

Estimates of risk of window-period transmission of blood-borne viral diseases in Quebec

We estimated the risk of transfusion-related transmission of viral disease attributable to the window period of laboratory testing (the period between infection and detection of the virus by the test) for the blood supply in the province of Quebec.

We studied donors who gave blood between Apr. 1, 1997, and July 31, 2002. Because incident cases can be ascertained only in repeat donors, we included only donors who made 2 or more donations since 1992, when the second-generation enzyme immunoassay (EIA) for hepatitis C virus (HCV) was introduced, to avoid inclusion of any false-negative results obtained with the earlier version of the assay.

We evaluated the window-period residual risk using the methodology recently reviewed by Glynn and associates.¹ For each donor, the person-year contribution was calculated on the basis of the time between the beginning of the study period (or the date of the first negative screening result for those who did not have a negative result before 1997) until the most recent donation.

For donors with a confirmed infection, the person-year contribution was adjusted by assuming that infection occurred halfway between the last 2 donations. Incident cases were defined as donors with a serologically confirmed infection during the 5-year study period who had had a negative screening result on the previous donation.

Héma-Québec annual statistics for fiscal year 2001/02 showed that 10.7% of allogeneic donations came from first-time donors. The incidence of HIV and HCV infection among first-time donors is 2.1 and 2.4 times higher respectively than among repeat donors.² We therefore adjusted the overall incidence rates according to the algorithm proposed by Glynn and associates.¹ For hepatitis B virus (HBV) and human T-cell lymphotropic virus (HTLV), we also assumed conservatively that the incidence rate was twice as high in first-time donors. For HBV, we applied another correction factor of 2.38 to take into account the transient appearance of hepatitis B surface antigen (HbsAg).²

Window-period estimates were taken from the recent literature.² For HBV, we subtracted 13 days from the value suggested by Dodd and colleagues² because of the greater sensitivity of the test method used by Héma-Québec (PRISM HBsAg, Abbott

Laboratories, Diagnostics Division, Abbott Park, Ill.) relative to that of EIA.³

The calculated residual risk per million donations attributable to window-period transmission was 0.82 for HCV, 0.20 for HIV, 3.59 for HBV and 0.18 for HTLV. Table 1 (available at www.cmaj.ca/cgi/content/full/170/7/1077/DC1) shows the incidence rate estimates for each virus and the residual risks with their confidence intervals.

These residual risk estimates are comparable to similar estimates for the Western world,⁴⁻⁶ including those published recently for the rest of Canada.⁷ It is remarkable that the current HIV window-period risk estimate for Quebec as a whole (Table 1) is 5 to 10 times lower than an estimate for the Montréal transfusion centre for the period 1989-1993.⁸ This difference results mainly from the substantial decrease in HIV incidence within our donor population, from 3.3 per 100 000 person-years⁸ to 0.67 per 100 000 person-years. The incidence of HBV among Quebec blood donors (Table 1) is similar to the recently reported US rate of 3.02 per 100 000 person-years.² The resulting residual risk in Quebec is somewhat smaller because of the shorter window period assumed with our current testing technology. Our HBV incidence rate appears lower than that for the rest of Canada, re-

ported at 5.3 per 100 000 person-years.⁷ However, this difference is probably not significant, given that the method applied to correct for transient antigenemia among incident HBV cases is susceptible to some imprecision.

These data confirm the declining risk associated with window-period donations, which represent the major residual source of transfusion-related transmission of HIV, HCV and HTLV. For HBV, in addition to the window period, there is also the risk posed by chronically infected donors whose antigen level is too low to be detected by the HbsAg screening test. This risk, estimated at approximately 1 in 50 000 donations,⁹ can be mitigated by testing donors for hepatitis B core antibody.

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Management of dysphagia

It is gratifying to see attention paid to the nutritional status of stroke patients with dysphagia, an often overlooked aspect of care.¹ However, it is unfortunate that Hillel Finestone and Linda Greene-Finestone promulgate some of the misperceptions that abound in the area of managing patients with a swallowing disorder.

One of the most distressing errors, which often leads to inappropriate management, appears in the article title.¹ Dysphagia cannot be “diagnosed.” Rather, it is a symptom of several hundred conditions and cannot be managed properly without identification of the source. Dysphagia has come to be discussed as though it were a disease in and of itself, which leads to the misperception that there is a standard approach to its management. This has in turn led to various inappropriate strategies for care,² including some that contribute significantly to dehydration,³ as the authors have noted elsewhere.⁴ Where Finestone and Greene-Finestone refer to “overnight intravenous fluid administration,” it is to be hoped that they mean hypodermoclysis, the long-term hydration method of choice.⁵

The case presented¹ illustrates the most problematic of all issues associated with oropharyngeal dysphagia: aspiration. The patient in this case is described as having “pneumonia” in both lungs on the day of admission (also the day of insult). However, this is clearly a case of aspiration pneumonitis, caused by inhalation during the reported vomiting, not bacterial pneumonia requiring antibiotics.⁶⁻⁹ Antibiotic therapy, as mentioned in the case description, might well be prophylactic against the secondary bacterial infection that often occurs but would not be effective for chemical pneumonitis. Secondary pneumonia is most often caused by as-

piration of saliva, an event that also occurs in healthy adults and that is best avoided by scrupulous mouth care.¹⁰

In the final section, “The case revisited,” the authors state that “Mr. B’s pneumonia is a strong indicator that aspiration occurred. His pneumonia is a probable sequela of aspirating saliva. Mr. B is not allowed to have anything by mouth when he is admitted to hospital.”¹¹ Finestone and Greene-Finestone have missed the obvious at several levels. The patient’s “pneumonia” on admission was certainly the result of aspiration but could not have been due to aspiration of saliva (bacterial pneumonia). The solution is not to give him nothing by mouth but instead to identify the real cause of the problem and ensure scrupulous mouth care while maintaining good nutrition and hydration.

Of the remaining misperceptions, one in particular requires mention: there is no relation between the presence or absence of a gag reflex and the ability to swallow.¹¹

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