the same result, in practical terms whether or not one knows the degree of fibrosis or viral load generally does not affect the decision whether to treat or not. In other words, because of the high expected SVR, even in the cirrhotic genotype 2/3 patient with high viral load, we would still proceed to treatment.

Dr. Pijak's point about potentially shorter courses of treatment (12–16 wk) in genotype 2/3 patients with a rapid viral response (RVR), defined as undetectable HCV-RNA at week 4 is well taken. However, despite promising results from studies with relatively small sample sizes,[2-4] we believe it is still premature to adopt this strategy, even in patients with RVR. Our contention is based on the results of the large multicentre ACCELERATE study recently presented at the European Association for Study of Liver annual meeting.[5] This study randomized 1469 genotype 2/3 patients to 16 or 24 weeks of treatment. The 16-week treatment group showed a significantly lower SVR rate compared to the 24-wk group (intention-to-treat analysis, 62% v. 70%; $p = 0.004$).

We agree that there are probably subgroups of highly-responsive patients with both genotypes 2/3 and 1 who may benefit from shorter courses of treatment, but feel that such groups have yet to be definitively identified.

Samuel S. Lee
University of Calgary
Calgary, Alta.
Tom Wong
University of Ottawa
Public Health Agency of Canada
Ottawa, Ont.

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Corrections

In the unabridged version of a research article on planned cesarean versus planned vaginal births,[1] there was an error in Table 3. The data should be presented as mean (SD) [median], not midpoint as indicated.

REFERENCE

1. Palencia R, Gafni A, Hannah ME, et al.; for the Term Breech Trial Collaborative Group. The costs of planned cesarean versus planned vaginal birth in the Term Breech Trial. *CMAJ* 2006;174(8). DOI:10.1503/cmaj.050796.

DOI:10.1503/cmaj.060700

The DOI published with a recent research article[1] was mistakenly listed as 10.1503/cmaj.060044. It should have been 10.1503/cmaj.060664.

In this same article, the following sentence should have been the first one in the contributors statement for "The first two authors (Suh JW & Koo BK) equally contributed to this work ."

REFERENCE

1. Suh JW, Koo BK, Zhang SY, et al. Increased risk of atherothrombotic events associated with cytochrome P450 3A5 polymorphism in patients taking clopidogrel. *CMAJ* 2006;174(12):1715-22.

DOI:10.1503/cmaj.060701

The DOI published with a letter to the editor[1] was mistakenly listed as 10.1503/cmaj.060066. It should have been 10.1503/cmaj.1050244.

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1. Hoey J. Unnecessary exposure? [letter]. *CMAJ* 2006;174(4):499-500.

DOI:10.1503/cmaj.060702