To the editor:
In their recent article, Macé et al. pointed appropriately to the many disturbing clinical similarities between haemorrhagic fever with renal syndrome (HFRS) due to Seoul virus (SEOV) infection – an emerging zoonosis – and two pregnancy-related pathologies affecting mainly the liver, acute fatty liver of pregnancy (AFLP) syndrome and haemolysis, elevated liver enzymes and low platelet count (HELLP) syndrome [1]. At present, HFRS is one of the most frequent, but still heavily underestimated, forms of acute infectious kidney injury, with up to 200,000 cases per year worldwide. Moreover, SEOV affects not only the kidneys, but also often the liver as well [2].

Whereas urgent delivery is the gold standard for preserving both mother and fetus when a pregnant women has AFLP or HELLP, such intervention is rarely if ever needed when the women have in fact HFRS, as spontaneous remittance within two to three weeks is the rule [2,3]. Thus, therapeutic decision-making should be early and quick, preferably without awaiting time-consuming hantavirus serology, but should also be guided by subtle clinical differences at presentation. Anomalies in blood levels (not specified here) of alkaline phosphatase, bilirubin, glucose, uric acid and fibrin degradation products are more suggestive of AFLP or HELLP, rather than of HFRS [4,5]. Moreover, the classic symptom triad of pre-eclampsia (hypertension, proteinuria and oedema) often seen in women with AFLP or HELLP [4,5] was apparently absent in the reported case before and after admission. Conversely, C-reactive protein levels greater than 100 mg/L (norm: 1-5 mg/L), hyponatraemia, hypokalaemia or the ‘lipid paradox’ (very low acute cholesterolaemia, contrasting with hypertriglyceridaemia) could have pointed to HFRS [2,3]. Sudden, massive and nonselective proteinuria, even before hospital admission, is distinctive for HFRS, and simple urine examination should not be delayed until day 7, as in this case. Moreover, nephrotic-range proteinuria of 3.35 g/24 h, diminishing within three days to less than a fifth of its value, is highly atypical for AFLP (or HELLP), but is commonly seen in HFRS, where all ‘lesions’ heal rapidly without sequelae. A sudden renal deterioration a few days after worsening thrombocytopenia is very typical for HFRS, but should not suggest the need for any form of surgery or biopsy, even less so at a time (day 7) when activated partial thromboplastin time and levels of platelets, lactate dehydrogenase, C-reactive protein and most liver enzymes were already clearly normalising.

The authors of the article call, justifiably, for large studies focusing on SEOV epidemiology. Such studies would add to the findings of an earlier (1994) large seroepidemiological hantavirus study carried out in Northern Ireland [6]. Similar clinical cases of SEOV-induced HFRS, with both kidney and liver involvement, were also seroconfirmed in Portugal (1993), Northern Ireland (1994) and Bosnia and Herzegovina (1994) [as described in 7].

The presence of SEOV sero- and/or antigen-positive rats was documented in 1994, using an immunofluorescence assay (IFA) and/or enzyme-linked immunoabsorbent assay (ELISA), in 34 countries in the New and Old World (including France) [8]. This study yielded several SEOV isolates and stressed the importance of SEOV – the only worldwide pathogenic hantavirus, which is transmitted by the omnipresent wild rat.

Conflict of interest
None declared.
Authors’ contributions

J. Clement conceived the article, and wrote the text. V. Vergote and L. Laenen performed or controlled the laboratory work. M. Van Ranst coordinated and edited the text.

References


