

Iatrogenic Left Ventricular Dysfunction after Exogenous Administration of Intravenous Adrenaline

Antonio L Arrebola Moreno

Department of Cardiology, Inmaculada Concepcion Hospital, Spain

***Corresponding Author:** Antonio L Arrebola Moreno, Department of Cardiology, Inmaculada Concepcion Hospital, Spain.

Received: December 12, 2016; **Published:** January 09, 2017

Abstract

We present the clinic case of a healthy 19 year-old women that suffered an anaphylactic reaction after the sting of a vermin. She was administered intravenous adrenaline and developed acute left ventricular dysfunction.

Keywords: *Adrenergic myocardopathy; Adrenaline; Ventricular dysfunction*

Volume 1 Issue 1 January 2017

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Manuscript

We present the clinical case of a healthy 19 year-old woman without any relevant past clinical history that suffered the sting of a vermin (probably a wasp) while riding a bicycle. Afterwards she presented an allergic reaction with skin rash and dyspnea. She went to the nearest emergency center and was administered a single dose of subcutaneous adrenaline relieving her symptoms. A few minutes later she started a new dyspnea episode and was administered an intravenous 80mg of methylprednisolone and 250 mg of adrenaline. After the last dose, she started an intense palpitation and sensation an oppressive non-irradiated chest pain with vegetative symptoms, vomiting and sweating. She was performed and EKG (Figure 1A) were sinus tachycardia, mild ST elevation and inverted T waves in the inferior-lateral territory, mild descended ST segment in V3 and V4, and marked (1.5 mm) elevation of ST segment in I and aVL were found. 20-30 minutes later the chest pain and EKG changes disappeared (Figure 1B). The peak levels of troponin-I were 2.75. The following days she was performed an echocardiogram (Figures 2A and 2B) were a mild dilated left ventricle (54 mm) was found together with global hypokinesia and moderately decreased ejection fraction (37%). The patient remained asymptomatic in the Cardiology ward and started treatment on Bisoprolol 2.5 mg. One week later a control echocardiogram revealed an ejection fraction of 50% and she was discharged asymptomatic. Finally, another echocardiogram was performed 6 months later showing an ejection fraction of 65% and bisoprolol was removed.

Several clinical cases have been reported regarding transient apical dyskinesia (Tako-Tsubo syndrome) after the administration of intravenous catecholamine [1,2]. Moreover, some catecholaminergic myocardopathy cases have been reported [3,4] in patients presenting pheochromocytoma. However, no global left ventricular dysfunction (without segment wall motion abnormalities) in a complete healthy heart has been reported in this context. This case support the theory of myocardial contusion as a cause of ventricular dysfunction in patients suffering pheochromocytoma, thus, the catecholamine excess would lead to calcium overload mediated by cyclic adenosine-triphosphate that would decrease the myocyte's synthetic activity and viability [5]. Although other pathophysiological pathways cannot

Citation: Antonio L Arrebola Moreno. "Iatrogenic Left Ventricular Dysfunction after Exogenous Administration of Intravenous Adrenaline". *Therapeutic Advances in Cardiology* 1.1 (2017): 12-14.

be ruled out, like the production of diffuse vasospasm [6]. In the majority of these cases the ventricular dysfunction have good response to beta blockers, as well as in our patient [7]

To sum up, the novelty of this clinical case is that no other global ventricular dysfunction has been reported after the administration of intravenous catecholamines in a complete healthy heart, and helps to understand the pathophysiology of other illnesses (Tako-Tsubo Syndrome, Ventricular dysfunction in pheochromocytoma...) as well as to determine the usefulness of beta blockers in such a cases, and the possible adverse effects of exogenous adrenaline in patients with previous myocardialopathy.

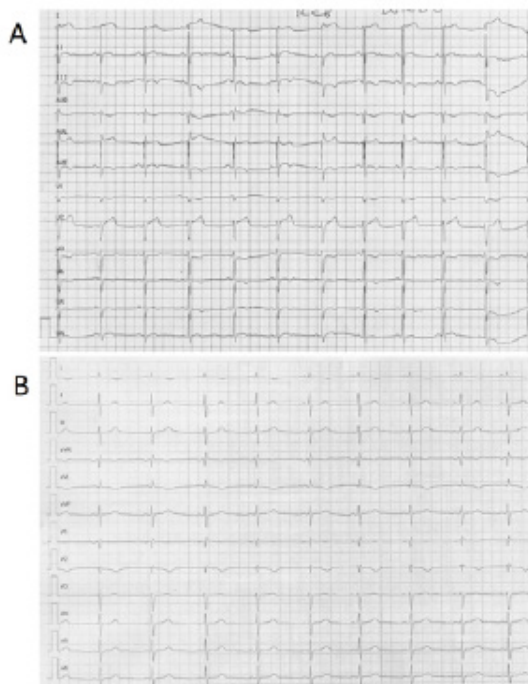


Figure 1A: Electrocardiograma tras la administración de adrenalina intravenosa.

Figure 1B: Electrocardiograma de control.

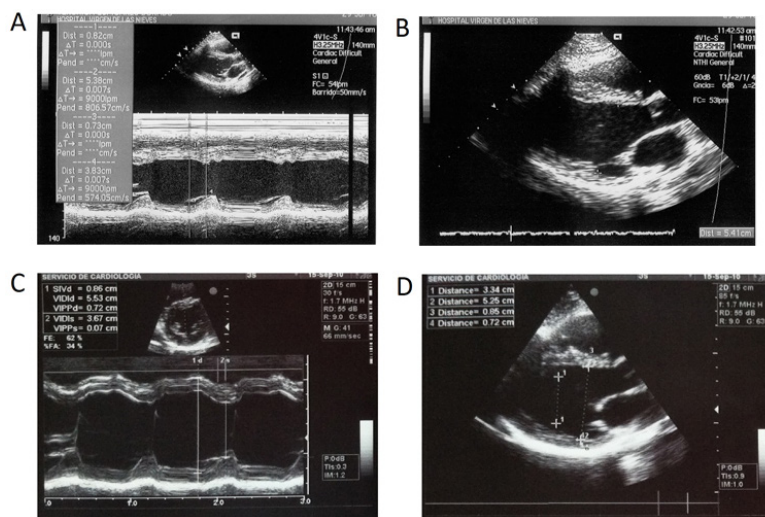


Figure 2: Ecocardiograma al ingreso en 2 dimensiones (A) y modo M (B).

Ecocardiograma de control a los 6 meses en 2 dimensiones (A) y modo M (B)

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