CASE REPORT

Nicks procedure for critical aortic stenosis presenting acute myocardial infarction and normal coronary arteries

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Aortic stenosis is associated with an increased risk of death from cardiovascular events. Nevertheless, acute myocardial infarction in patients with severe aortic stenosis and normal coronary arteries is very rare. We report a case of acute myocardial infarction occurring in a patient with severe aortic stenosis and left ventricular hypertrophy. A coronary angiogram performed during the acute phase of evolving myocardial infarction excluded coronary obstruction as the cause of acute myocardial infarction. This case was successfully treated by aortic root enlargement (Nicks procedure) to alleviate the severe aortic stenosis.

Key words: **Aortic Stenosis; Acute myocardial infarction, normal coronary arteries; Nicks procedure; Cardiac Surgery, surgical procedures.**

La estenosis aórtica se asocia con un mayor riesgo de muerte por eventos cardiovasculares. Sin embargo, el infarto agudo de miocardio en pacientes con estenosis aórtica grave y arterias coronarias normales es muy raro. Presentamos un caso de infarto agudo del miocardio en un paciente con estenosis aórtica critica e hipertrofia ventricular izquierda. La angiografía coronaria realizada durante la fase aguda del infarto en evolución excluyó la obstrucción coronaria como causa del infarto agudo de miocardio. Este caso fue tratado exitosamente tratado mediante alargamiento de la raíz aórtica (Procedimiento de Nicks) para aliviar la estenosis aórtica.

***Palabras clave*: Estenosis aórtica; Infarto agudo del miocardio, arterias coronarias normales; Procedimiento de Nicks; Cirugía cardiaca, procedimientos quirúrgicos.**

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Aortic stenosis (AS) is the most common primary valve disease leading to surgery or catheter intervention in Europe and North America, with a growing prevalence due to the ageing population [1]. In patients with AS, the development of left ventricular systolic dysfunction and heart failure predicts poor prognosis, including a less favorable outcome after valve replacement [2].

Severe AS may produce angina pectoris in of coronary artery stenosis, as a result of both decreased coronary reserve and aortic transvalvular pressure gradient, which favors imbalance between myocardial oxygen supply and demand. Heart failure is preceded by structural and functional alterations in the heart muscle with left ventricular hypertrophy followed by degeneration and death of the cardiac myocytes [3].

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Serum cardiac troponins I and T are heart specific contraction-regulating proteins released into the circulation from injured myocytes. Their value in acute coronary syndrome is well known and they are assuming a growing prognostic role in heart failure as well [4]. Studies have shown that at least 20% of patients with severe AS have detectable serum troponins. Critical AS (defined as valve area <0.5 mm^2) has been reported to cause modest elevations in cardiac biomarkers such as troponin I [2].

This case report describes a patient who presented as suspected acute coronary syndrome with markedly elevated troponin levels, who was later found to have normal coronaries and critical AS.

CLINICAL CASE

A 60-year-old male with a medical history of hypertension, type II diabetes mellitus, obesity (BMI 38.1) and moderate aortic stenosis with left ventricular ejection fraction

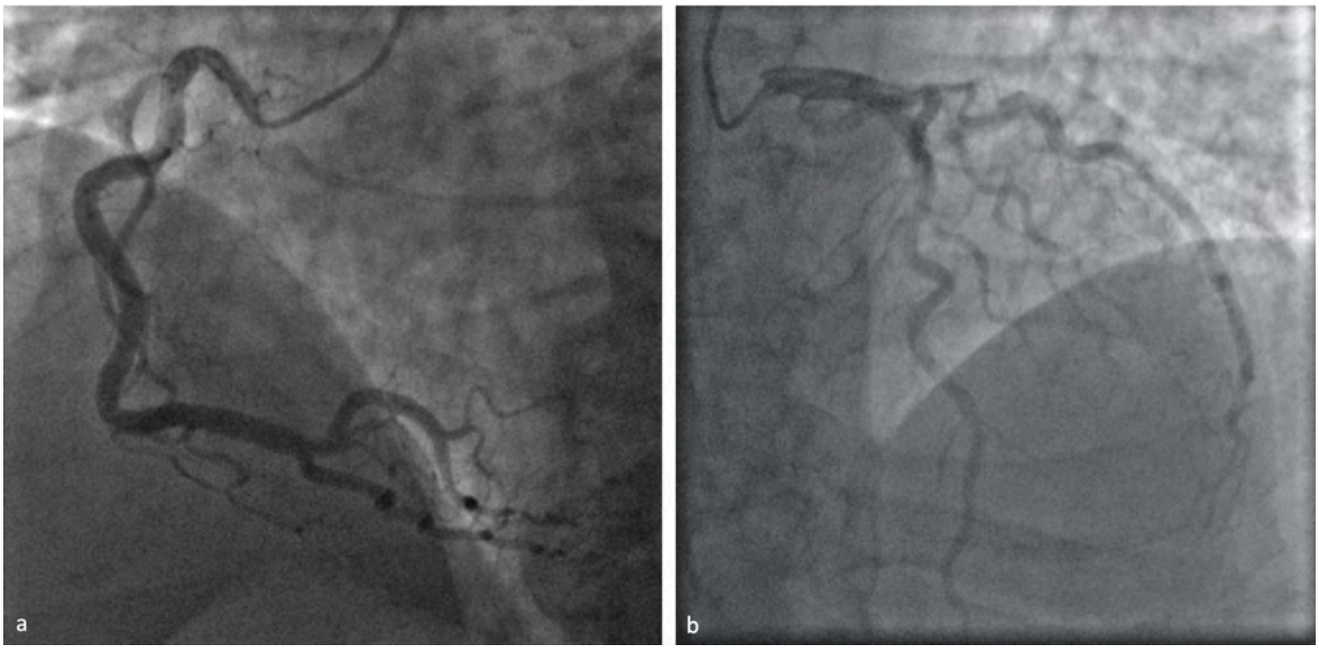

Figure 1. Selective coronary angiography showing (A) nonsignificant obstructive lesions of patent right coronary artery and (B) left anterior descending and circumflex artery.

(LVEF) of 60% diagnosed a year ago; presented to our emergency department with complaints of chest discomfort and heaviness for 2 hours. On arrival at our emergency department, his electrocardiogram revealed regular sinus rhythm with ST-segment depression in lead II, III, aVF and V2-6; biochemical data of cardiac enzymes were creatine kinase (CK) 368U/L; CK-MB 76U/L; troponin-I 5.96ng/mL. Coronary angiography was performed by an interventional cardiologist and revealed normal coronary arteries with nonsignificant obstructive lesions (**Fig. 1**). Echocardiography showed critical AS with estimated valve area 0.36cm2 and low LVEF (22%),indexed left ventricle mass 152g/m2. He was admitted to our intensive care unit for 5 days. We reevaluate echocardiogram improving left ventricular function to 40%. One week after myocardial infraction surgery was performed.

Operation was performed as open chest surgery through median sternotomy on cardiopulmonary bypass using a single two-stage venous cannula. Once on cardiopulmonary bypass, the aorta was crossclamped and anterograde Nido cardioplegia through the ascending aorta was administered. An anterior transverse aortotomy is made and the incision is extended obliquely toward the noncoronary sinus. The aortic valve leaflets are resected and annular calcium debrided (**Fig. 2**). Aortic valve sizer was 21mm so we decide to made an aortic root enlargement extending the aortotomy across the aortic annulus in the mid-portion of the noncoronary sinus and into the fibrous subaortic curtain (Nicks technique) [5]. Dacron graft was used to reconstruct the aortic defect; biologic aortic valve was implanted (25mm Edwards Inspiris Resilia aortic valve) with pledget–reinforced 2-0 ethibond sutures. (**Fig. 3**) (**Fig.4**). The rest of the operation was made as usual. extracorporeal circulation time of 178 minutes and aortic cross-clamping time of 140 minutes. Surgical findingswere as follows: aortic valve severely calcified, destructured with merged commissures, aortic root of 21mm, aortic root after root enlargement of 25mm.

The postoperative course in intensive care unit was uneventful, with a lenght of stay of 2 days. Hospital discharge was 5 days after surgery. An echocardiogram control was performed 1 month later, reported: LVEF 52%, without paravalvular leaks and transvalvular mean gradient of 4mmHg.

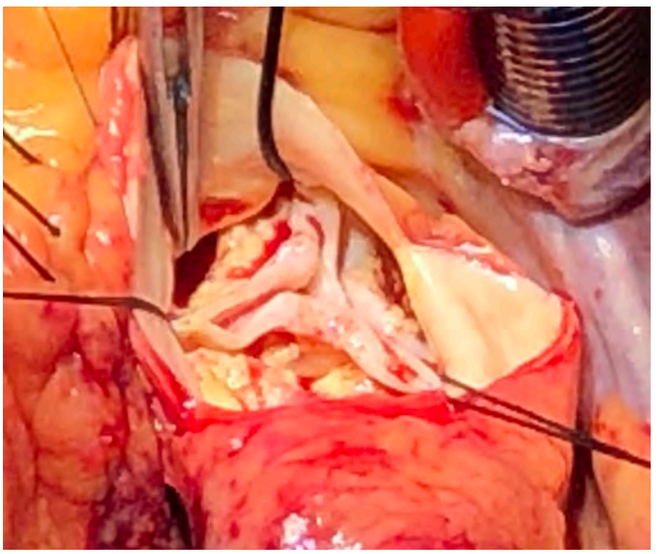

Figure 2. Surgical view: Calcified native aortic valve.

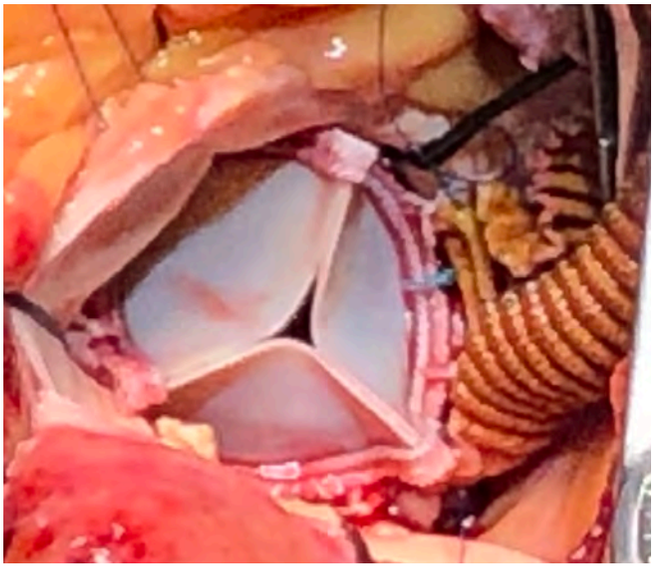

Figure 3. Surgical view: Nicks procedure with Dacron graft and biological aortic valve implanted.

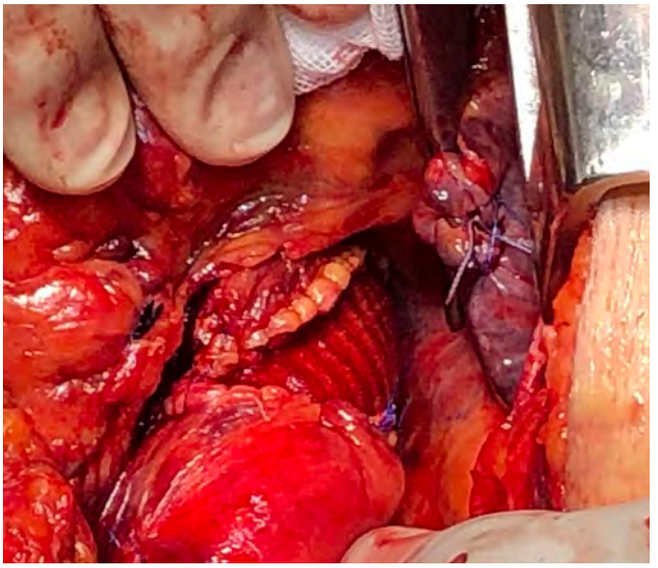

Figure 4. Final aspect of the operative field, with Dacron graft in its final positions

COMMENT

AS affects 2–9% of the population over 65 years and increases with age. The risk factors for AS are similar to those of atherosclerosis (age, male sex, smoking, hypertension and elevated LDL) [6]. Angina pectoris is the most frequent symptom in AS and is present in 30 to 40% of patients with normal coronary arteries, the association of acute myocardial infarction and normal coronary anatomy in patients with severe AS is unusual [7].

AS, even in its early stages, may be associated with the pathogenesis of acute coronary syndromes. About 20-50% of subjects with AS and angina pectoris have significantly obstructive coronary arteries disease. On the other hand, the overall rate of myocrdial infarction in patients with normal coronary angiography varies from 1% to 12% [8]. Platelet hyperaggregation, activation of blood coagulation, coronary microcirculatory dysfunction, imbalance of the supply and demand of oxygen in the hypertrophied myocardium and subendocardial ischemia predisposed by AS are possible mechanisms to explain this serious event. However, it is still uncertain whether antiplatelet or anticoagulant therapy is effective in preventing thrombin generation and platelet activation predisposed by AS [8].

Lumley et al. [5] showed that the underlying mechanism for ischemia in patients with severe AS is due to an abnormal cardiac coronary coupling: the inability to increase blood flow in proportion to the cardiac workload results in ischemia [6]. This case highlights the importance of the clinical examination correlated with the research focused next to the bed, such as echocardiography, in the treatment of patients presenting with chest pain.

Miyagawa et al., used echocardiography to assess the deterioration of blood flow in the hypertrophied myocardium of patients with AS, myocardial blood flow in the subendocardium was significantly lower than in normal individuals, while there was no significant difference in subepicardial blood flow. The decrease in subendocardial blood flow involves microcirculatory myocardial dysfunction and a reduced coronary vasodilatory reserve. These abnormalities may contribute to the induction of subendocardial ischemia in patients with AS who have normal epicardial coronary arteries [8].

In patients with severe AS and AMI with normal coronary arteries, the ST segment ascent may be due to total obstruction of blood flow in an epicardial coronary artery caused by coronary thrombosis with subsequent recanalization, to a persistent coronary spasm, to a Pedunculated calcified mass that intermittently occludes the ostium of the left coronary artery from the aortic valve or an acute endocarditis with abscess in the aortic root that compresses an epicardial artery [7]. In the current case, the mechanism involved was most likely a disproportion in oxygen demand, since our patient didn't show ST segment elevation.

Serum cardiac troponins I and T are reliable and highly specific markers of myocardial injury. Studies have shown that at least 20% of patients with severe aortic stenosis have detectable serum troponins [2]. In our case the cardiac enzymes were creatine kinase (CK) 368U/L; CK-MB 76U/L; troponin-I 5.96ng/mL.

Myocardial ischemia commonly occurs in patients with severe aortic stenosis during hemodynamic stress, even in the context of angiographically documented normal coronary arteries. The responsible mechanism is the myocardial oxygen demand that exceeds the supply [9]. Tachycardia associated with shortening of the diastolic perfusion time in combination with the hypertrophy of the left ventricle, with the affectation of the relaxation of the left ventricle and with a high tension of the diastole wall that delays the rapid rise of the diastolic perfusion of the endocardium after of systolic com-

pression explain the mechanism of AMI. Therefore, in the AMI associated with severe AS, the appearance of tachycardia should be avoided [2,3]. When the infarcts are located in the circumferential subendocardium, they are believed to be caused only by AS [9].

There have only been a few documented cases of acute myocardial infarction caused only by severe aortic stenosis, although silent ischemic necrosis has often been observed [9]. Our patient had marked left ventricular hypertrophy secondary to severe AS and normal coronary arteriography, the acute phase of myocardial infarction involving the circumferential subendocardial wall of the left ventricle was clearly documented by detecting a transient increase in biochemical markers of myocardial necrosis and EKG without ST elevation.

The left ventricular endocardium usually shortens more than the epicardium and, consequently, has a higher oxygen consumption. The myocardial oxygen demand is increased by the increase in the mass and thickness of the left ventricular wall secondary to severe aortic stenosis [9]. In addition, it should be borne in mind that this association involves a higher rate of cardiac rupture due to the hyperpression in the left ventricle, a risk that must be closely monitored to detect this serious complication [3].

This case is interesting because it is a documented case of acute myocardial infarction that involves the circumferential subendocardial wall of the left ventricle in a patient with severe AS, marked left ventricular hypertrophy and angiographically normal coronary arteries. The infarction was caused by severe AS and the result of the extreme disparity between the supply and demand of myocardial oxygen.

Reduced subendocardial blood flow and microcirculatory dysfunction were recovered significantly after the surgical replacement of the aortic valve demonstrated both echocardiographically and in the improvement of the patient's functional class one month after the surgery, demonstrating the great benefit of aortic valve replacement in this pathology.

By using the largest possible prosthesis makes possible to reach very low transprosthetic gradients in the postoperative. In our case here, a Nicks procedure was very useful for this purpose by enlarging the aortic annulus while placing a 25 mm biological prosthesis.

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REFERENCES

1. Baumgartner H, Falk V, Bax JJ, et al. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J 2017; 38(36):2739-91.
2. Wayangankar SA, Dasari TW, Lozano PM, Beckman KJ. A case of critical aortic stenosis masquerading as acute coronary syndrome. Cardiol Res Pract (2010). doi: 10.4061/2010/423465.
3. Jondeau G, Dubourg O, Partovian C, et al. Acute myocardial infarction in a patient with severe aortic stenosis and normal coronary arteries. Eur Heart J 1994;15(5): 715-7.
4. Sato Y, Kita T, Takatsu Y, Kimura T. Biochemical markers of myocyte injury in heart failure. Heart 2004; 90(10): 1110-3.
5. Grubb KJ. Aortic root enlargement during aortic valve replacement: Nicks and Manouguian techniques. Oper Tech Thorac Cardiovasc Surg 2016; 20(3): 206-18.
6. Gue YX, Bhandari SS, Kelly DJ. Critical aortic stenosis presenting as STEMI. Echo Res Pract. 2017;4(3): K7-10. doi:10.1530/ERP-17-0017.
7. Ambrós Checa A, Gómez Grande LM, Ortíz Ferrán R, Ortega Carnicer J. Estenosis aórtica grave e infarto agudo de miocardio con arterias coronarias normales. Emergencias 2000; 12:127-9.
8. Lin CF, Chu KC. Acute myocardial infarction in an elderly patient with severe aortic stenosis and angiographically normal coronary arteries. Int J Gerontol 2010; 4(3): 157-60.
9. Kodama-Takahashi K, Ohshima K, Kurata A, et al. Myocardial infarction in a patient with severe aortic stenosis and normal coronary arteriograms involvement of the circumferential subendocardial wall of the left ventricle. Circ J 2003; 67: 891-4.