Original Research Paper

Volume -10 | Issue - 4 | April - 2020 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Cardiovascular

MITRAL STENOSIS AND VASCULAR EPILEPSY : ABOUT 36 CASES

Abdellatif Ezzouak*	Department of Cardiology MilitaryHospital Moulay Ismail Meknes-Morocco*Corresponding Author
Abdellah Taous	Department of NeurologyMilitaryHospital Moulay Ismail Meknes-Morocco
AchrafZaimi	Department of cardiologyMilitaryHospital Moulay Ismail Meknes-Morocco
Mehdi Badidi	Department of cardiologyMilitaryHospital Moulay Ismail Meknes-Morocco

ABSTRACT **INTRODUCTION:** Mitral stenosis (MS) is a major embolic heart disease, especially when associated with atrial fibrillation. Ischemic strokes are the first manifestations of these embolic accidents, and they may lead to vascular epileptic seizures. We present a sample cases of MS complicated of vascular epilepsy, which can sometimes reveal this valvularcardiopathy. **PATIENTS AND METHODS:** retrospective study reporting 36 patients, Who presented at least one epileptic seizure related to MS complicated of an ischemic stroke, confirmed by cerebral imaging (CT scan and / or MRI) **RESULTS:**20 men (55.5%), with an average age of 35.8 +/- 5.7 years. Atrial fibrillation was permanent in 29 cases (80.5%). On the echocardiography, the MS was severe in 27 cases (75%). The left atrium was dilated in all patients. Spontaneous contrast was demonstrated in 31 cases (86%). A thrombus of the left atrium and especially of the left atrial appendage detected in 22 cases (61%). The ischemic stroke was of cortical site in 25 cases (69.4%). The electroencephalogram was abnormal in 31 patients (86%). All patients were put on a vitamin k antagonist with indication for a valve replacement or a mitral commissurotomy in case of moderate MS. **CONCLUSION:** MS is a potentially embolic source, causing ischemic stroke and exposing to an increased risk of developing vascular epilepsy. Primary prevention, in our context, by eradicating rheumatic fever and early detection of rheumatic valve disease, remains necessary to prevent valvular disease's complications.

KEYWORDS : mitral stenosis, schemic strokes, vascular epilepsy

INTRODUCTION:

Mitral stenosis (MS) is an obstacle to leftatrioventricularblood flow during diastole. It remainslargelydominated by the rheumaticetiology. Thromboembolic complications occurin 10 to 20% of cases and these are mainlyischemicstrokeswichcanbesubclinical orclinicallymanifest,revealingheartdisease. Therfore, they expose to the occurrence of vascularepilepsy, representing about 7% of epilepsies [1].

PATIENTS AND METHODS :

This is a retrospectivestudyconducted over a period of 18 years, betweenJanuary 2000 and December 2018, carried out within the cardiologydepartment of the Moulay Ismail MilitaryHospital in Meknes-Morocco, reporting 36 patients, havingpresentedat least an epilepticseizurerelated to a MS complicated by an ischemicstoke,confirmedeither by CT scan and/or brainMRI. Thus, all patients followed for epilepsyduringchildhood and/or adolescence or thosewith normal brainimagingwereexcluded. All our patients underwent a cardiovascular and neurologicalexamination, a restingelectrocardiogram(ECG), a 24-hours Holter ECG if the basal rhythmwas sinusal to the resting ECG, a transthoracic and transoesophagealechocardiography (TTE and TOE) and CT scan and / or brainMRI.

RESULTS:

These were 20 men (55.5%) and 16 women (44.5%), average age 35.8 +/- 5.7 years (range: 22 and 52 years), selected on the basis of 986 cases of MS followed in our department, a frequency of 3.6%.

The inaugural epileptic seizure was an exclusive mode of revelation of the MS in 5 cases (13.8%). The EEG was abnormal in 31 cases (86%). The resting ECG revealed permanent atrial fibrillation (AF) in 29 cases (80.5%), while it was paroxysmal in 7 cases, detected with the 24hoursHolter ECG.

On TTE and TOE, the MS was severe (mitral valve area ≤1.5cm²) in 27 cases (75%) and moderate in the other cases The left atrial (LA) was dilated in all patients with an average area of 33.6 cm². A thrombus of the LAparticularly of the LA appendage was detected by TOE in 22 cases (61%). While an aspect of spontaneous contrast was present in 31 cases (86%).

Brain CT was performed in all patients, showing a cortical stroke in 25 cases (69.4%). In the absence of abnormalities, MRI detected cortical lesions in T1 hyposignal and T2 hypersignal consistent with stroke, mostly small.

Therapeutically, all our patients were put on a vitamin K antagonist (VKA) . A mitral valve replacement by mechanical prosthesis was indicated in 10 cases (27.7%), a mitral commissurotomy in 17 cases (47%) and regular follow-up in case of moderate MS.

DISCUSSION:

The MS forms an obstacle to the diastolic filling of the LV, which creates a pressure gradient between the LA and the LV. The average pressure of the LA increases, then it expands, thus favoring the occurrence of arrhythmia, particularly AF.

The dilation of the LA, the severity of the mitral stenosis and especially the presence of spontaneous contrast (SC) are the main predictors of the occurrence of thromboembolic complications in MS [2].

The size of the LA is a marker of the severity and chronicity of the pressure and / or volume overload to which it is subjected.

Thus, intra-LA CS is reported in 55 to 67% of cases of MS; even 100% in case of associated AF. Its presence is considered to be a thromboembolic risk marker justifying the prescription of VKA, even in patients with sinus rhythm [3].

In our sample, the SC was observed in 86% of the cases, due to the severity of the MS and the size of the LA, which is very dilated in the majority of the cases.

AF is present in 30 to 40% of symptomatic MS cases. It is first paroxysmal and then becomes permanent. Its embolic risk is well demonstrated by the various studies and constitutes a major predictive factor for the occurrence of thromboembolic accidents. These accidents can be expressed in the form of strokes which are sometimes fatal or disabling.

Strokes present a high risk of epilepsy [5]. Although the pathophysiology of post-stroke epileptic seizures (ES) is not fully understood, several theories have been discussed, usually differentiating early and late attacks (occurring more than 6 to 12 months after stroke). Indeed, the occurrence of an ischemic stroke is considered to be an acute cerebral aggression, which can induce ES or

even a status epilepticus, by increasing the extracellular concentration of an excitatory neurotransmitter: Glutamate . The latter, is likely to favor the occurrence of ES or neuronal discharges during experimental studies [6]. In addition, the occurrence of ES in the acute phase of stroke increases the risk of developing autonomous vascular epilepsy. Thus, the vascular lesion constitutes an aberrant structural modification, forming a potential epileptogenic focus [7].

Vascular epilepsy is defined as the occurrence of at least one unprovoked seizure at least one week after the occurence of the stroke according to the International League Against Epilepsy [8]. In the majority of cases, the series reported concern elderly subjects with multiple cardiovascular risk factors. In our sample, epilepsy is observed in a younger population, less than 52 years old; this is due to the specific presence of an embolic heart disease (MS).

The incidence of epilepsy after a stroke is assessed differently in several studies and varies from 4 to 50% depending on the types of stroke [9,10]. Thus, the frequency of epilepsies is higher in cases of hemorrhagic stroke than ischemic [11,12]. The cortical location of the stroke is an epileptogenic factor; it is considered in the various studies as an independent factor, predictive of ES in comparison with subcortical lesions [13-15].

Performing the EEG after a stroke lacks specificity to judge patients who will develop epilepsy. Among the EEG of the patients in our series, five were without abnormalities, which justified that the normality of recording does not always eliminate the presence of epileptogenicity. We will retain the typical electroencephalographic image: the Periodic Lateralized Epileptiform Discharges [16].

Regarding treatment, there are no specific therapeutic trials on patients with vascular epilepsy.

Thus, it is consistent to consider that beyond the early period of the first two weeks after the stroke, the occurrence of a seizure in the context of acquired brain injury exposes to a risk of recurrence sufficiently high to prescribe treatment antiepileptic. [17].

This prescription must take into account the comorbidities of the patient, his age and the drug interactions in this case VKA treatment.

It is likely that most first generation antiepileptics, including phenytoin, phenobarbital and carbamazepine, are not the most suitable for the treatment of post-stroke epilepsy due to their potentially negative impact on functional recovery and their pharmacokinetic profile including the risk of drug interaction [18].

These antiepileptic drugs, having an enzyme-inducing property dependent on cytochrome P450 3A4, are likely to have an impact on the balance of anticoagulation and to be responsible for thromboembolic complications in the event of overdose or hemorrhagic complications in the event of stop treatment. New generation antiepileptics (Lamotrigine, Gabapentin and Levetiracetam) present less risk of drug interaction and provide a beneficial contribution to functional recovery [19,20].

CONCLUSION:

MS is a potentialembolic source causing stroke and exposing to an increased risk of occurrence of vascular epilepsy, which can reveal valvulopathy even before the appearance of specific clinical signs. Antiepileptic drugs are indicated from the first seizure and their interaction with VKA requires close monitoring of coagulation and perfect therapeutic compliance.

Primary prevention, in our context, by eradicating rheumatic fever and early detection of rheumatic valve disease, remains necessary to prevent valvular disease's complications.

REFERENCES :

1. Zou S and al. The pooled incidence of post-stroke seizurein 102008 patients. Top Stroke Rehabil. 2015 Dec;22(6):460–7
2. Keenan NG and al. Usefulness of left atrial volume versus diameter to assessthromboembolicrisk in mitral stenosis. Am J Cardiol. 2010;106:1152–1156
3. Daniel WG and al. Left Atrial spontaneousechocontrast in mitral valve disease : an indicator for an increasedthromboembolicrisk. J Am CollCardiol 1988;11:1204–11
4. Braunwald'sHeartDisease: A Textbook of CardiovascularMedicine. 10th Edition. Philadelphia: Elsevier; 2015. P.1446-1480
5. Mendez M and al. Seizures in elderly patients withdementia: epidemiology and management. DrugsAging. 2003;20:791–803
6. Sun, D.A. and al. Calciumdependentepileptogenesis in an in vitro model of stroke-induced "epilepsy". Epilepsia, 2002
7. Strzelczyk, A. and al. Prospective evaluation of a post-stroke epilepsyriskscale. J. Neurol., 2010, 257(8), 1322-1326
8. InternatIonalLeagueAgaInstEpIlepsy. Guidelines for epidemiologicstudies on epilepsy. Epilepsia 1993;34:592–6.
9. Benbir G and al. The epidemiology of post-stroke epilepsyaccording to stroke subtypes.Acta NeurolScand. 2006 Jul;114(1):8-12.
10. Ferro JM and al.Poststrokeepilepsy: epidemiology, pathophysiology and management.DrugsAging. 2004;21(10):639-53.
11. Labovitz, D.L and al. Prevalence and predictors of earlyseizure and statusepilepticusafter first stroke. Neurology, 2001, 57(2), 200-206
12. Burn, J. and al. Epilepticseizuresafter a first stroke: the Oxfordshire Community Stroke Project. BMJ, 1997, 315(7122), 1582-1587
13. Bladin, C.F. and al. Seizuresafter stroke: a prospective multicenterstudy. Arch. Neurol., 2000, 57(11), 1617-162
14. Procaccianti and al. Seizures in acute stroke: incidence, riskfactors and prognosis.Neuroepidemiology, 2012, 39(1), 45-50.
15. Waterhouse, E. Predictors of earlyseizuresafter stroke. EpilepsyCurr., 2002, 2(3), 75-76
16. Mecarelli, O. and al. EEG patterns and epilepticseizures in acute phase stroke. Cerebrovasc. Dis., 2011, 31(2), 191-198
17. De Reuck, J. Management of stroke-relatedseizures. Acta Neurol. Belg., 2009, 109(4), 271-276.
18. Stockley IH. Anticonvulsantdrug interactions. In: Stockley IH, Cochrane Library. Drug interactions. 5th ed. London: Pharmaceutical Press, 1999: 273-330
19. Karceski, S. and al. Treatment of epilepsy in adults: expert opinion, 2005. EpilepsyBehav., 2005, 7(Suppl 1), S1-64. quiz S65-6
20. Shetty, A.K. Prospects of levetiracetam as a neuro protective drug against status epilepticus, traumaticbraininjury, and stroke. Front. Neurol., 2013, 4, 172