Diagnosis and treatment of human sparganosis

I read with interest the Clinical Picture by Hong Li and colleagues[1] describing a human case of ocular sparganosis. Sparganosis is a human parasitation caused by the plerocercoid larvae of tapeworms belonging to the *Spirometra* genus. Humans acquire the disease either orally or by active penetration of the larvae. The oral route involves the ingestion mainly of raw or undercooked snakes and frogs infected with the plerocercoid larvae, as well as the accidental ingestion of microscopic water fleas infected by the procercoid larvae while drinking. For particular curative practices that include the use of snakes and frogs as poultices, any plerocercoid larvae infecting the animals can actively penetrate the individual through wounds or the eye.

The authors identify the species causing the case of ocular sparganosis (*Spirometra mansoni*) only by histopathological examination.[1] However, morphology does not allow specific diagnosis because plerocercoid larvae do not have specific morphological features. Histopathology can confirm only that the worm extracted from the eye is a plerocercoid-type larva. The most rapid methods for the specific diagnosis are molecular techniques—for instance, PCR restriction fragment length polymorphism.[2]

Surgical removal is the required treatment when the parasite is accessible, as in this case. According to the authors, after the worm extraction, albendazole was given to prevent dissemination of the parasites.[1] In my opinion, the pharmacological treatment would be needed only in the case that the larval scolex had remained in the patient's eye, because the worm grows (via a process known as strobilation) at the neck zone located behind the scolex.[3] Figure 2 of their supplementary material shows the removed worm with an attenuation at one end. The scolex is located in this thinner part. Assessing whether the worm had or did not have a scolex would have been easy under a microscope. If the extracted worm had a scolex, albendazole would not have been needed. Only *Sparganum proliferum* has the ability to disseminate by branching and budding.[3] Apparently, this form of dissemination was not the case as the extracted plerocercoid did not show signs of either of these two processes. Furthermore, a point to consider is that in potential endemic areas of *Taenia solium* cysticercosis such as China—the patient's country in this case—asymptomatic human neurocysticercosis can become symptomatic because of the cysticidal effect of drugs such as praziquantel and albendazole,[4] which therefore should be used under constant medical supervision and only if strictly necessary.[5]

I declare no competing interests.

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Sensitivity and negative predictive value for a rapid dengue test

In 2016, WHO recommended Dengvaxia, the first-ever licensed dengue vaccine, for use in children aged 9 years or older residing in high-burden settings. The vaccine had shown efficacy against all four dengue serotypes in two large phase 3 trials.[1,2] A safety signal of an excess risk of severe dengue was detected in vaccinated children aged 2–5 years.[3] This finding prompted further analyses with a new immunological assay to assess whether the risk was associated with age per se or was due to a higher proportion of children in this age group having had no previous dengue infections. These analyses[4] showed that although the vaccine offered substantial protection among children who had been previously infected with dengue, dengue-naive vaccinees were at increased risk of dengue hospitalisation and severe dengue during the 5-year trial follow-up compared with similar children in the placebo group. This effect had been previously postulated in mathematical models.[5]

In response to this new evidence, WHO revised their previous advice and now recommends a pre-vaccination screening strategy to avoid the vaccination of dengue-naive children.[6] In the absence of a licensed rapid diagnostic test to detect previous dengue infection, discussions around a target product profile for such a test have focused on the need for high test specificity to limit the number of false-positive test results, and thus minimise the risk of vaccinating dengue-naive children. However, increasing the specificity of a test typically decreases its sensitivity, and a test with poor sensitivity would leave many unvaccinated who would have potentially benefited from vaccination.

Isabel Rodriguez-Barraquer and colleagues[7] argue for the use of the positive predictive value (PPV) of a