Aortic Intramural Hematoma (IMH): A Case Report
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Abstract

Aortic intramural hematoma (IMH) accounts for 10-30% cases of acute aortic syndrome. Although less common, IMH has the identical clinical manifestations, risk factors, clinical courses and treatment principles to aortic dissection. Without prompt recognition and proper management, IMH can also result in high mortality. We reported a 52-year-old man who was admitted to our hospital due to sudden onset of severe tearing chest pain, radiating backward to intra-scapular area. Initial chest computed tomography (CT) showed a focal wall thickening at the aortic arch without a demonstrable intimal flap or false lumen. Under the impression of IMH, the patient was admitted to medical intensive care unit for blood pressure control and further evaluation. The next day, the patient's blood pressure was controlled within an optimal range without complications. He was then transferred to general ward with oral anti-hypertensive agents. However, one day later, he complained of severe tearing chest pain again radiating to his left flank area. An immediate follow-up chest CT showed aortic dissection, type B, extending from aortic arch to the bifurcation of distal abdominal aorta. The patient was subsequently referred to a nearby medical center for further management.

Key Words: Aortic intramural hematoma.

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Received: 1Aug 2008; Accepted: 29 Aug 2008
Introduction

Aortic intramural hematoma (IMH) is a medical emergency. It has comparable clinical manifestations and risk factors to aortic dissection. Although it has a better prognosis than aortic dissection, cardiovascular catastrophes can result without prompt recognition and proper management. Hereby we reported a case of IMH in a 52-year-old man and reviewed the relevant literature.

Case Report

A 52-year-old man was admitted to our hospital with the chief complaint of severe and persistent tearing chest pain radiating to the back for 5 hours in January, 2007. The patient had hypertension for several years without regular medical control. Physical examination showed that his blood pressure was 190/120 mmHg, same in both arms. Chest radiography demonstrated a widened aorta (Figure 1). His electro-cardiogram showed anterior ST elevation and left ventricular hypertrophy. Cardiac-specific enzymes were within normal range. Immediate chest computed tomography (CT) with contrast media showed a focal wall thickening with relatively high radiodensity at the aortic arch without a demonstrable intimal flap or false lumen (Figure 2).

The patient was immediately admitted to medical intensive care unit for strict blood pressure control with intravenous beta-blocker infusion. The next day, his chest pain subsided and his blood pressure was controlled at 130/70 mmHg. He was then transferred to general ward with oral beta-blocker, calcium channel blocker and nitrates. However, one day after, chest pain recurred and propagated to left flank area. An immediate follow-up chest CT with contrast media (Figure 3 and Figure 4) showed aortic dissection, type B, extending from aortic arch to the bifurcation of distal abdominal aorta. Under the strong request of the patient and his family, he was then immediately referred to a nearby medical center for further management.

Figure 1. CXR showed a widened aorta

Figure 2. A focal wall thickening with relative high radiodensity lesion at the aortic arch
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Discussion

A variant form of aortic dissection, aortic intramural hematoma (IMH), although less common, still accounts for 10 – 30 % cases of all acute aortic syndrome (1). The exact causes of IMH remain controversial. Several studies proposed that rupture of the vasa vasorum of the aorta resulted into intramural hemorrhage of the aortic wall without an intimal flap or, less commonly, due to an atherosclerotic penetrating aortic ulcer(2). The risk factors and clinical presentation are similar for both IMH and classic aortic dissection. For example, systemic hypertension, as a strong risk factor in aortic dissection, also accounts for 53 % of patients with IMH(3). The typical clinical manifestations of IMH include chest pain (50-74%), intra-scapular back pain (44-84%), neurological deficits and cardiovascular complications such as transient ischemic attack, syncope, pericardial effusion, mesenteric ischemia, acute renal failure and shock (4), which can also be found in patients with aortic dissection. Therefore, it is difficult to distinguish IMH from aortic dissection based solely on clinical grounds. Interestingly, the study made by Kim et al(5) showed that IMH tend to affect older individuals (mean age of 67 years) without particular gender predominance, comparing to classic dissection (mean age of 55 years) affecting predominantly men. In addition, this study also stated that patients with IMH had a lower proportion of ascending aorta involvement comparing to aortic dissection (32 % vs. 58%, respectively).

Aortography directly visualizes the intimal flap or double lumen in acute aortic syndrome. For once, it was the only modality to diagnose aortic dissection.

Figure 3 and Figure 4. Aortic dissection, type B, extending from aortic arch to the bifurcation of distal abdominal aorta
To some, it still remains as the “gold standard”. However, its accuracy was challenged. In 1989, Erbel et al (6) reported that aortography had a sensitivity of 88% and a specificity of 94%. It is generally agreed that the absence of an intimal flap or intraluminal flow in IMH limits aortography for detection (7). The advent of new imaging modalities, such as magnetic resonance imaging (MRI), CT and transesophageal echocardiography (TEE), has basically replaced aortography for detection of aortic dissection. In these imaging modalities, IMH is typically described as a focal thickening of the aortic wall without an intimal flap. An aortic wall thickness $\geq$ 7 mm with no evidence of intimal tear and communication of flow, was suggested by some authors initially, as a diagnostic criteria indicative for IMH(8). However, in 2004, Song et al(9) reported an aortic wall thickness $\geq$ 5 mm with typical clinical symptoms is sufficient to establish the diagnosis of IMH. MRI is non-invasive and requires no intravascular contrast. It yields a sensitivity of 98 – 100 % and a specificity of 100 % in detecting aortic dissection(10). Comparing to MRI, previous studies showed conventional CT is somehow less accurate in identifying the intimal flap, which might lead to over-diagnosis of IMH in actual cases of aortic dissection(11,12). In addition, CT carries the inherent risk of intravascular contrast. However, in 2003, Yoshida et al(12) reported that helical CT has an accuracy of 100% in detecting aortic dissection and IMH of the thoracic aorta. But the availability of a helical CT remains a challenge in many medical institutions. TEE has a sensitivity and specificity of 95% to detect aortic dissection(6, 13). It accurately detects the intimal flap, making it a remarkable technique to differentiate IMH from aortic dissection. Furthermore, it can detect aortic valvular regurgitation, proximal coronary artery involvement and pericardial effusion. Comparing to CT or MRI, TEE establishes a diagnosis more quickly, and is easier to perform in intubated or critically ill patients.

It is agreed to classify IMH into type A (IMH involving the ascending aorta), and type B (IMH involving the descending aorta) using Stanford Classification. Similar to aortic dissection, IMH of the ascending aorta (type A) may result in pericardial effusion, hemothorax and mediastinal hemorrhage, thus raising the mortality to 34 %, comparing to 14 % in distal IMH (type B) (10). While some cases of IMH may remain stable, others may reabsorb completely, develop into aortic aneurysm, or even progress to classic dissections. Evangelista et al reported the most frequent evolution of IMH, which includes pseudo-aneurysm formation (54%), regression (34%) and progression to classic dissection (12%)(1), while other authors reported of 33% of aortic dissection progression (8,10). Ide et al (14)suggested that patients with aortic diameter $> 5$ cm tend to progress to class dissection, and this may serve as a predictive factor for dissection and aneurysm. Similar results were also found in the study of Kaji et al (15).

Most authorities currently recommend similar treatment in both IMH and aortic dissection. In acute phase of IMH, hospitalization with prompt blood pressure control is a must. The study of Blanchard et al(7)recommended that intravenous (IV) infusion of labetalol, either alone or in combination with sodium nitroprusside, is used to achieve immediate blood pressure control. The recommended dose of labetalol is 20 mg IV over 2 minutes, followed by 40-80 mg IV every 10 minutes until blood pressure is
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controlled (maximum of 300 mg) or 20 mg IV bolus over 2 minutes followed by 2-mg/min IV infusion. If any contraindications (e.g. hypersensitivity to beta-blockers, asthma, congestive heart failure, severe bradycardia, second-or third-degree heart block) and severe side effects are encountered, alternative medications such as calcium channel blockers or trimethaphan should be used. Sodium nitroprusside is a rapid-acting arterial and venodilator, which potentiates the effects of all antihypertensive agents. The recommended dose range is 0.5 to 10 μg/kg/min. However, due to the toxicity of metabolic products and possible severe cardiovascular side effects, sodium nitroprusside should be used only in intensive care units with close cardiovascular monitoring.

Immediate surgery to replace aortic root and decompress hematoma in patients with type A IMH is generally recommended. Kodolitsch et al (16) suggested aortic dilatation (>50 mm) and ascending aortic involvement as the most important predictors of early progression and death in IMH. They also reported a high risk of early aortic rupture and high mortality in patients with type A IMH who did not undergo urgent surgery. However, some Asian authors reported medical therapy alone might be adequate in elderly patients with proximal IMH (without dissection) (17). Further studies are needed to investigate these issues.

For patients with acute type B IMH, medical therapy alone may suffice unless end-organ perfusion is compromised (7). Kodolitsch et al also reported a better long-term prognosis in patients with IMH taking oral beta-blockers indefinitely. Second line agents such as verapamil or clonidine may be used if absolute contraindications to beta-blocker are encountered (16). Blanchard et al recommended that patients with medically treated IMH should undergo serial imaging follow-ups at 3, 6, 9, and 12 months after initial presentation, then every 6 to 12 months. If there is complete resolution of IMH, or no change in aortic diameter over the first year, annual imaging thereafter should suffice (7).

In conclusion, IMH accounts for 10-30% cases of acute aortic syndrome. It is not uncommon for IMH to progress to classic dissection and cause cardiovascular catastrophes. Clinicians should keep in mind the possible clinical presentations, correct interpretation of diagnostic images and selection of optimal managements for patients with IMH. Regular imaging follow-up and constant observations of clinical signs and symptoms are strongly recommended for this type of patients.

References

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個案報告

主動脈內皮血腫：
一病例報告
林志勇1 吳中興1* 謝俊雄2
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摘 要
在所有急性冠心病的病人中，主動脈內皮血腫病人佔了約百分之十到三十。雖然盛行率不及主動脈剝離，但其臨床表徵、危險因子、病程變化、和治療原則，卻與主動脈剝離相似。若無及時診斷與治療，主動脈內皮血腫也會造成相當高的死亡率。我們報告一位五十二歲的男性，因突然急性胸痛牽延至後背求診。胸部電腦斷層顯示主動脈弓壁有局部增厚情形，但無明顯內膜瓣剝離或主動脈假腔的形成。病人初步被診斷為主動脈內皮血腫，隨即被轉入內科加護病房密集觀察及血壓控制。次日，由於血壓穩定且無任何併發症，病人被轉回一般病房並持續給予口服降壓劑控制血壓。隔天凌晨，病人血壓突然升高且合併有左胸極度疼痛並且牽延至左側腹部的情形。此次胸部電腦斷層顯示出B型主動脈剝離；從主動脈弓延伸至腹部主動脈分叉點。在病人及其家屬的強烈要求下，病人隨即被轉介至鄰近的醫學中心做進一步治療。

關鍵字：主動脈內皮血腫

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收件日期：2008年8月1日；接受日期：2008年8月29日