Commentary

The case herein presented[1] is interesting at different levels. First, it illustrates well the severity of extraparenchymal neurocysticercosis (NCC), mainly due to the frequent hydrocephalus and to the requirement

of surgical treatment. Although parasite location in this compartment is the less common one, it involves a very severe disease, accounting for most fatalities in NCC cases. In my opinion, these disease forms demand growing attention and effort from clinical and basic research teams to improve their management.

Second, the presentation of new ventricular parasites after endoscopic removal is an intriguing fact that we have also seen in our patients.[2] We have seen this condition also in patients with parasites located in the subarachnoid cisterns at the base of the brain (SaB).[3] As the authors stated, the reasons behind this phenomenon are currently not known, but incomplete removal of parasites during endoscopy, partial destruction of parasites by cestocidal drugs, or re-infections are the most likely causes.

Against the re-infection possibility goes the fact that parasite recurrence after treatment (surgical or drug-based) always involves, in our experience, parasites lodged in the SaB or the ventricular system at diagnosis time. If re-infections occurred, why would parasites go to the same area as in the first infection? It could be hypothesized that some parasites show a particular tropism, genetically determined, to these locations. This possibility cannot be completely discarded since it is most likely that a re-infected individual would be infected by eggs from the same adult *Taenia solium* worm. But although genetic differences have been found between *T. solium* parasites, for the time being we do not have a conclusive argument supporting that these differences are involved in the pathogenesis heterogeneity.[4] Another argument against the re-infection hypothesis is the time elapsed between the first treatment and the parasite recurrence. In general, this period of time is relatively short, 2 years at most, as in the case presented here. This time is probably too short to allow the parasite development in these locations for causing symptoms. On this issue, it is interesting to note that extraparenchymal forms of NCC are very rarely diagnosed in children, compared to adults.[5] One probable explanation is that parasites in these locations require time to grow and cause symptoms since the symptomatology in these forms is mainly due to a mechanical effect.

If these cases were not secondary to re-infections, it is most likely that medical or surgical treatment is incomplete in these locations, and that radiological studies are unable to visualize small parasites. As it is known and has been reported before, cestocidal drugs are less effective on extraparenchymal parasites than on parenchymal parasites, and it is frequently the case that several cestocidal treatments are necessary to "clear out" patients from parasites.[6] The reasons underlying this observation are not known. Previous studies failed to show a clear correlation between albendazole concentration in sera and CSF and the treatment efficiency, and thus, the treatment result did not seem to depend on the drug only.[7] It is possible that efficiency depends also on the parasite developmental stage, and perhaps not all parasites are in a similar stage at a given moment. Regarding parenchymal parasites, it is frequent to diagnose cysticerci in different developmental stages (vesicular and colloidal, vesicular and calcified, colloidal and calcified or the three phases together) in the same patient, and it is probable that this occurs in the SaB or ventricular locations as well. When patients are diagnosed with vesicular cyst, other parasites may be present in earlier developmental stages, not visible yet in MRI and non-responsive to cestocidal drugs. On the other hand, it is also known that radiological studies (CT scan and MRI) are less effective to visualize parasites in these locations where cysts are surrounded by cerebrospinal fluid. The reasons are that parasites cysts emit a signal similar in intensity to that of the CSF, they generally do not enhance after contrast intravenous administration, and parasites commonly lack a scolex.[8] Considering these two factors (lower efficiency of cysticidal drugs and higher difficulty to visualize parasites), it seems probable that the rationale behind these cases be an under-diagnosis of small parasites in these locations, added to a failure of cysticidal drugs to destroy all cysts.

In conclusion, this paper illustrates well the necessity of: (1) improving diagnosis tools for parasites in these locations. New MRI sequences have been described, but they are not standardized yet;[8,9] (2) advancing in the development of new, more efficient drugs on all parasite stages.

Agnès Fleury

Peripheral Unit of the Institute of Biomedical Research in the National Neurology Institute, National Autonomous University of Mexico, Mexico DF, Mexico.

Address for correspondence:
Dr. Agnès Fleury,
Peripheral Unit of the Institute of Biomedical Research in the National Neurology Institute,
National Autonomous University of Mexico,
Mexico DF.
E-mail: afleury@biomedicas.unam.mx

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