Protracted Refractory Pain Post TEVAR: Post Implantation Syndrome?

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Received date: July 14, 2016; Accepted date: August 19, 2016; Published date: August 27, 2016

Abstract

Aortic dissection is a life-threatening condition and has one of the highest mortality rates of cardiovascular diseases. It remains a devastating disease; with multiple unanswered questions concerning treatment modalities. The role of thoracic endovascular aortic repair (TEVAR) in these patients; especially those with uncomplicated acute Type B dissections (AAD-B) is especially controversial although it has been shown to have better long term outcomes compared to medical therapy alone. For those who have TEVAR, up to 60% may develop an acute, largely transient systemic inflammatory response syndrome (SIRS) that remains vaguely defined and explained, and of unclear significance. Defined as a non-infectious fever and leukocytosis post procedure; the role of local inflammation in clinical management of this post-implantation syndrome has not been highlighted. We present a case of a 57 year old male patient who presented with hypertensive emergency and was found to have an acute uncomplicated Type B aortic dissection managed with TEVAR, and the subsequent long standing refractory pains that developed thereafter, only later managed as an ‘atypical’ post implantation syndrome.

Keywords: TEVAR; Post-implantation syndrome; Local inflammation; Pain; Steroids

Introduction

Aortic dissection is a life-threatening condition affecting up to 30 people per million each year [1]. It has one of the highest mortality rates of the cardiovascular diseases [2]; and has remained a devastating disease; with multiple unanswered questions concerning treatment modalities. The role of endovascular repair in patients with uncomplicated acute Type B dissections (AAD-B) is especially controversial [3] and its associated post implantation syndrome still vague [4]. We present a case of an acute uncomplicated Type B aortic dissection in a 57-year-old man managed with TEVAR in our hospital; and the subsequent long standing refractory pains that developed thereafter, only later managed with steroids. Although most definitions do not include local features of inflammation (including pain) as part of post implantation syndrome (PIS); its place in SIRS and PIS is highlighted.

Case Report

A 57 year old man of African descent known to have systemic hypertension; presented to our facility with chest pain radiating to the back. This was of acute onset, progressively worsening, of severity >8 according to the numeric pain rating scale; and associated nausea and vomiting. The patient had no other co-morbidities; was a non-smoker, and didn't volunteer any history of alcohol use. He had no history of other drugs' use. He was on metoprolol, irbesartan and hydrochlorothiazide; which he admitted to using only occasionally when he felt unwell. On examination he was noted to be in much discomfort and was vomiting during examination. Blood pressure was 172/119 in the right arm and 137/92 in the left arm. The rest of the cardiac examination was unremarkable. He had a full complement of pulses, no bruits or murmurs. High-sensitivity Troponin was elevated (19.29 ng/ml), but not rising. An electrocardiogram (ECG) and a transthoracic 2D-Echocardiogram showed left ventricular hypertrophy and no wall motion abnormalities. He was also noted to have an elevated creatinine (177 µmol/L). In the absence of a magnetic resonance aortogram (MRA), a Computed tomography aortogram (CTA) was done showing an aortic dissection involving the descending aorta (Stanford Type B) extending to the left common iliac, with iliac, superior mesenteric and both renal arteries arising from true lumen (Figure 1A). He was admitted to the intensive care unit and started on morphine and labelatal infusion at 2 mg/hour. Due to the inevitable contrast, and the baseline renal dysfunction, he was given N-Acetyl cysteine and later dialyzed.

The patient was selected for TEVAR according to Cooper et al. [5] proposed algorithm and the decision reinforced by the history of non-compliance to medical therapy. A Valiant Captiva (Medtronic) 38x200 mm stent was deployed through the right femoral artery into the aorta, landing proximally just distal to the subclavian artery and distally just distal to the celiac artery with an overlap of 110mm and effective occlusion of the false lumen (Figure 1B). 12 hours post TEVAR however; the patient started complaining of colicky, non-specific abdominal pains; associated with a leukocytosis, decreased platelet counts, absence of fever and rising C-reactive protein (CRP) (Figure 2). He was managed conservatively with non-steroidal anti-inflammatory agents, antispasmodics and PPIs. 72 hours post procedure; he was transferred into the high-dependency unit and two days later to the ward. Here, the pains worsened with associated diaphoresis. Platelets remained low; leukocytosis persistent and CRP elevated. This however resolved by day 5 post TEVAR, leaving only CRP way above the baseline. Procalcitonin was normal and cultures negative. The abdomen was soft, surgical sites clean, and bowel sounds present. Liver function tests, serum amylase and lipase were all normal. The pain was not relieved by antispasmodics and six-hourly non-steroidal anti-
inflammatory agents. A repeat CTA (initially held back due to renal dysfunction) was obtained and the graft shown to be in place without endoleaks. An array of other investigations was done to investigate the cause of the abdominal pains including an abdominal ultrasound (minimal gall bladder sludge), repeat electrocardiograms, repeat CT scans of the chest, abdomen and pelvis, oesophagoscopy and magnetic resonance imaging of the thoracic and lumbar spine (no change from pre-operative assessment). At this point, 16 days post procedure; he was started on intravenous steroids (for post implantation syndrome) with dramatic clinical improvement and subsequently discharged home after 48 hours, pain-free.

Figure 1: (A) CT Aortogram showing the dissection, 3D reconstruction; (B) Immediate post TEVAR showing contrast only in true lumen.

Discussion

An aortic dissection results from an intimal tear that leads blood into a false lumen between the intimal and medial layers of the aortic wall [5]. Although this calamitous condition was first described in the medical literature over two centuries ago; its management remains complex and mortality high [1]. As classified by Daily et al. [6] in 1970 at Stanford; Type A dissections involve the ascending aorta and Type B are dissections involving only the descending aorta. Type B dissections account for over a third of aortic dissections [1,5]. Majority of these are associated with hypertension and are uncomplicated; so assigned by the absence of associated malperfusion, hypotension, refractory hypertension or findings suggestive of impending rupture [2].

As soon as a diagnosis of an uncomplicated AAD-B is established, a majority of patients are started on medical therapy in an intensive care unit; and this has been widely accepted as the standard treatment [2,3]. This includes intensive anti-impulse therapy; the cornerstone of which is reduction of pressure on the aortic wall and subsequent decreased propagation of the false lumen. While a majority of patients on this treatment are discharged with no complications; the long term outcomes are perturbing with high mortality and complication rates [7]. Subsequently, questions abound about the possibility of multimodal therapy with adjunctive TEVAR in a bid to improve the outcomes [5]. This entails stent-graft placement commonly through the trans-femoral route under fluoroscopy guidance; the premise of which is to occlude the primary intimal tear with a stent-graft promoting false lumen thrombosis and subsequent aortic remodelling [3].
Although the Investigation of STEnt grafts in patients with type B Aortic Dissections (INSTEAD-XL) [7] trial, did not demonstrate a significant decrease in all-cause mortality with use of TEVAR in chronic uncomplicated type B dissections, there was a significant difference in aortic-specific mortality at 5 years (6.9% versus 19.3%) and lower disease progression with TEVAR compared to medical treatment alone. This has been replicated in two recent studies confirming the feasibility of TEVAR in AAD-B [8,9]. Careful patient selection for adjunctive TEVAR in uncomplicated AAD-B is however important with priority to those prone for developing complications [10] as it is not without risks, one of which is PIS.

First described in 1999, PIS, is a vaguely defined inflammatory response following endovascular repair that presents as a SIRS with a noninfectious fever, leukocytosis (>12,000/μL), elevated CRP (>10 mg/L) and coagulation disturbances including thrombocytopenia in the context of negative blood culture results; occurring in up to 60% patients [4]. Though the exact pathophysiology remains an enigma, PIS is believed to result from complex endothelial-stent fabric interactions where tissue injury activates complement and induces production of various pro-inflammatory mediators including TNF-α, IL-1, IL-6; with local and systemic effects [4,11]. While the systemic effects are well appreciated, and define post-implantation syndrome; there is hardly any mention of its local effects, including local pain.

In Kenya, compliance to antihypertensives has been shown to be low [12], and thus an added benefit with TEVAR in management of this case postulated. While the patient developed evident SIRS with tachycardia, leukocytosis, thrombocytopenia, and high serum CRP post TEVAR; it is his refractory pain that was worrying. Evaluation for the etiology of this, from endoleaks to cord ischemia yielded no positive result and thus the eventual management as an atypically presenting post-implantation syndrome, refractory to non-steroidal anti-inflammatory agents. Although PIS is a largely systemic inflammatory response; perigraft inflammation has been shown in experimental studies [13] and thus features of local inflammation (like pain in our case) should be borne in mind to avoid unnecessary tests and delayed treatment with associated lengthy hospital stays.

References

