To The Editor: Thank you for providing me with the opportunity to comment on the above viewpoint. I perfectly understand that in this difficult situation it is hard to face the facts particularly when scientists from the outside provide a view which is not shared by at least part of the Libyan medical community. So let me summarise how I was involved in the analysis of the causes of the outbreak. In June 1998, 111 coded plasma samples were sent from El-Fateh Children Hospital to a private international laboratory, Covance, located in Geneva, Switzerland for HIV-1 serology; all but one plasma had anti HIV-1 antibodies. I was at the time head of the HIV reference laboratory in Geneva and thus received in a routine procedure samples for confirmation of the diagnosis including western blot. When I found out that most of them were indeed HIV-1 infected, I contacted the Libyan WHO representative; Dr Amer Rahil and suggested further investigations. It was decided that a group of infected children and their parents would come to the Geneva University Hospital for investigations. We proposed to set up an epidemiological study. Dr. Claire Anne Siegrist, infectious disease specialist at the Children Hospital in Geneva and I also proposed to help in the management of treatment issues and, for this reason, asked for a medical contact in El-Fateh Children Hospital. These latter propositions were not retained but in April 1999, a group of 37 children came with 46 parents. They had a clinical check-up and anamnesis was done with the help of an interpreter fluent in Arabic.

A summary of clinical finding and biological data are published [1]. What we found is that all children were infected by a HIV-1 monophyletic recombinant circulating form from West Africa and 40% of them were infected with HCV. We excluded vertical transmission for both HIV and HCV on the basis of serology in parents. Thus the assumption on page 2 of the viewpoint mentioning that HCV was acquired through vertical transmission is incorrect since neither the WHO nor us have more or less controlled the propagation of HIV-1 infection in intravenous drug users but still frequently observe HCV infections in this context was that around 30 % of HBV vaccinated children had both markers induced by vaccination (anti HBs Ag) and also markers (anti Hbc Ag) induced either by natural infection or by a vaccine preparation containing full inactivated virions. This suggests that either the children have been immunised with an “old” vaccine preparation or that they have possibly been contaminated with HBV at the time of vaccination like it has been described in other circumstances when the same needle is used several times.

Concerning the origin of HIV-1 infection, it is invariably difficult to find the source, for example we have observed, 7 years ago, a monophyletic infection with CRF-11 in around 30 drug addicts in the western part of Switzerland (2 and unpublished). This recombinant form is known to originate from Africa but, as in the Libyan outbreak, we have been unable to identify the initial infection. We have a number of hypotheses for the initial event including travelling of a drug addict in Africa and sharing of needles, contamination of a drug addict through sexual contact with an African etc but once there is an individual with high viremia sharing needles with partners, the mini-epidemic will soon develop. So it is untrue that monophyletic infection cannot occur (last paragraph of the viewpoint) since we observe it in drug addicts in Switzerland. Others have reported similar observations in Spain [2,3]. Epidemiologists also know that it is very difficult to identify the source of an infection when the initial event occurred several years before.

Overall, all our observations point towards a nosocomial infection due to suboptimal medical practices. We were prevented from having a definite evidence since neither the WHO nor us were allowed to explore further the situation in Libya with the help of our colleagues from El-Fateh Children Hospital. A WHO report has clearly mentioned that HBV, HCV, HIV infections due to nosocomial transmission is incorrect since most of the mothers were not infected [1]. The fact that we found clusters of four HCV genotypes reflect simply the fact that HCV is much more common in Libya than HIV-1, thus in the case of improper sterilisation procedures or reuse of contaminated needles it is expected to have different HCV genotypes.

On top of that we had the vaccine booklets from the children, most of them were vaccinated for hepatitis B. What was unexpected in this context is that 40% of them were vaccinated for hepatitis B. What was unexpected in this context was that around 30 % of HBV vaccinated children had both markers induced by vaccination (anti HBs Ag) and also markers (anti Hbc Ag) induced either by natural infection or by a vaccine preparation containing full inactivated virions. This suggests that either the children have been immunised with an “old” vaccine preparation or that they have possibly been contaminated with HBV at the time of vaccination like it has been described in other circumstances when the same needle is used several times.

In terms of public health it would be in the best interest of the Libyan children to conduct a large study in Children’s hospitals for a systematic detection of HCV infection whose consequences will appear in several years. In Western countries we have more or less controlled the propagation of HIV-1 infection in intravenous drug users but still frequently observe HCV infections in this context.
population suggesting that transmissibility of HCV is higher than that of HIV-1.

Finally we have transmitted written reports of our investigations in 1999 and 2000 to Dr. Amer Rahil for the Health authorities of Libya. In these reports we also asked to have a medical contact in Libya. We did not receive any answer and I was prevented from testifying and presenting scientific evidence.

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