Reviewer's report

Title: Acute cardiac injury after subarachnoid haemorrhage. A clinical study of two cases.

Version: 2 Date: 20 November 2009

Reviewer: Hannibal Baccouche

Can you understand the case report?: Yes

Do you think the case report is authentic?: Yes

Do you see any ethical problems?: No

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

Is it possible that this case is the first of its kind or may represent an important advance in general medical knowledge?: No

Comments to authors:

Marci and colleagues present two very interesting cases of cardiac complications in the setting of intracranial bleeding in their paper. Based on these two examples pathophysiologic considerations and clinical implications are discussed.

I recommend the paper to be published, pending MINOR REVISION.

My questions and comments are:

1st clinical case:

1.) Did the first patient demonstrate repolarization changes in her resting 12 lead ECG?

2.) Could the observed wall motion abnormalities of the apex and distal interventricular septum be further specified? Was it hypokinesia, akinesia or rather dyskinesia and did other regions of the heart possibly exhibit hyperkinesia (e.g. basally, like in Tako-Tsubo-Cardiomyopathy)?

3.) I recommend a consistent labeling as the authors switch between “abnormalities” and “anomalities”.

4.) Range values should be provided for the laboratory values.

5.) Are the authors willing to present any pictorial material, e.g. echo-images, ECG or CT-scan? Although it is no obligation for this work, pictorial material could grade up the work for the readers.
2nd clinical case:

1.) What was the measured left ventricular ejection fraction, as this patient had significantly increased NT-pro-BNP levels, but only little troponin. Did she have clinical symptoms of heart failure requiring medical therapy additionally to treatment of the rhythm-disturbance?

2.) Could the observed wall motion abnormalities of the apex and distal interventricular septum be further specified? Was it hypokinesia, akinesia or rather dyskinesia and did other regions of the heart possibly exhibit hyperkinesia (e.g. basally, like in Tako-Tsubo-Cardiomyopathy)?

3.) Range values should be provided for the laboratory values.

4.) Are the authors willing to present any pictorial material, e.g. echo-images, ECG or CT-scan? Although it is no obligation for this work, pictorial material could grade up the work for the readers.

Discussion:

1.) How often do repolarization-changes in subarachnoid hemorrhage occur? Concrete numbers should be provided here similar to the ones for rhythm- and conduction abnormalities given in the previous paragraph.

2.) Citation number 8 is not correct:

The correct citation is: J Neurosurg. 2003;98:524-528.

3.) The “classical definition” of Tako-Tsubo-Cardiomyopathy formerly excluded patients with head trauma and intracranial bleeding (1), however there seems to be good evidence that Tako-Tsubo-Cardiomyopathy has some significant similarities with the cardiac dysfunction seen in subarachnoid hemorrhage and may therefore create a substantial overlap with neurogenic stress cardiomyopathy (2-8).

I suggest that the authors should comment on this aspect in their discussion section in detail!

4.) As the authors emphasize the importance of distinguishing acute coronary syndrome from neurogenic myocardial injury as the primary information of their paper, I recommed to clearly state that to date there is no secure method to differentiate both entities.

Although an acute coronary lesion is a rare finding in the setting of subarachnoid hemorrhage, compared to ischemic stroke for example (9), and ECG-alterations and wall motion abonormalities are more likely due to “toxic-catecholamine-effects”, a simultaneous acute coronary artery problem is possible (10-12), perhaps caused by stress-induced-plaque rupture?
Therefore it remains an interdisciplinary clinical decision and challenge whether to catheterize a patient, taking into account multiple aspects of the patient’s clinical presentation.

5.) The secondary information of the paper, whether to consider brain dead patients with potentially reversible cardiac dysfunction as transplant-donors is an important issue because of organ shortage. This aspect is however not easy to elucidate and I would ask the authors to make a comment on the availability of consensus-data or further clinical experiences (if available), when or if dysfunctional hearts can be or are used as donor-organs (13,14).

General remark:

In the appendage of the paper the authors state that: “...written informed consents were obtained from the patients for publication of this case report...” As the first patient died in the course of her illness, I ask the authors to state whether an affiliated had given consent and to respectively adjust the declaration if appropriate.

Reference List


(5) Gnecchi-Ruscone T. Letter regarding article by Banki et al., "Acute neurocardiogenic injury after subarachnoid hemorrhage.". Circulation 2006 May 9;113(18):e751.


(9) Sheifer SE, Gersh BJ, Yanez ND, III, Ades PA, Burke GL, Manolio TA.


**Declaration of competing interests:**

I declare that I have no competing interests