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Aortic Dissection with Hemopericardium and Thrombosed Left Common Iliac Artery Presenting as Acute Limb Ischemia: A Case Report and Review

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Abstract Aim: An aortic dissection is an uncommon serious condition, which usually presents with chest pain or upper back pain. Symptoms of aortic dissection may mimic those of other diseases, often leading to delay in diagnosis. Methods: We report an unusual case of aortic dissection with hemopericardium and thrombosed left common iliac artery presenting as acute limb ischemia. Conclusion: Maintaining a high index of clinical suspicion for aortic pathology could possibly lead to identification and timely management of a greater number of patients who have atypical presentations. This would be especially true for patients who have catastrophic presentations with unexplained symptoms.

Keywords: *aortic dissection, type a dissection, type b dissection, hemopericardium, cardiogenic shock, limb ischemia, arterial thrombosis*

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1. Introduction

An aortic dissection (AD) is an uncommon serious condition, which is usually fatal even before arrival to emergency department (ED). Symptoms of AD may mimic those of other diseases, often leading to delay in diagnosis. The acute onset of severe chest pain or upper back pain was the most common initial complaint and less common manifestations include widened mediastinum on chest x-ray, congestive heart failure, syncope, cerebrovascular accident, shock, paraplegia, and lower extremity ischemia.

2. Case Presentation

A 43 year old African American male with past medical history of Hypertension (HTN), current smoker, Motor vehicle accident (MVA) few months ago with back trauma was getting physical therapy presented with sudden onset of lower back pain while getting into the car which radiated to left leg followed by numbness and weakness of the left leg. He had multiple episodes of vomiting containing clear contents before coming to ED. On arrival to ED, his vitals were: heart rate (HR) regular at 83 beats per minute, blood pressure (BP) 153/74 mm Hg, respiratory rate 16 cycles/min and saturation 100% on room air. On physical examination patient had feeble femoral, popliteal, dorsalis pedis pulses in right leg, and absent femoral, popliteal, dorsalis pedis pulses in left leg. Power: right leg 4/5 and 0/5 in left leg. Sensations were intact in right leg but there was loss of touch and pain sensation in the left leg below the knee. Both lower extremities were cold but left lower extremity was extremely cold. Remaining physical exam was normal.

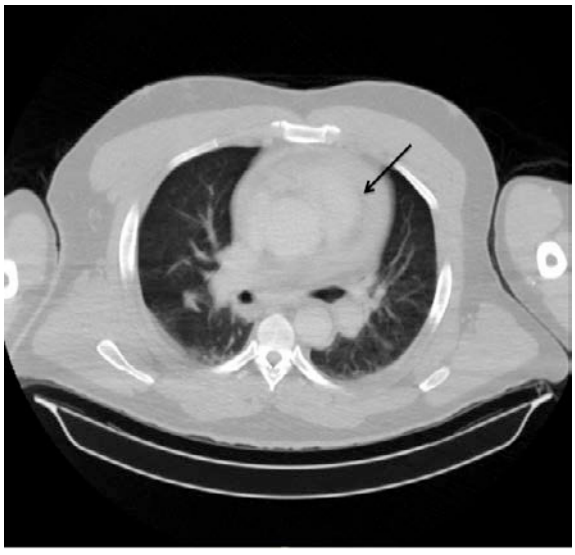

Figure 1. Computed Tomography of chest showing hemopericardium

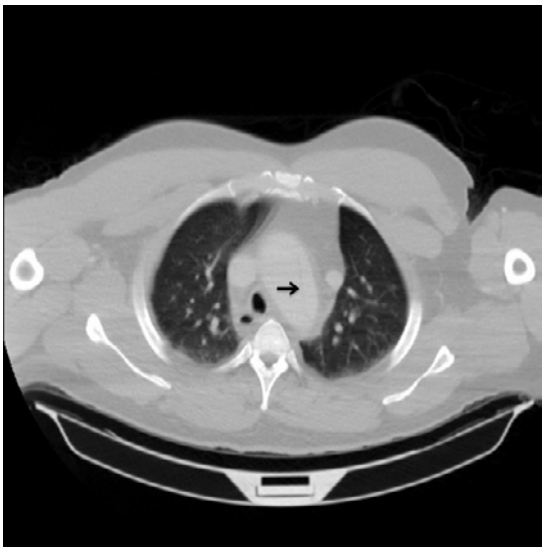

Figure 2. Computed Tomography of chest showing aortic dissection involving aortic root

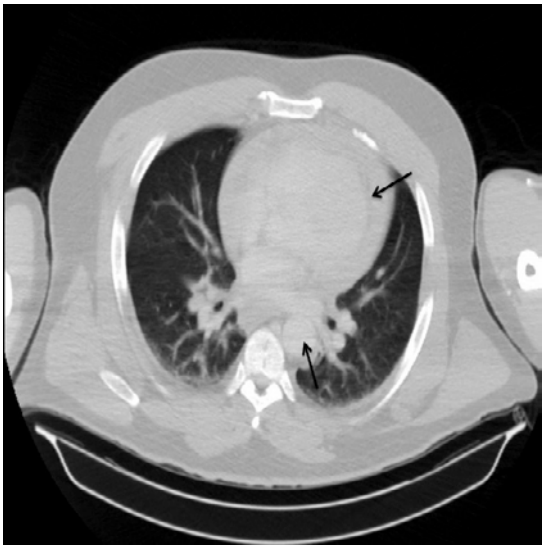

Figure 3. Computed Tomography of chest showing hemopericardium and descending aortic dissection

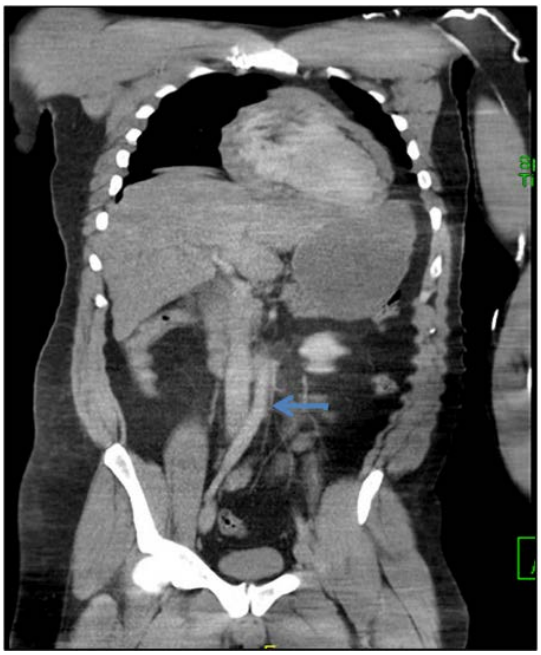

Figure 4. Computed Tomography of chest showing thrombosis of left common iliac artery

Initial work up that included electrocardiogram, chest radiography and basic labs were completely normal. Vascular surgery was consulted to rule out acute limb ischemia. In next couple of hour's patient's BP dropped to 78/50 with HR ranging from 114-130 beats per minute. Patient did not respond to fluid bolus and was started on a norepinephrine drip. Repeat labs showed drop in hemoglobin from 13.2 to 12.1 g/dl, increase in blood urea nitrogen/creatinine 16/1.0 to 19/2.2 mg/dl, lactic acid > 12 mmol/L and negative troponins. After stabilizing the patient, CT chest, abdomen, pelvis and lower extremity was done which revealed large hemopericardium [Figure 1] with aortic dissection originating at the aortic root, extending into the distal thoracic aorta [Figure 2, Figure 3] with thrombosed left common iliac artery [Figure 4].

Patient was evaluated by vascular and cardio-thoracic surgery and was transferred to other facility for emergent surgery. Patient underwent replacement of ascending aorta, hemi-arch and resuspension and repair of aortic valve with a 32 mm Hemashield double Velour Dacron graft. Total circulatory arrest time was 42 minutes and received antergrade cerebral perfusion for 41 min. Patient also underwent femoral-femoral bypass surgery. After the surgery, patient's limb got better and was transferred to rehabilitation center. Patient continues to follow at cardiovascular and vascular surgery clinics and was doing well for the last 18 months.

3. Discussion

3.1. Introduction

AD is an uncommon serious condition, which usually presents with chest pain and upper back pain but can have many atypical presentations such as stroke, myocardial infarction, syncope, mesenteric and limb ischemia [1,2,3,4]. Many patients die even before they present to the ED or prior to the diagnosis [5]. Our patient presented with low back pain and acute lower limb ischemia and found to have type A aortic dissection with hemopericardium in addition to occlusive thrombus; incidentally diagnosed on complete vascular imaging, which was done to investigate the cause of hemodynamic instability. Aortic dissection can present with impaired or absent blood flow in peripheral vessels (carotid, brachial, femoral) [1]. This pulse deficit most commonly results from intimal flap or compression by a hematoma [5]. Literature review showed, very few cases have been reported with concomitant AD and acute distal arterial thromboembolic occlusion of a vessel [6,7,8,9].

Olsson C. et al. reported an incidence of 9.1 cases per 100,000 in women increasing to 16.3 per 100,000 in men based on study from national heath care registry data and out of hospital death autopsies [10,11] and Howard et al. reported incidence of 6 cases per 100,000 population in Oxford Vascular study [12]. AD is more frequently seen in men compared to females in a ratio of 1.5: 1 and men developed at younger age compared to females (79.3 verses 67.1 years) per Oxford vascular study [12]. Marfan syndrome patients develop AD at very young age (less then 40 years) [5]. Diagnosis is often missed in up to 38% of cases on initial evaluation and first established in 28% of cases during autopsy [1,13].

3.2. Etiology and Pathogenesis

AD is a longitudinal tear initiated in intima progressing to disruption of the medial layer thus creating true and false lumen, which could extend the tear in either directions of aorta resulting in rupture of aorta, hemopericardium, massive blood loss, end organ ischemic damage as in our case and death [5,6,11]. AD is described as acute if symptom onset is less than 2 weeks and chronic if more than 2 weeks [5,11]. AD type based on anatomy is classified by two classification systems. Stanford system classifies dissections that involve the ascending aorta as type A and all other dissections as type B regardless of the site of primary intimal tear. [2,4,5,11] Where as the DeBakey classifies as type I involving the ascending aorta and the aortic arch, type II involving the ascending aorta only and type III involving the descending aorta only [2,5, 11]. Chronic systemic hypertension is the most common predisposing risk factor implicated in AD. It is prevalent in 62 to 78% patients with AD [1,5,11,12,13] and increases the aortic wall stress initiating the tear in intima [5,11]. Hypertension leads to structural (atherosclerosis, myo-intimal thickening) and functional (increased stiffness, loss of endothelial function) changes in the arterial system and eventually leading to structural changes of left ventricle (hypertrophy). This interplay between ventricle and arterial system is called as ventricular-vascular coupling [14]. Initially it is an adaptive phenomenon and over time with chronic hypertension, aging and other factors lead to heightened arterial stiffness, change in pressure wave amplifications, velocity and its timing, leading to ventricular-vascular mismatch which acts as a main underlying pathophysiology for cardiovascular diseases such as myocardial ischemia, heart failure, aneurysms and dissections [14]. Understanding ventricular-vascular coupling helps in determining pathophysiology of cardiovascular manifestations [14]. Other etiologies that would increase the wall stress are physical trauma, deceleration injury in motor vehicle accidents and falls [15,16] pheochromocytoma and cocaine use from catechocholamine surge [17], and coarctation of aorta. Smoking also increases the risk of dissection by affecting Transforming growth factor TGF-B that promotes smooth muscle growth and function in the media [11]. Our patient had chronic systemic hypertension, chronic smoker and also experienced motor vehicle accident which would be predisposing factors for AD. Other predisposing factor contributing to AD is medial degeneration seen in conditions such as Marfans syndrome, Noonan syndrome, Ehlers-Danlos syndrome, Turner syndrome, inflammatory diseases of aorta and bicuspid aortic valve [7,18,19]. Hagan et al. reported 4.3% in IRAD study [13] Litchford et al. [20] and Archer AG et al. [21] reported 5% had direct iatrogenic AD from previous cardiac surgery during arterial cannulation or during catheter based diagnostic or therapeutic interventions. Most of the iatrogenic dissections result in type B dissections.

3.3. Management

Having high index of clinical suspicion is the foremost important step in diagnosing devastating disease. Computed tomography (CT) is most common, readily available and widely used modality for quick diagnosis of AD. It has high sensitivity (83-94%) and specificity (87-100%) in diagnosing AD [22]. The presence of an intimal flap is characteristic of AD as shown in Figure 2 and Figure 3. Helical or spiral CT is shown to be superior compared to conventional CT in detecting AD. Trans-esophageal echocardiogram is highly sensitive (97-99%) and specific (97-100%) in diagnosing AD, identifying the entry site of dissection, presence or absence of thrombus, involvement of coronaries and arch vessels, aortic valve involvement and hemodynamics across the aortic valve and pericardial effusion [23,24,25]. Magnetic resonance imaging also has very high sensitivity and specificity in range of 95-100%, provides all the information comparable to TEE and helpful in patients with previous aortic disease, surgical repair and in construction of three dimensional images [26,27]. Chest radiography, electrocardiogram and trans-thoracic echocardiogram are less specific in diagnosing AD. Chest radiography lacks specificity but helps in predicting the disease. Widened mediastinum is seen in 50% of cases with AD. Presence of non-specific changes on electrocardiogram in a patient with chest pain, diagnosis points towards AD over myocardial infarction. Aortography is an invasive, time consuming procedure and rarely used in recent days.

Type A dissections require urgent surgical intervention where as Type B dissections are managed medically with blood pressure control and pain management [2,4,5]. Understanding ventricular-vascular coupling helps in determining pathophysiology of cardiovascular manifestations and its management modulation [14]. Unoperated type A dissections have estimated mortality rate of 1% per hour to 2% per hour during first 48 hours [28]. AD resulting in rupture has a 90% mortality rate [6]. Fann et al. reported overall operative mortality rate for acute AD is 24% ± 8%, acute type A AD it is 26% ± 3%, acute type B dissection 39% ± 8% [29] and 50 to 70% for renal ischemic patients [30]. Per IRAD study in-hospital mortality rate for type A dissection patients treated surgically was 27 % compared to medical management (high operative risk and elderly patient population) was 58% and for type B dissection patients managed medically was 10.7 % and 31.4% in surgical group.

Surgery is the standard of care and it is indicated in all type A dissections, unless serious comorbidities preclude surgical intervention [31]. The aim of surgery is to excise and replace the aortic segment with dissection but not to replace entire dissected aorta and to maintain aortic valve integrity. Best-known surgical procedures for type A dissections are the Bentall, David (valve sparing), Yacoub, Ross, aortic homograft and Elephant technique [5,11,19,32]. Glue aortoplasty is usually combined with AD surgical procedure. Our patient underwent valve sparing aortic root replacement surgery (David procedure). There is no role for endovascular stents in type A dissections [33].

Type B dissections are managed medically unless complicated. Medical management is shown to be superior to management with either surgical or endovascular stent approach in uncomplicated type B dissections. The cornerstone of medical management is tight Blood pressure and heart rate control with B-blockers and vasodilators such as Angiotensin-converting-enzyme inhibitors (ACEI) aiming to reduce the force of left ventricular contractions, to decrease steepness of the rise

of the aortic pulse wave (ie, dp/dt) and pain management [5]. Surgical or endovascular intervention is indicated in complicated type B dissections, which comprise of type B dissection with persistent or recurrent pain despite adequate antihypertensive therapy with at least two parenteral agents at moderate to high dose, aortic diameter ≥ 5.5 cm, diameter increase > 4mm, acute expansion of the false lumen, periaortic or mediastinal hematoma (contained rupture) and in visceral, renal or limb malperfusion syndrome [34]. Surgical interventions include replacement of aorta or bypass surgery. Our patient underwent femoral-femoral bypass surgery, which resulted in complete recovery of left leg ischemia. Hybrid procedure includes combination vascular surgical procedure and endovascular stents. IRAD study showed, patients who underwent thoracic endovascular aortic replacement (TEVAR) for type B dissections had lower 5-year mortality compared to patients treated medically [11,35]. Based on IRAD study data, the food and drug administration (FDA), Society for Vascular surgery (SAS), American Association for Thoracic surgery, Society of Thoracic Surgeons and the Society for Interventional Radiology recommended TEVAR for complicated type B dissections [11,36].

AD managed medically or surgically needs long-term care and follow up as they have potential to develop aortic aneurysm and propagate dissection. Long-term management consists of aggressive blood pressure control, smoking cessation and clinical and imaging assessment of the aorta for early detection and correction of the potential complications [5,37]. Tight blood pressure control may reduce incidence of re-dissection in about one third of patients [38]. IRAD database showed B-blockers are associated with improved survival in type A dissections [39].

Our patient presented with concomitant Stanford type A AD and left common iliac arterial thrombosis. It presents a therapeutic dilemma as the management of arterial thrombosis usually involves inhibition of the coagulation cascade. There were no clear guidelines for management of AD with malperfusion or arterial thrombosis and use of anticoagulants, but the general consensus was to treat aortic dissection first as it would correct malperfusion in most patients [40]. Thoracic aortic surgery is associated with hemorrhage due to numerous interrelated causes such extensive surgical exploration, arterial and venous bleeding, consumptive coagulopathy, antithrombotic use, platelet dysfunction, coronary bypass and hypothermia [40]. The Society of Thoracic Surgeons (STS) and The Society of Cardiovascular anesthesiologists (SCA) have defined clinical practical guidelines for use of blood products during perioperative period. However there was no clear time frame defined to start anticoagulation or venous thromboembolic prophylaxis post-operatively [41]. Study by Kincaid et al. showed that patients receiving low molecular weight heparin (LMWH) within 12 hours of cardiac surgery have significantly greater blood loss and increased blood transfusion compared with patients receiving intravenous heparin or a dose of LMWH more than 12 hours before operation [42]. Medalion et al. showed that the risk of postoperative bleeding or transfusion requirement is less if enoxaparin administered more than 8 hours before coronary artery bypass surgery [43]. For patients undergoing cardiac surgery, it is recommended to use mechanical prophylaxis with intermittent pneumatic compressions (IPC) in uncomplicated post operative course, low dose unfractionated heparin (LDUH) or LMWH in addition to IPC in patients whose hospital course is complicated by one or more non hemorrhagic surgical complications and in patients with moderate risk for venous thromboembolism (VTE) and not high risk for perioperative bleeding, LDUH or LMWH or mechanical prophylaxis with IPC over no prophylaxis [44].

Unfractionated Heparin is widely used in perioperative period especially in cardiac surgery [40] due its shorter half life, less bleeding risk and availability of strong reversal agent, but its use is limited by concerns of heparin rebound, heparin resistance, protamine reaction, heparin-induced thrombocytopenia, heparin- induced platelet dysfunction and less anti-thrombotic activity compared to LMWH. Literature review showed wide spread usage of a second generation LMWH - Bemiparin, in Europe in perioperative period including high-risk surgical procedure [45]. Its usage is getting wider given its better pharmacological profile – anti-factor Xa:anti-factor IIa (8:1), increase in release of tissue factor pathway inhibitor from endothelial cells under both static and shear stress conditions, more favorable efficacy: safety ratio and shown to be superior to fondaparinex, and unfractionated heparin in suppressing angiogenesis and vasculogenesis [45]. Bemiparin may have a potential for use in cardiac surgeries with malperfusion like our case in perioperative period but needs further studies evaluating its use. Our patient was severely coagulopathic after he was rewarmed and weaned of cardiopulmonary bypass necessitating multiple rounds of clotting factors. He was placed on venodyne boots in perioperative period and started on subcutaneous heparin on first post-operative day.

4. Conclusion

Maintaining a high index of clinical suspicion for aortic pathology could possibly lead to identification and timely management of a greater number of patients who have atypical presentations [1,2,4,5,13]. This would be especially true for patients who have catastrophic presentations with unexplained symptoms.

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None.

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