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Spastic Angor Revealed by Acute Coronary Syndrome: A Case Report with Literature Review

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Abstract **Case Report**

The Prinzmetal angor or spastic angor is considered rare but its prevalence is probably underestimated in particular in cases of atheroscopic coronary lesions,its diagnosis remains important, however, because of its poor prognosis and its therapeutic particularities, we report the case of a young patient and the problem of the diagnosis of spastic angor. We will propose a review of the medical literature concerning the diagnostic means at our disposal and the modalities of care of these patients.

Keywords: Angor spastic; coronary; treatment.

INTRODUCTION

The spastic angor is a rare condition, affecting especially the young subject, its diagnosis is retained in subjects with a typical angina chest pain associated with STEMI or NSTEMI and focal changes in the vessel caliber observed during coronary angiography, its diagnosis is however important because of its poor prognosis and its therapeutic peculiarities. We report a positive ST coronary syndrome in relation to severe coronary spasm.

CASE REPORT

Mrs E.M, 22 years old, with no special history and no cardiovascular risk factor other than passive smoking, is admitted to the CICU for the management of acute coronary syndrome with STEMI. The clinical examination is normal, the ECG records a regular sinus rhythm FC = 72/mn, a ST offset in ASA with sequelae of necrosis in the same territory. The troponin is negative. Echocardiography has objectified an aspect of ischemic heart disease with disorders of segmental kinetics with anterior wall akinesia type, antero-septal, inferro-septalseptal, tip and adjacent segments in severe left ventricular dysfunction LVEF at 30%.An emergency coronary angiography showed a spastic occlusion of the proximal left anterior descending artery (LAD) that was lifted after injection of Risordan, with atheromatous plaques on the middle circumflex and the first segment of the right coronary. MRI showed a sequel of non-viable necrosis in the LAD territory. The patient is discharged on calcium inhibitor Vesapamil full dose, Risordan, anti-caking platelet and statin 40mg. The evolution was marked by angina recurrence despite optimal treatment. Indication of preventive angioplasty of proximal LAD is discussed.

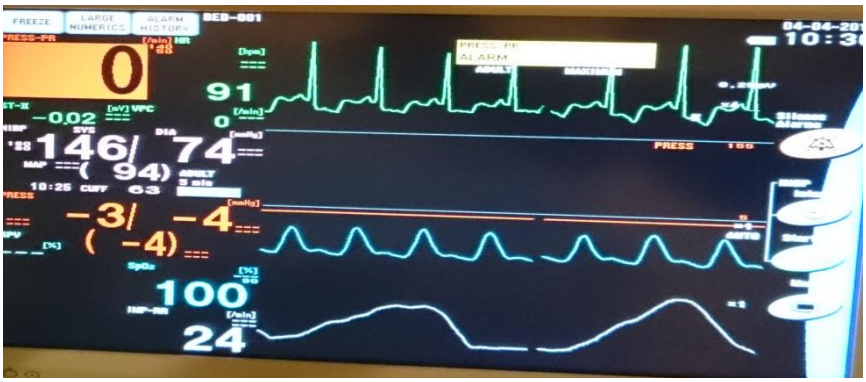

Fig-1: Spastic angor episode revealed by NSTEMI on the scope

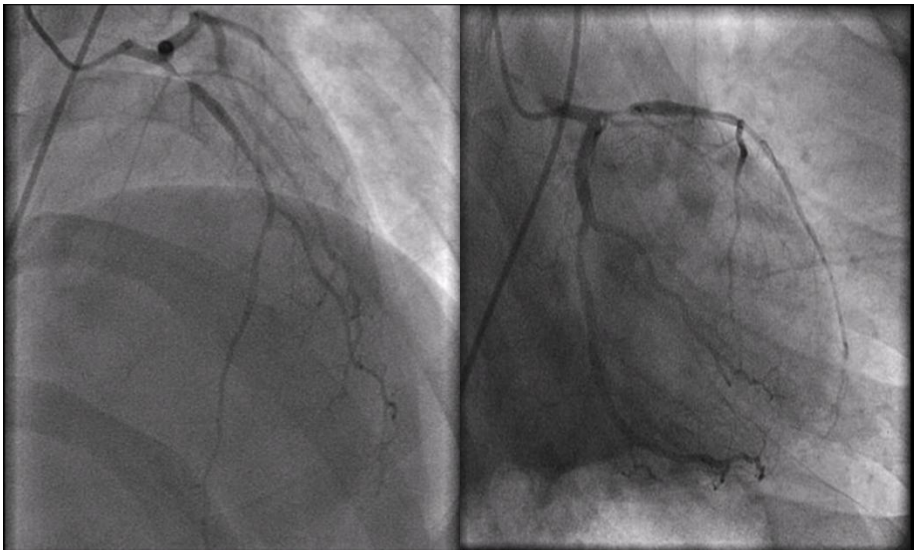

Fig-2: Very severe spastic lesion of proximal LAD

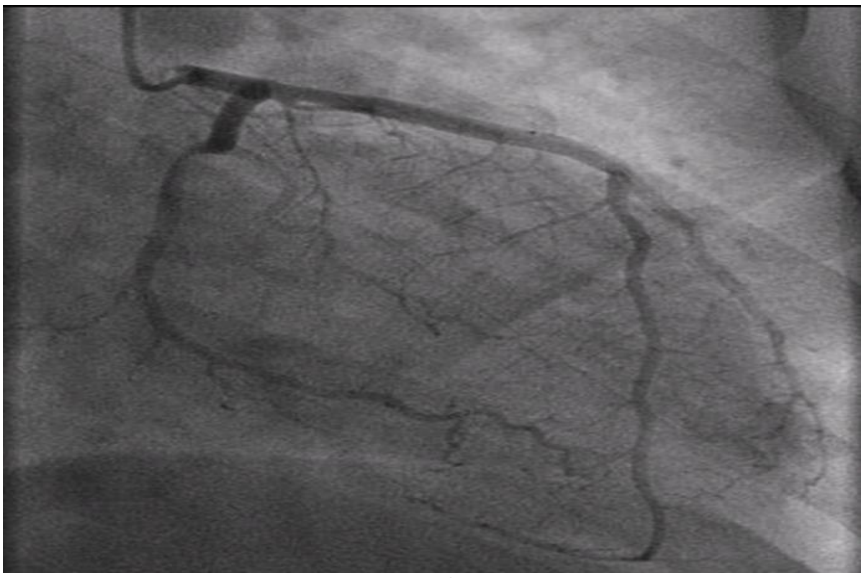

Fig-3: Resolutive lesion after risordan injection

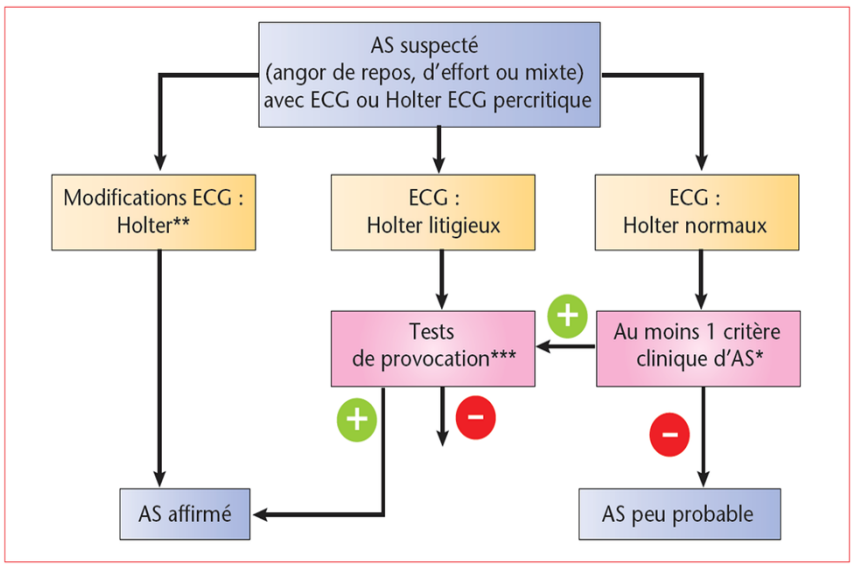

Fig-4: Diagnosis of angor spastic according to JAPENESE circulation society

DISCUSSION

Spastic angor is common in Asian populations, encountered in 40 % of cases of ischemic heart disease in Japan [2], it is assumed very rare in the European population representing only 1,5% of cases [3].

Spastic angor affects most men since women account for only 12-25% of cases in the largest series of published patients [5], coronary spasm causes a significant number of myocardial infarctions in the 5% range. The modes of revelation of the spastic angor are diverse and one encounters in the literature most often a resting angor or mixed, exceptionally an angor of pure effort, cardiac arrest or syncope and less rarely acute coronary syndrome. in our patient the coronary spasm is very severe it is responsible for an anterior myocardial infarction with severe LV dysfunction. It is recognized that coronary spasm results from endothelial dysfunction and or hyperreactivity of smooth muscle cells with endogenous stimulis (acetylcholine, serotonin, catecholamines) or exogenous (therapeutics such as ergonovin, toxic with vasoconstrictor effect) [6]. Most cases of spastic angor are medically treated [4, 7], it is recognized that spasm disappears or decreases significantly due to treatment with coronary vasodilators [4, 7], the drugs used are essentially calcium inhibitors and nitrated derivatives [9]. They reduce the frequency of episodes of pain and the risk of sudden death. In our observation, despite an optimal and well conducted vasodilator treatment, the patient remains very symptomatic hence the discussion of a preventive angioplasty.

The Méthergin test is very sensitive, especially if it is performed shortly after the last spontaneous pain. Strict diagnostic criteria and a specific decision tree are proposed by the Japanese Circulation Society (2) (Figure-3). The particularity of the European population is the highest incidence of associated atherothal lesions that can complicate the diagnosis. Coronary angioplasty may have a place in the treatment of SA but it is limited to patients with symptomatology refractory to maximum medical treatment and with well-localized spasm [10], it has a purely mechanical effect and does not solve the problem of endothelial dysfunction. The existence of atheromatous lesions associated with spastic angor is an aggravating factor in prognosis [11].

Smoking cessation is indisputably definitive in the treatment, not only in the case of the hygienic-dietary rules of atherosclerous disease but also because of its involvement in the genesis of spasms. In addition, no study validates or invalidates the use of platelet antiaggregates in pure vasospatic angor.

CONCLUSION

Coronary spasm is the result of an acute disturbance of coronary vasomotricity. Better diagnostic support for this entity would allow for specific adaptation of treatment and improved prognosis.

REFERENCE

1. Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N. Angina pectoris I. A variant form of angina pectoris: preliminary report. The American journal of medicine. 1959 Sep 1;27(3):375-88.
2. JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (coronary spastic angina)(JCS 2008). Circulation Journal. 2010;74(8):1745-62.
3. Lanza GA, Sestito A, Sgueglia GA, Infusino F, Manolfi M, Crea F, Maseri A. Current clinical features, diagnostic assessment and prognostic determinants of patients with variant angina. International journal of cardiology. 2007 May 16;118(1):41-7.
4. Mishra PK. Variations in presentation and various options in management of variant angina. European journal of cardio-thoracic surgery. 2006 May 1;29(5):748-59.
5. D'Agate DJ, Schwarz R, Lazar JM. Coronary vasospasme-induced ventricular tachyarrythmias. JInvasive Cardiol, 2002;14: 609-14.
6. du Fretay XH, Blanchard-Lemoine B, Schnebert B, Viossat J. Angor spastique: quel regard en 2011?. InAnnales de Cardiologie et d'Angéiologie 2011 Dec 1, 60(6):317-323.
7. Chevalier P, Dacosta A, Defaye P, Chalvidan T, Bonnefoy E, Kirkorian G, Isaaz K, Denis B, Touboul P. Arrhythmic cardiac arrest due to isolated coronary artery spasm: long-term outcome of seven resuscitated patients. Journal of the American College of Cardiology. 1998 Jan 1;31(1):57-61.
8. Tani S, Nagao K, Anazawa T, Kawamata H, Furuya S, Takahashi H, Iida K, Fuji T, Matsumoto M, Kumabe T, Sato Y. Treatment of coronary spastic angina with a statin in addition to a calcium channel blocker: a pilot study. Journal of cardiovascular pharmacology. 2008 Jul 1;52(1):28-34.
9. Krishnan U, Win W, Fisher M. First report of the successful use of bosentan in refractory vasospastic angina. Cardiology. 2010;116(1):26-8.
10. Hajlaoui N, Tarragano F, Raisky P, Beaufils P, Henry P. Angor spastique réfractaire au traitement médical traité par angioplastie avec mise en place d'un stent. À propos d'un cas et revue de la littérature. InAnnales de cardiologie et d'angeiologie 2010 Apr 1, 59(2):103-106.
11. Beltrame JF, Sasayama S, Maseri A. Racial heterogeneity in coronary artery vasomotor reactivity: differences between Japanese and Caucasian patients. Journal of the American College of Cardiology. 1999 May 1;33(6):1442-52.