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Case Report

Dunning type III aortocoronary dissection after percutaneous coronary intervention for chronic total occlusion. A case report

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ABSTRACT

Iatrogenic aorto-coronary dissection is an intimal tear that creates a false lumen and may propagate into the aorta. We report the case of a 68-year-old woman with a history of hypertension, obesity, a previous unrevascularised inferior transmural myocardial infarction, and limiting angina. Myocardial perfusion imaging demonstrated 15% ischaemia of the inferior wall, and coronary angiography revealed a chronic total occlusion of the right coronary artery with heterocoronary collateral circulation. The patient underwent percutaneous coronary intervention with implantation of three drug-eluting stents. A dissection flap originating in the right coronary sinus with extension into the aortic root was observed; contrast-enhanced computed tomography confirmed dissection of the ascending aorta with extension to the aortic arch and the brachiocephalic trunk. Medical therapy (beta-blockers, nitrates, and analgesia) was initiated, and surgery was deferred with close clinical and imaging surveillance. The clinical course was favourable, with a stable intramural haematoma of the ascending aorta and complete resorption at 7 months of follow-up.

Keywords: Aortic Dissection; Coronary Angiography; Iatrogenic Diseases (Source: MeSH-NLM).

RESUMEN

Disección aortocoronaria Dunning clase III luego de intervención percutánea de oclusión coronaria total crónica. Reporte de un caso

La disección aortocoronaria iatrogénica es un desgarro intimal que genera una falsa luz y puede propagarse hacia la aorta. Presentamos el caso de una mujer de 68 años, con antecedentes de hipertensión arterial, obesidad, infarto de miocardio ST elevado inferior previo no revascularizado y angina limitante. La perfusión miocárdica evidenció isquemia del 15% en la pared inferior, y la coronariografía reveló oclusión total crónica de la arteria coronaria derecha, con circulación colateral heterocoronariana. Se realizó angioplastia e implante de tres stents medicados. Se evidenció un flap de disección originado en el seno coronariano derecho, con propagación hacia la raíz aórtica. La angiotomografía confirmó la disección de la aorta ascendente, con extensión al arco aórtico y al tronco braquiocefálico. Se instauró tratamiento médico (betabloqueantes, nitratos y analgesia), con conducta expectante quirúrgica ante una posible progresión. La evolución fue favorable, con evidencia de hematoma intramural estable en la aorta ascendente al alta y reabsorción completa al séptimo mes de seguimiento.

Palabras clave: Disección Aórtica; Angiografía Coronaria; Enfermedades Iatrogénicas (Fuente: DeCS-BIREME).

Introduction

Iatrogenic aortocoronary dissection (IACD) is defined as the creation of a false lumen in the aortic wall, caused by an intimal tear that extends from the coronary arteries into the aorta [1]. It occurs during diagnostic or therapeutic coronary angiography, with an incidence of 0.006% and 0.098%, respectively, and carries a mortality rate of 5-10% [2,3]. The Dunning classification divides it into three groups: Class I: involvement of the ipsilateral aortic cusp. Class II: extension into the ascending aorta <40 mm. Class III: extension into the ascending aorta >40 mm [4,5]. We present the case of a patient who underwent elective percutaneous coronary intervention for chronic total occlusion (CTO), in whom, despite being classified as Dunning class III, medical management was performed.

Case report

MA 68-year-old woman with a history of obesity, hypertension, and a prior inferior ST-elevation myocardial infarction one year earlier, without revascularisation, presented with typical angina despite optimal medical therapy with a beta-blocker, calcium channel blocker, and nitrates at maximally tolerated doses. Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction (LVEF) of 58% with basal inferior hypokinesia. Myocardial perfusion imaging demonstrated 15% residual ischaemia in the inferior wall, and diagnostic coronary angiography was performed, revealing CTO of the right coronary artery (RCA); percutaneous intervention was therefore planned. Via a radial approach, an Amplatz Left 2 (AL2) and a Judkins Right 4 (JR4) 6 Fr catheter were advanced, along with 0.014 angioplasty guidewires (Fighter and Whisper Extra Support). Percutaneous coronary intervention was then performed with implantation of three drug-eluting stents in the proximal and mid segments of the RCA, using balloon dilation with manual inflation up to 12 atm. During angiographic control with manual injection, contrast extravasation into the wall of the ascending aorta was observed, establishing the diagnosis of IACD **(Figure 1)**.

Simultaneously, the patient developed intense chest pain, tachycardia, and hypertension; the electrocardiogram showed T-wave inversion in leads V1-V4. Haemodynamic control was initiated with labetalol infusion and analgesia with morphine, achieving a heart rate of 60 beats per minute and systolic blood pressure <120 mmHg; dual antiplatelet

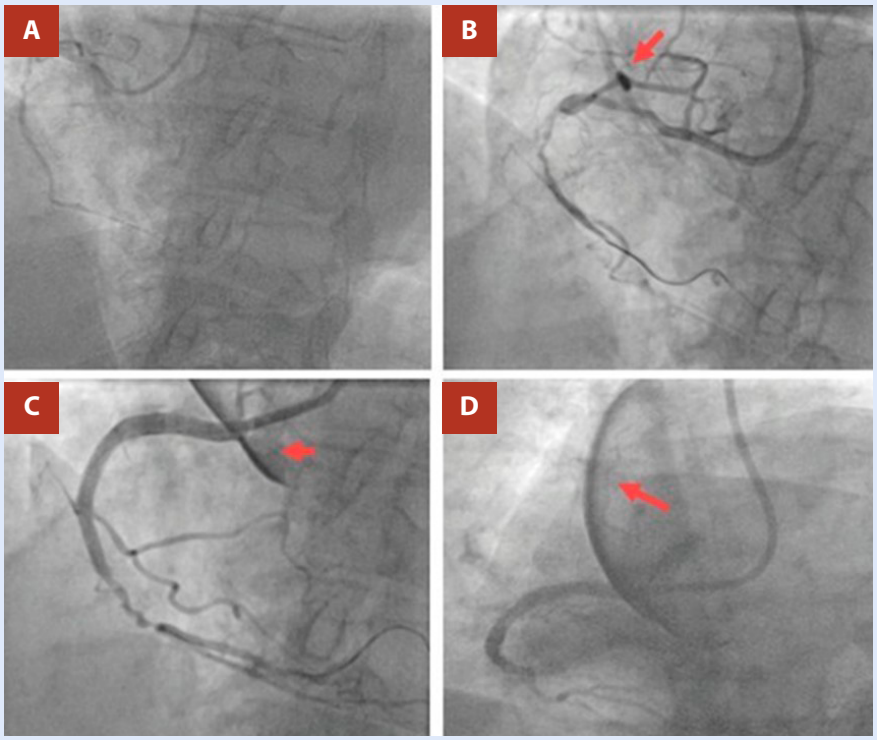

Figure 1. A. Occlusion in the mid segment of the right coronary artery. **B.** Advancement of the guidewire to the distal segment of the right coronary artery, demonstrating contrast extravasation at the level of the right coronary sinus extending to the aortic root. **C.** Angioplasty with three drug-eluting stents in the proximal segment of the right coronary artery, showing retrograde increase in contrast extravasation towards the ascending aorta. **D.** Dissection extending into the ascending aorta >40 mm.

therapy was temporarily withheld. Computed tomography angiography (CTA) of the aorta and great vessels revealed an ascending aortic dissection originating from the right and non-coronary sinuses of Valsalva, extending to the aortic arch and terminating before the origin of the left subclavian artery. The total diameter of the ascending aorta was 41.4 x 39.5 mm (21.5 mm/m^2), with a true lumen diameter of 27 x 14.6 mm. The dissected intimal flap involved approximately 50% of the true lumen, and the right coronary artery originated from the false lumen, findings consistent with dissection secondary to iatrogenic intimal trauma following initial catheter engagement and stent implantation. The case was classified as Dunning class III IACD **(Figure 2)**.

A conservative medical management strategy was adopted, with continuous monitoring and multimodal imaging follow-up. This decision was based on clinical stability, resolution of chest pain, and absence of signs of hypoperfusion or complications such as cardiac tamponade, severe aortic regurgitation, or progression of the dissection. During hospitalisation, strict control of heart rate and blood pressure was maintained, initially with labetalol and subsequently with propranolol, nitroglycerin, and morphine. CTA of the aorta and great vessels on days 2 and 6 showed formation of a stable intramural haematoma, without progression of the dissection or increase in aortic diameters. On day 3, transoesophageal echocardiography excluded severe aortic valve insufficiency and confirmed a stable intramural aortic haematoma.

On day 3, dual antiplatelet therapy was gradually reintroduced, and oral treatment was initiated with irbesartan 150 mg twice daily, amlodipine 10 mg once daily, and propranolol 40 mg every 8 hours. The patient remained stable, without complications, and was discharged on day 9.

At 3- and 6-month follow-up, the patient remained asymptomatic. CTA of the aorta and great vessels performed at 7 months demonstrated complete resolution of the intramural aortic haematoma and reduction of the ascending aortic diameter to 33 x 34 mm **(Figure 3)**.

Discussion

Ascending aortic dissection is a cardiovascular emergency, with a mortality of 22% in patients undergoing surgery and 58.6% in those managed medically [6]. Therefore, management is usually surgical and, although most cases are spontaneous, cases following interventional procedures have been reported, with IACD being one of the most feared complications of coronary angiography [7].

Risk factors include excessive catheter manipulation, high-pressure contrast injection, significant atherosclerosis, uncontrolled hypertension, structural abnormalities of the aorta or coronary arteries, and percutaneous intervention for CTO [8,9]. Our patient was hypertensive, had significant atherosclerosis, and underwent PCI for RCA CTO.

In more than 85% of cases, the ostium of the RCA is involved, as in our case. This predisposition is explained by the fact that the left main coronary artery (LMCA) offers greater resistance to traction and pressure than the RCA [9,10]. This difference is related to the larger diameter of the LMCA, less pronounced angulation, a wall richer in type I collagen, and the greater technical difficulty of cannulating the RCA [10,11].

A high proportion of cases occur during PCI for CTO, as in the present case, likely due to increased catheter manipulation; the most frequently associated catheters are JR4 and AL1 6 Fr [12]. In this case, both catheter types were used.

Diagnosis can be established during angiography by identifying contrast extravasation from the sinus of Valsalva

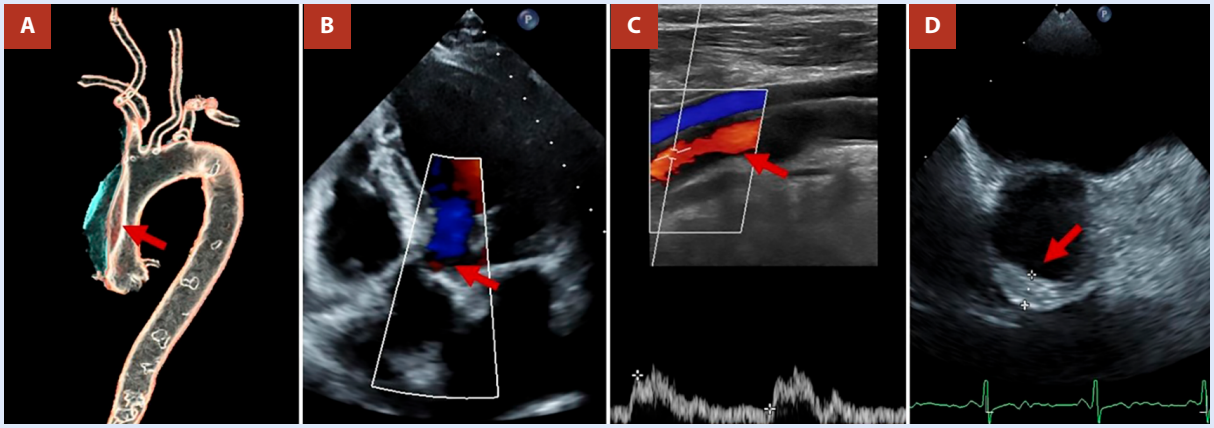

Figure 2. A. Three-dimensional reconstruction of aortic computed tomography angiography (CTA), showing the ascending aortic dissection (red arrow) terminating before the origin of the left subclavian artery. **B.** Transthoracic echocardiography demonstrating absence of significant aortic valve disease and no evidence of pericardial effusion. **C.** Carotid ultrasound showing no impairment of flow in the right carotid artery. **D.** Transoesophageal echocardiography demonstrating an intramural haematoma in the ascending aorta, without signs of progression of the dissection.

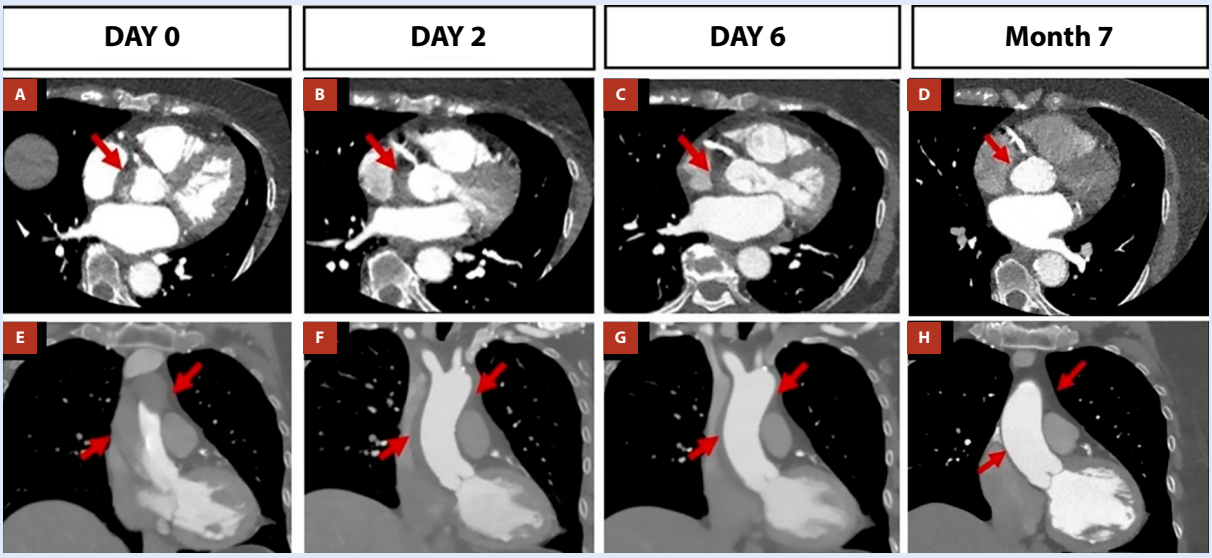

Figure 3. (A to C) Dissection originating from the right coronary sinus, without increase in diameter on day 6. **(E to G)**. Dissection extending from the aortic root to the aorta, reaching just before the origin of the left subclavian artery on day 0, with reduction in extension and thickness and appearance of residual haematoma on day 6. **(D and H)**. Tomographic follow-up at 7 months showing complete resolution of the intramural aortic haematoma, without evidence of wall thickening or dissection flap.

and partial collapse of the true lumen. When suspected, all contrast injection should be stopped, and further catheter manipulation avoided [13]. When the entry point is identifiable, the preferred strategy is ostial sealing with a stent to prevent propagation; in our case, despite early recognition of IACD, the entry tear could not be localised, and therefore haemodynamic stabilisation and multimodal imaging were pursued [13,14].

TTE allows detection of pericardial effusion, aortic regurgitation, and regional wall motion abnormalities suggestive of coronary hypoperfusion due to true lumen collapse, findings that would indicate emergency surgery [14]. None of these complications were observed.

CTA of the aorta and great vessels is the diagnostic modality of choice due to its high accuracy (sensitivity 98-100% and specificity 95-99%); it allows identification of the origin, measurement of extension, and assessment of involved vascular territories. The protocol includes ECG-gated acquisition of the ascending aorta followed by a scan from the neck to the pelvis [15]. The lesion was classified as Dunning class III; serial imaging follow-up demonstrated evolution to a stable intramural haematoma, supporting continuation of conservative management.

The goal of treatment is to reduce aortic wall stress without compromising systemic perfusion. A heart rate ≤60 bpm and systolic blood pressure <120 mmHg are recommended. Adequate analgesia is essential, and reflex tachycardia should be avoided; therefore, vasodilators should be introduced after beta-blockade [15,16]. This was the strategy applied, with intravenous labetalol during the initial phase.

Regarding definitive treatment, in limited forms (Dunning I-II), ostial sealing combined with medical management is usually sufficient [17,18]. Traditionally, Dunning III cases were considered to require surgical management [7]; however, decision-making should not rely solely on extension. We propose, for Dunning III, an approach based on four criteria: effective sealing of the entry tear; absence of moderate-to-severe aortic regurgitation or cardiac tamponade; haemodynamic stability without signs of hypoperfusion; and no progression on CTA of the aorta and great vessels within the first 24-72 hours. If these criteria are met, intensive conservative management with close monitoring is reasonable; otherwise, emergency surgery should be prioritised. In our patient, the absence of complications and lack of progression on serial CTA supported conservative management. Intravascular ultrasound (IVUS) may be useful for identifying the origin of the dissection flap, facilitating ostial sealing with a stent [19]. Although it was not used in this case due to prioritisation of haemodynamic stabilisation, it represents a valuable diagnostic alternative. The largest case series reported to date found that 54% of patients had Dunning class III IACD, of whom only 40% underwent surgery, indicating that not all cases require surgical management, in line with our findings [1].

Follow-up should include TTE to assess the aortic valve and detect pericardial effusion, as well as CTA of the aorta and great vessels to evaluate progression or resolution within 4-6 weeks after discharge [20]. In our patient, no valvular involvement or pericardial effusion was observed, and there was no progression of the dissection, with complete resolution of the residual intramural aortic haematoma at 7 months of follow-up.

In conclusion, in our patient with Dunning type III IACD without haemodynamic instability or associated complications, conservative management was a safe option. Therapeutic decisions should be individualised according to clinical and imaging evolution.

Ethical aspects

Written informed consent was obtained from the patient. In addition, the report was approved by the institutional ethics committee.

Author contributions

AVB, ACA, DDF, ZRU: Conceptualisation, investigation, original draft writing, and manuscript review and editing.

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