Reviewer’s report

Title: Spontaneous closure of a traumatic macular hole in a 50 year old female

Version: 2 Date: 23 January 2011

Reviewer: Ioannis Petropoulos

Which of the following best describes what type of case report this is?: New associations or variations in disease processes

Has the case been reported coherently?: Yes

Is the case report authentic?: Yes

Is the case report ethical?: Yes

Is there any missing information that you think must be added before publication?: Yes

Is this case worth reporting?: Yes

Is the case report persuasive?: Yes

Does the case report have explanatory value?: No

Does the case report have diagnostic value?: Yes

Will the case report make a difference to clinical practice?: No

Is the anonymity of the patient protected?: Yes

Comments to authors:

The authors have adequately addressed the majority of my comments. This case is quite interesting and, in my opinion, warrants publication. The following two issues remain:

1) In the second paragraph of the Discussion, the authors have replaced the proposed mechanisms of idiopathic macular hole spontaneous closure with the proposed mechanisms of traumatic macular hole formation. What is important, though, is to refer to the proposed mechanisms of traumatic macular hole spontaneous closure.

These are well explained in Yamada et al. AJO 2002;134:340-7 (p.346, “Glial cells or RPE cells proliferate from each bank of the hole’s edge to fill the hole..."
bottom and close the hole” in conjunction with a “high glial cell proliferation activity” in young patients), and in Menchini et al. Retina 2003;23:104-6 (p.106, “bridging of neuroretinal tissue over an optically empty subretinal area” and “proliferation of glial cells or regrowth of RPE-derived cells on the outer surface of the bridging neuroretinal tissue”). The release of vitreofoveal adhesion is also considered important for TMH spontaneous closure by Yamashita et al. AJO 2002;133:230-5 (p.235). It’s the reason why Reviewer #2 (Periklis Brazitikos), an experienced vitreoretinal surgeon, believes that, without PVD, the hole of the presented case probably would never have closed.

2) In their reply to my comment #8, the authors say: “To speak in absolute terms, one would have to review each TMH case that closed without surgery and look for the presence of blood that might have played a role in hole closure”. It’s obviously an exaggeration, which make the authors miss the most important point of their case. Glial cell proliferation activity is high in young patients, as all other healing activity in our body. It may not be as high in a 50-year-old adult. Blood may have offered extra proliferating agents to compensate for this and, in conjunction with PVD, may have accelerated the healing process. This, however, does not mean that all the other traumatic holes described in the literature have closed thanks to blood. Besides, to the best of my knowledge, no other report exists of blood on a TMH that closed spontaneously.

**Quality of written English:** Acceptable

**Declaration of competing interests:**

I declare that I have no competing interests.