Author's response to reviews

Title: Pseudo - subarachnoid haemorrhage due to chronic hypoxaemia: Case report and review of the literature

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Author's response to reviews:

Dear Editor and Referees,

thank you very much for considering our manuscript „Pseudo – subarachnoid haemorrhage due to chronic hypoxaemia: Case report and review of the literature“ for publication in “BMC Neurology”. We are grateful for your extensive review and commentaries to our work. We revised our manuscript and hope to have met the reviewer’s requests.

Please find our response to your comments in the following:

To Ms. Asma Zakaria (Reviewer 1):

A lumbar puncture and digital subtraction angiography to definitely exclude subarachnoid haemorrhage and intacranial aneurysms was discussed. We agree that a lumbar puncture is the “gold standard” - method to discriminate SAH and pseudo – SAH. However, given the impaired coagulation of our patient (INR 8.2, PTT 114 s), invasive methods would have carried significant risks of procedure – related bleeding complications. Without extensive further imaging, the NECT findings could not have been definitely discriminated from SAH. However, the findings of CT - angiography and dedicated MRI along with the clinical presentation gave us enough confidence to diagnose pseudo – SAH and refrain from invasive procedures. An imaging result which further supported our diagnosis and which was not mentioned in the original manuscript was that the hyperdensities found on NECT correlated with the course of the vessels on CTA. This finding is now also mentioned in the manuscript (page 4, lines 7 – 8).

Moreover, we rewrote the paragraph regarding invasive diagnostics in the discussion section (page 7, lines 7 – 16), now stating clearly that lumbar puncture should be considered in cases of suspected pseudo – SAH.

To Mr. Vasileios – Arsenios Lioutas (Reviewer 2):

Comments (1) and (2)
We believe that the highly raised level of haematocrit (75%) played an important role in the development of the SAH-like picture. Although literature is relatively sparse, the positive correlation between haematocrit and vessel density has been shown and reports of diffuse sulcal hyperattenuations due to polycythaemia exist. We agree that polycythaemia alone probably would not have caused images as similar to diffuse SAH as in our case. Venous engorgement, as pointed out by the reviewer, was another major factor. In our rewritten discussion, we have put more emphasis on the role of vessel dilatation (page 6, lines 21–25).

Concerning your comment about superficial siderosis, we reviewed the MRI scan, which included two T2*-weighted sequences, and did not find signs of siderosis. This negative finding is now also included in the results section (page 4, lines 22–23).

Comment (3)
As requested by the reviewer, the phrase “MRI can securely differentiate” was deleted in the revised manuscript. We agree that it might have caused false certainty in cases of suspected SAH / pseudo – SAH. In the rewritten paragraph in the discussion section we now state that CTA, MRI and clinical picture gave us enough confidence to diagnose pseudo – SAH in our particular case. In view of the comment by Reviewer 1, we also discussed the role of lumbar puncture in more detail (page 7, line 7–16).

To Ms. Nina Tsakadze (Reviewer 3):

Major essential revisions:

Comment (1)
The article has now been read and corrected by a native speaker of the English language.

Comment (2)
Thank you for your thorough review and commentary. We rewrote the discussion section, now reporting in more detail the results of previous authors regarding pseudo – SAH in patients with hypoxic/anoxic encephalopathy. The studies by Avrahami et al. and Ahn et al. as well as a report about pseudo – SAH after bee sting intoxication (Yasar Tekelioglu et al.) were added to the references. The statement that this is the first report of pseudo – SAH due to chronic hypoxaemia is now omitted and replaced by the phrase “unusual case, in which [pseudo – SAH] was caused by chronic hypoxaemia (page 2, lines 5–6). We believe, however, that the underlying pathology in our case is different from those reported before, as the imaging findings were caused by polycythaemia and prominent veins rather than cerebral edema. This is now discussed in more detail.

Comment (3)
In the original manuscript we had stated that the headache was either chronic
tension – type or attributed to hypoxia/hypercapnia. The reviewer is correct that under the given circumstances a secondary headache caused by hypoxia is more likely, and we changed the abstract and main text accordingly (page 2, lines 16 – 17, page 7, lines 24 – page 8, line 3).

Minor revisions:

Comment (1)

We performed HU measurements, which revealed relatively high values of 52 – 62. However, given the highly raised level of haematocrit, we believe these measurements not to speak against a diagnosis of pseudo – SAH. A paragraph about HU measurements was added to the discussion section (page 7, lines 1 – 6).