East African Scholars Journal of Medical Sciences
Abbreviated Key Title: East African Scholars J Med Sci
ISSN 2617-4421 (Print) | ISSN 2617-7188 (Online) |
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

DOI: 10.36349/easms.2019.v02i05.010 Volume-2 | Issue-5 | May -2019 |

Letter to the Editor

Myocardial damage from status epilepticus

Josef Finsterer, MD[1] and Claudia Stöllberger, MD[2]

[1]Krankenanstalt Rudolfstiftung, Messerli Institute, Austria
[2]Medical Department, Krankenanstalt Rudolfstiftung, Vienna, Austria

*Corresponding Author
Univ.Doz. DDr. J. Finsterer

Keywords: myocardial contraction bands (MCBs).

With interest we read the article by Manno, E.M. *et al.*, (2005) who interpreted the occurrence of myocardial contraction bands (MCBs) in patients who died during status epilepticus (SE) to result from excessive catecholeamine-release, causing myocardial over-contraction and lastly heart failure (Manno, E.M. *et al.*, 2005). The study raises concerns.

The term MCB is ambiguous (Baroldi, G. *et al.*, 2001). According to which criteria were MCBs diagnosed? How were MCBs quantified? Which was the distribution and morphology of MCBs? In cardiac disease MCBs may occur ubiquitously and in non-cardiac disease focally. From which parts of the myocardium were the specimens taken? There is no description of the stainings and microscopic methodology applied. Where does the evidence derive from that MCBs localize in the vicinity of sympathetic nerve-terminals? How to explain the relation between MCBs and heart failure? Do MCBs impede cardiac function? Are these foci triggers for malignant arrhythmias? How to explain the relation between MCBs and duration and intensity of seizure-activity without considering pre-morbid myocardial damage? According to table 1 only in a single patient MCBs can be attributed to SE. In all other patients other pathologies may have been causative. It is well known that MCBs also result from intravenous catecholamine-infusion, pheochromocytoma, adrenergic stress, coronary-heart-disease, or coronary-artery spasms (Baroldi, G. *et al.*, 2001). Were all these conditions considered? Patho-anatomic data, like wall thickness, trabeculation-rate, prevalence of coronary-artery-stenosis, valvular pathology, and scars are missing. Data about ECG findings are lacking. It remains unclear how coronary-heart-disease was defined? Does "global decompensation" mean sudden dysfunction of the right and left ventricle or does it refer to the Takotsubo phenomenon? Death during SE is assumed not only due to "cardiac decompensation" but also due to rhythm-abnormalities. Sympathetic overdrive from SE may cause coronary spasms, malignant arrhythmias, and consecutively heart failure and death.

There is complete absence of data concerning etiology and EEG morphology of seizures.

Data on current medication during SE are also missing. Of particular interest would be catecholamines, β-blockers, and anti-epileptic drugs. Why were the results of group 2 not presented?

Overall, there are several concerns attributing MCBs solely to adrenergic over-stimulation during SE. Before finally assessing the role of MCBs in death during SE, prospective, highly powered studies are warranted.

REFERENCES

1. Manno, E.M., Pfeifer, E.A., Cascino, G.D., Noe, K.H., & Wijdicks, E.F. (2005). Cardiac pathology in status epilepticus Ann Neurol, 58, 954-957.
2. Baroldi, G., Mittleman, R.E., Parolini, M., Silver, M.D., & Fineschi, V. (2001). Myocardial contraction bands. Definition, quantification and significance in forensic pathology. Int J Legal Med, 115, 142-151.

Quick Response Code

Journal homepage:
http://www.easpublisher.com/easjms/

Article History
Received: 15.04.2019
Accepted: 30.04.2019
Published: 19.05.2019