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Letter to the Editor

Stroke-induced myocardial damage: location-dependent or of extra-cerebral origin?

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In a recent article Ay *et al.* (2006) reported on a study of on 738 consecutive patients with ischemic stroke, of which 50 had elevated serum cardiac troponin T (cTnT) in the absence of any apparent cause (Ay, H., *et al.*, 2006). Elevated cTnT was associated with infarctions of the right insula and the right inferior parietal lobe. The report raises the following questions and concerns:

Myocardial damage in these patients is explained by enhanced sympathetic activity from desinhibition of the right anterior insula by stroke of the right posterior, superior and medial insula and the right inferior parietal lobule, resulting in increased heart rate and increased blood pressure. How to explain elevated cTnT in the 16 patients with infarcts in the left middle cerebral artery territory, where parasympathetic activities are assumed to be located, in 1 patient with brainstem infarction, and in 5 patients with cerebellar infarction? Was parasympathetic activity blocked in these patients such that sympathetic activity could become dominant?

cTnT may not only be elevated in patients with stroke but also in patients with chemotherapy, inclusion body myositis (Schwarzmeier, J. D. *et al.*, 2005), hemodialysis (Selby, N. M. *et al.*, 2006), pericarditis, myocarditis, cardiac amyloidosis, diagnosed only by myocardial biopsy, uremic cardiomyopathy in kidney failure (Fahie-Wilson, M. N. *et al.*, 2006). Takotsubo phenomenon, diabetic ketoacidosis, or in marathon runners. Were all these differentials adequately excluded in the 50 patients?

Electrocardiographic and echocardiographic data should be presented. Insular infarcts have been shown to be associated with QT prolongation (Tatschl, C. *et al.*, 2006). Did patients with elevated cTnT have a higher prevalence of arrhythmias, heart failure, or a higher mortality than controls? Since coronary angiography was not carried out in each of the 50 patients with cTnT of "unknown'' cause, coronary heart disease had not been excluded in all of them. How big was the fraction of patients who underwent coronary angiography? Were serum creatine-kinase levels also assessed and did they correlate with cTnT levels?

In addition to cTnT, also cTnI is cardio-specific. Why did the authors determine cTnT and not cTnI? In case they determined cTnI how was the correlation between cTnT and cTntI in the included patients?

No detailed description of the clinical neurologic findings is presented. Did the more severely disabled patients have higher cTnT values than those with minor neurological deficits?

Overall, attribution of cTnT elevation solely to affection of the central sympathetic or parasympathetic system by the stroke is not justified as long as not all other cardiac and extra-cardiac causes of cTnT elevation had been adequately excluded.

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REFERENCES

1. Ay, H., Koroshetz, W. J., Benner, T., Vangel, M. G., Melinosky, C., Arsava, E. M., ... & Sorensen, A. G. (2006). Neuroanatomic correlates of stroke-related myocardial injury. *Neurology*, *66*(9), 1325-1329.
2. Schwarzmeier, J. D., Hamwi, A., Preisel, M., Resl, C., Preusser, M., Sluga, E., ... & Shehata, M. M. (2005). Positive troponin T without cardiac involvement in inclusion body myositis. *Human pathology*, *36*(8), 917-921.
3. Selby, N. M., Fluck, R. J., Taal, M. W., & McIntyre, C. W. (2006). Effects of acetate-free double-chamber hemodiafiltration and standard dialysis on systemic hemodynamics and troponin T levels. *ASAIO journal*, *52*(1), 62-69.
4. Fahie-Wilson, M. N., Carmichael, D. J., Delaney, M. P., Stevens, P. E., Hall, E. M., & Lamb, E. J. (2006). Cardiac troponin T circulates in the free, intact form in patients with kidney failure. *Clinical chemistry*, *52*(3), 414-420.
5. Tatschl, C., Stöllberger, C., Matz, K., Yilmaz, N., Eckhardt, R., Nowotny, M., ... & Brainin, M. (2006). Insular involvement is associated with QT prolongation: ECG abnormalities in patients with acute stroke. *Cerebrovascular Diseases*, *21*(1-2), 47-53.