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Inappropriate bradycardia in patients with Ebola virus disease. About a cohort of nine consecutive patients in Conakry, Guinea

Gilles Rolland Cellarier*, Julien Bordes, Raphaël Poyet, Frédéric Pons, Eléonore Capilla, Christophe Jego, Sébastien Kerbel
HIA Sainte Anne, Toulon, France
*Corresponding author: gilles.cellarier@orange.fr (Gilles Rolland Cellarier)

Background West African outbreak of Ebola virus disease (EVD) currently affects urban areas in Guinea. Although some cases of bradycardia have been reported since the first Ebola outbreak in 1976, they have never been documented to our knowledge.

Methods we analyse heart rate (HR) for our cohort of nine consecutive patients (5 males, mean age: 38.7 years [range: 27-62]) testing positive for EVD and admitted to the EVD Treatment Centre (EVDTC) in Conakry, Guinea. Treatment and care were standardized, all healthcare workers by wearing personal protective equipment. ECG was recorded if HR was <50 bpm. Eight patients recovered. Mean hospitalization duration was 11.1 days [range: 2-21]. No patient had cardiovascular risk factor or cardiac treatment.

Results we observed significant sinus bradycardia (<45 bpm) in one patient, without conductive disturbance or repolarization abnormalities on ECG, whereas mean HR recorded no/moderate tachycardia at admission (mean [±SD], 76±23 beats per minute [bpm], range: 43-107) for all patients. During follow-up, there is a trend to significant increase in mean HR between Day 1 and Day 11 (89±19 bpm) [Wilcoxon test: p=0.051].

Discussion Despite several factors of tachycardia (stress, fever, pain, infection, dehydration), we can note a relative bradycardia for all patients, with a paradoxical increase in HR during hospitalization. Clinical inappropriate bradycardia or dissociated pulse was previously described during the first Ebola outbreak in 1976, as in the current epidemic. The hypothesis for toxin inducing bradycardia in EVD has never been documented. Despite the low number of patients in this cohort, we therefore hypothesized the possibility of a central neurological cause, due to encephalitis. Indeed, most of our patients presented significant clinical encephalitis signs.

Conclusion patients infected with Ebola virus may have an inappropriate bradycardia. The cause of this bradycardia could be multifactorial.

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Why distribution of our valvular heart disease differs from the European one?

Nora Nabila Ali-Tatar Chentir*, Yacine Tir, Sabrina Alane, Mohand Tayeb Chentir
CHU Mustapha, Alger, Algérie
*Corresponding author: chentir.nora@gmail.com (Nora Nabila Ali-Tatar Chentir)

Purpose To assess the particular features of valvular heart disease (VHD) in patients (pts) visited our outpatient clinic.

Methods We studied prospectively since December 2006 clinical and echocardiograms data of 1173 pts mean age 48±11.9years. Among them, mitral stenosis (MS):286 pts 201female, aged 16-84, mean 44±12yearsOrganic Mitral regurgitation: 299 pts 143F, aged 07-98, mean 54±18 years. Aortic stenosis: 91pts 39F, aged 27-82, mean 60±14.23 years, Aortic regurgitation: 80pts 24F aged 11-97, mean 57±16years. Mixed valve disease: 417pts 243F aged 11-84, mean 48±18years.

Results Regarding MS from the assessment of mitral anatomy according to the Cormier score only 20% of the pts were in the group 1 and underwent percutaneous mitral valve commissurotomy (PMC) whereas 72% of them had replacement with prosthetic valve. In MR, only 12 patients were on type 2 of Carpentier, 76% of the patients underwent replacement with prosthetic valve. Despite low risk score for intervention in 68% of the pts, almost half of them were not operated.

Conclusion Despite the disappearance of rheumatic fever, the valvular sequelae are the predominant etiology of valvulopathy. This explains the high rate of mechanical prosthesis as compared with repair and percutaneous mitral commissurotomy.

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