Reviewer’s report

Title: Vestibular evoked myogenic potentials in 5 patients with superficial siderosis

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Reviewer: dimitri renard

Reviewer’s report:

This report describes extensive vestibular testing in patients with superficial siderosis (SS).

However, I have some major concerns despite the interesting question of clinical and extensively paraclinical examined vestibular involvement described by the authors.

Major concerns:

- English is poor overall.

- The authors did include “any type of” superficial siderosis. However, symptomatic and idiopathic SS have to be separated. The SS subtype most typically leading to VIIIth nerve involvement is idiopathic SS. So I don’t understand why the authors included also other causes of SS. In case nr 4, the authors don’t even describe the hemorrhagic orbitofrontal lobar lesion. What is this lesion? In case nr 5, the authors have noted cerebral microbleeds attributed to hypertension. However, amyloid angiopathy (AA) is both a cause of microbleeds and SS. AA is never evoked in this report, neither in the patient description nor in the discussion. In case nr 2, SS is quit sure not idiopathic, since probably related to cavernous hemangioma.

- One of the major signs in SS is dementia and cerebellar ataxia. This is mentioned nowhere in the article. This is of extreme importance since ataxia probably contributes a lot to instability and nystagmug in the SS patients studied.

- I was not able to localize Fig 6.

- Ataxia and lower cranial nerve involvement is generally explained by a gravity-related process. Thus, one should expect an early and more severe inferior as opposed to superior vestibular dysfunction. Indeed, other mechanisms could be evoked (e.g. longer nerve). But in that case, one should expect also a VIth nerve dysfunction (the longest and the smallest in diameter cranial nerve), which is generally not seen in SS.

- Surprisingly, only 1 patient had radiological VIIIth nerve SS, although a rather typical sign in idiopathic SS.
Why a hemostatic agent of chelating agent should be given in case of progressing deficit? This has never been proven. And what kind of hemostatic agent do the authors have in mind?

In my opinion, the most interesting type of approach would be extensive vestibular testing (bearing in mind also cerebellar testing) in patients with idiopathic typical SS.

Minor concerns:

-“Bloody “ CSF. Please be scientific and specify the number of red blood cells/mm³.
-“Cause of hemorrhage: subarachnoid hemorrhage”. I thought that SS and subarachnoid hemorrhage were more or less synonyms.
-“somewhat” cloudy membrane. The membrane is cloudy or it is not.
-“somewhat” saccadic pursuit: idem

Level of interest: An article whose findings are important to those with closely related research interests

Quality of written English: Not suitable for publication unless extensively edited

Statistical review: No, the manuscript does not need to be seen by a statistician.

Declaration of competing interests:

I declare that I have no competing interests’ below