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Letter to the Editor

Are reusable blood collection tube holders the culprit for nosocomial hepatitis C virus transmission?

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To the Editor—Cheng et al[1] published an interesting report of a case of nosocomial transmission of hepatitis C virus with a reusable blood-collection tube holder postulated as the vehicle for transmission. Healthcare-associated hepatitis C virus transmission has been most often related to breakdown of infection control practices such as poor hand hygiene, use of contaminated gloves or equipment, and practices such as syringe reuse contaminating multiple-dose vials of infusions.[2–4] In their report, investigations were performed to identify the source of transmission. However, their investigations were associated with flaws and were performed disproportionately.

First of all, the authors arguably excluded the possibility of lapses in infection control measures simply by direct observation. It is inconceivable to rule out such an important aspect solely by direct observation after the incident has just occurred, which is inevitably confounded by the Hawthorne effect.

Second, the authors stated that by reviewing the time log in the barcoding system of the computerized laboratory information system, they identified 14 instances of phlebotomy from the source patient followed by the index patient. However, they did not mention that many phlebotomists were involved, all using their own blood collection sets. Also, there was clearly no instance of one phlebotomist taking blood from the source patient that immediately followed the index patient. Furthermore, the 2 patients in this episode were different genders. The practice of the affected unit is to have 2 phlebotomists working together, one serving male patients and the other serving female patients. The chance of transmission from the male (source) patient to the female (index) patient via contaminated tube holder is remote.

Third, for the environmental surveillance specimens collected for the presence of HCV, 28 of 34 environmental samples were collected from tube holders. Only 1 was obtained from the glucometer tray; 3 were obtained from the tray for phlebotomy; and 2 were obtained from the phlebotomy trolley. Thus, the focus on this environmental surveillance is too narrow and not global enough to identify other possibilities. The authors' claim of reusable tube holders being the only shared items cannot be substantiated before a complete workup has been done. Notably, investigations concerning peripheral and central intravenous catheter insertion as well as intravenous injections of medication including the use of multiple-dose vials were missing. The authors reported that HCV was found in the inner side of a single tube holder. But the date of surveillance sampling was not stated and was likely to have been performed months after the HCV transmission has occurred, and we have no affirmation regarding whether this tube holder, of the 14 tube holders sampled, had been used by both the source and index patient. Surely, the likelihood is that the tube holder was in fact entirely used for and contaminated by the index patient.

All of these items provide precise circumstantial evidence to refute the postulation. As such, molecular genetic study is superfluous and if done, whole-genome sequencing of HCV isolates would have been the preferred method.

We are also unconvinced of the validity of the in vitro experiments. In the simulated phlebotomy experiment, the 5-mm tip of the rubber sleeve capping the sleeved-needle was dipped into HCV-containing plasma to deliberate contaminate the inner wall of tube holder. This is exaggerated and unlikely to happen in real life. Using HCV-negative EDTA blood at atmospheric pressure to mimic the venous side of a patient is also incorrect because venous pressure at cubital fossa remains positive during venesection. Moreover, the risk of return flow had been vigorously investigated. Even in the most extreme scenario, where the temperature inside blood collection tube (blood plus air) reaches 37°C, combined with an eccentric penetration of cap, the maximal return flow volume is still less than the dead volume of smallest needle; therefore, no back flow occurs.[5]

For the radionucleotide study, the tip of the sleeve needle, instead of the rubber sleeve, was smeared with gauze containing a few drops of ^{99m}TcO4. ^{99m}TcO4 is a very small molecule, with a molecular weight of only 30×10^{-5} attograms. If we assume that HCV has a density similar to water, each HCV weighs ~21.6 attograms (given a 60-nm size). Comparing a molecule with a particle is a very inappropriate analogy. Furthermore, the negative pressure created by releasing the manual pressure from the saline bag to simulate the suction of virus into the bloodstream was unquantified. In the next experiment, the authors

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measured the blood pressure drop upon release of tourniquet but omitted to mention whether the pressure remained positive. Simulation studies must be precise and if the in vitro experiments are so indirect, it cannot be trusted to reflect the real-life situation.

We appreciate the authors' effort in investigating the exact route of transmission for their nosocomial HCV infection. But in vitro simulation cannot be taken as solid evidence. Although it is tempting to implicate the finding of HCV containing blood on the inner surface of a single tube holder as the source for cross transmission, in our opinion, the transmission was most likely due to contamination by the index patient's blood. We believe that hasty and premature acceptance of the blood-collection tube as the source of HCV cross infection, not solidly supported by a validation study, is misleading and may do more harm than good if attention is not given to actual practices that can more certainly enhance infection control.

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