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Long-term follow-up after successful multi-stage intravascular repair of acute aortic dissection associated with cocaine exposure

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We report a 16-year follow-up (2009–2026) of a male patient who developed cocaine-related Stanford type B aortic dissection (AD) at the age of 36 years. He was treated in one clinical centre with multi-stage endovascular interventions, leading to the complete resolution of AD complications.

In 2009, during extensive downhill skiing the patient reported tearing chest pain, followed by syncope. He was immediately transferred to a local emergency unit, where markedly elevated

blood pressure was noted on initial presentation, although the exact value was not available in the source documentation. Initial management included sedation and blood pressure (BP) control. Computed tomography angiography (CTA) showed a Stanford type B AD involving the abdominal aorta (AA), and both renal and iliac arteries.

The patient was a smoker with a history of untreated hypertension and reported BP values of up to 160/100 mmHg. Otherwise, his medical history was unremarkable. He did not take any medications, but had a known history of cocaine use and, by self-report, had taken intranasal cocaine about 2 hours prior to the event mentioned above.

The patient was then transferred by air ambulance to a tertiary vascular surgery unit in Warsaw, Poland. At initial presentation, he was considered to be at increased risk of morbidity and mortality and required emergency intervention. Features of complicated AD included a history of uncontrolled hypertension, intractable pain, branch artery occlusion, and distal extension of the dissection [1].

He underwent immediate thoracic endovascular aortic repair. CTA (day 7) demonstrated stable appearance of the stent-graft (Figure 1A). The celiac trunk, superior mesenteric artery and left renal artery (LRA) were supplied from the true lumen (TL). The right renal artery (RRA) and inferior mesenteric artery were supplied from the false lumen (FL).

After the acute event, annual follow-up visits were carried out at our institution (Figure 1B). After 2 years, CTA showed widening of the AA. Therefore, the patient was re-admitted in 2011 and a balloon-expandable covered stent was implanted to bridge the RRA to the TL (Figure 1C). The post-interventional course was uneventful.

In 2013, CTA showed progression of AA diameter due to retrograde filling of the FL through re-entry tears located in the common iliac artery (CIA) and external iliac artery (EIA). Stent-grafts were implanted in the right CIA and EIA, successfully sealing re-entry tear at the origin of right internal iliac artery (IIA) (Figure 1D). However, the procedure using two

peripheral stent-grafts to close the entry tears in the left EIA was only partially successful (Figure 1E). In December 2025, complex repair was performed using a branched stent-graft with a limb to the left IIA and a follow-up CTA showed complete sealing of the re-entry tears (Figure 1F).

Currently, the patient is taking a beta-blocker, an angiotensin receptor blocker, a calcium channel blocker, a thiazide-like diuretic, dual antiplatelet therapy, and a statin. With antihypertensive therapy, his systolic and diastolic blood pressure values are maintained within the ranges of 120–130 mmHg and 70–80 mmHg, respectively. This is in accordance with the current guidelines of the European Society of Hypertension, which recommend chronic blood pressure lowering to <130/80 mmHg in patients after AD [2]. The patient has good exercise tolerance, as evidenced by activities such as scuba diving to depths of up to 40 meters, skiing, and motocross; he maintains good functional status, works full-time, and reports very good quality of life, with no significant pain or fatigue. He denied any further use of cocaine or any other drugs. During 16 years of follow-up, no other cardiovascular events occurred.

Cocaine, a commonly abused drug, exerts adverse effects on the cardiovascular system that can result in a variety of acute cardiovascular complications, including stroke, myocardial infarction, and sudden cardiac death [3,4]. Cocaine has also been postulated to be one of the risk factors for dissections of arteries, including AD, which is uncommon and has been described in a few case reports [3,5].

The International Registry of Acute Aortic Dissection (IRAD), which collected data on acute AD from 17 international centres, reported a prevalence of cocaine-related AD as low as 0.5% [5]. In contrast, a single-centre study in the United States reported that AD associated with cocaine use accounted for 37% of AD cases [5]. This discrepancy may be explained by differences in the enrolled populations across these studies. It has also been reported that,

compared with patients with AD unrelated to cocaine use, patients with cocaine-related AD were predominantly male, younger, and more frequently had a history of untreated hypertension [5].

Mechanisms leading to AD are not well understood; however, various studies have shown a temporal association of cocaine intake and AD, which appears to occur more frequently in younger individuals [3].

The pathophysiology of cocaine-induced acute AD is multifactorial. Acute exposure increases heart rate and BP via sympathetic activation and enhanced myocardial contractility, thereby increasing shear stress on the aortic wall [3,4]. Cocaine has also been shown to induce vascular smooth muscle cell apoptosis and cystic medial necrosis with consequent vessel wall weakening [4]. Therefore, a high index of suspicion should be maintained, particularly in young patients presenting with acute chest pain who report recent cocaine use [5].

This case report also highlights the importance of careful, individualized long-term follow-up in patients with AD and illustrates stepwise challenges and therapeutic possibilities currently available through modern imaging and endovascular repair techniques.

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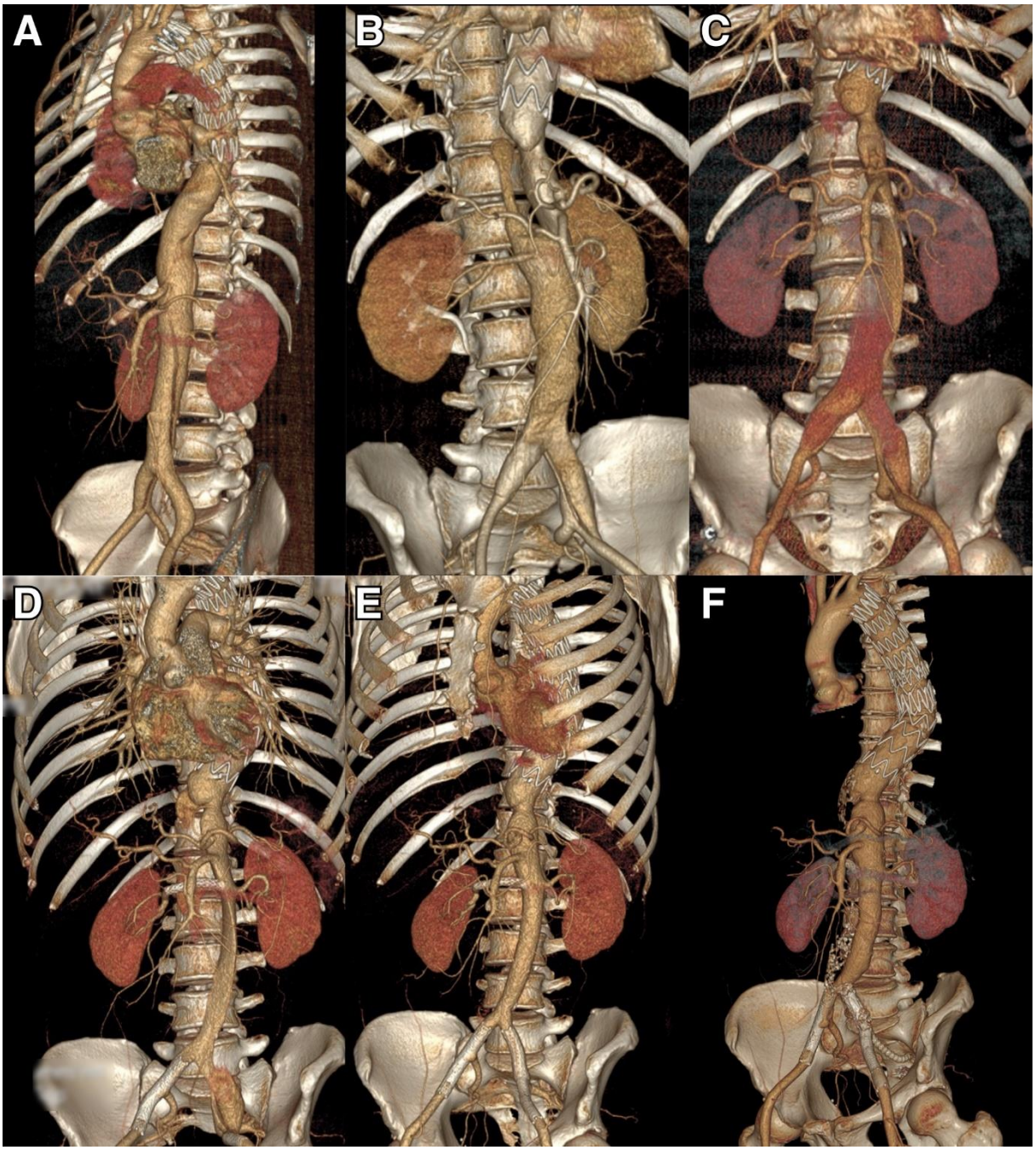

Figure 1

A. Stent-graft covering the primary entry tear (a large abdominal FL persists, with residual inflow via the primary entry tear and secondary re-entry tears at the level of the LRA and both iliac arteries) (2009).

B. Implantation of the additional stent-graft to reinforce coverage of the primary entry tear (2010).

C. Stent-graft implanted in the RRA to reduce FL inflow via secondary re-entry tears, successfully securing renal perfusion from the TL (2011).

D. Persistent FL inflow via a secondary re-entry tear in the left EIA. The aortic diameter is slightly larger than in 2012 (2013).

E. Additional stent coverage of secondary re-entry tears in the left iliac artery (2015).

F. Branched stent-graft with a limb to the left IIA, showing complete sealing of re-entry tears in control (2025)

Short title: Post-cocaine aortic dissection: treatment and follow-up