รายงานกรณีศึกษา: ภาวะหลอดเลือดแดงใหญ่ฉีกขาดโดยที่ไม่มีอาการเจ็บหน้าอกร่วมกับโรคหลอดเลือดสมองและภาวะหัวใจล้มเหลว

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Painless Aortic Dissection Presented with Ischemic Stroke and Developed Congestive Heart Failure: A Case Report

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Abstract

Aortic dissection (AD) is a life-threatening condition that presents with diverse and atypical symptoms. Although transient or permanent neurological symptoms at onset of aortic dissection are not frequent (17-40% of the patients), a diagnosis with predominant neurological symptoms can be difficult and delayed especially in pain-free dissection (which occur in 5-15%). Additionally, only 6% of aortic dissections present with acute congestive cardiac failure, recognized as a potential complication of aortic dissection. We reported the case of 53-year-old female who presented with left-side weakness and numbness without chest pain. After 5 days of admission, she developed atrial fibrillation, congestive heart failure and respiratory failure. Transthoracic echocardiography (TTE) revealed dilated ascending aorta, dissection flap at the ascending aorta extended to descending aorta with hemopericardium. She received a delayed diagnosis of type A aortic dissection. Aortic dissection should always be included in the differential diagnosis of ischemic stroke and unexplained acute congestive heart failure, regardless of the presence of chest pain.

Keyword: Painless aortic dissection, Stroke, Congestive heart failure

บทคัดย่อ

ภาวะหลอดเลือดแดงใหญ่ฉีกขาด เป็นภาวะที่อันตรายรุนแรงถึงชีวิต ผู้ป่วยสามารถมีอาการนำที่มาพบแพทย์ได้หลากหลาย โดยอาการนำทางระบบประสาทพบได้ไม่บ่อยนัก โดยพบได้ประมาณ 17-40% ซึ่งการวินิจฉัยเป็นสิ่งที่ท้าทายและอาจทำให้การวินิจฉัยล่าช้า โดยเฉพาะในผู้ป่วยที่ไม่มีอาการเจ็บหน้าอกร่วมด้วย ซึ่งพบได้ 5-15% ของภาวะหลอดเลือดแดงใหญ่ฉีกขาด ยิ่งไปกว่านั้นภาวะหัวใจล้มเหลวพบได้น้อยในผู้ป่วยภาวะหลอดเลือดแดงใหญ่ฉีกขาด ซึ่งมักเกิดจากภาวะแทรกซ้อนของตัวโรคพบประมาณ 6% รายงานกรณีศึกษาผู้ป่วยหญิงไทยอายุ 53 ปีมาพบแพทย์ด้วยอาการแขนขาซีกซ้ายอ่อนแรงและชา โดยที่ไม่มีอาการเจ็บหน้าอก ภายหลัง 5 วันหลังเข้ารับการรักษา ผู้ป่วยมีภาวะหัวใจห้องบนเต้นพลิ้ว และมีภาวะหัวใจล้มเหลวตามมา ผลตรวจหัวใจด้วยคลื่นเสียงความถี่สูงพบหลอดเลือดแดงใหญ่เอออตาร์ส่วนต้นมีขนาดใหญ่ รวมถึงพบการเซาะแยกตัวของชั้นผนังหลอดเลือดแดงใหญ่เอออร์ตาส่วนต้นลงไปถึงหลอดเลือดแดงใหญ่เอออตาร์ส่วนปลาย นอกจากนี้ยังพบเลือดออกในเยื่อหุ้มหัวใจ ผู้ป่วยได้รับการวินิจฉัยภาวะหลอดเลือดแดงใหญ่ฉีกขาด (Stanford type A) และได้รับการผ่าตัดหลอดเลือดแดงใหญ่เออตาร์เร่งด่วน ผลการผ่าตัดเป็นที่น่าพอใจ ผู้ป่วยสามารถจำหน่ายออกจากโรงพยาบาลและใช้ชีวิตประจำวันได้ปกติ ในรายงานกรณีศึกษาผู้ป่วยในรายนี้เน้นถึงความสำคัญของการวินิจฉัยที่รวดเร็ว โดยเฉพาะผู้ป่วยหลอดเลือดแดงใหญ่ฉีกขาดที่ไม่มีอาการเจ็บหน้าอก ที่มาพบแพทย์ด้วยอาการทางโรคหลอดเลือดสมองและเกิดภาวะหัวใจล้มเหลวตามมา แพทย์จำเป็นต้อง

ตระหนักถึงภาวะนี้ร่วมด้วยเสมอ เพื่อให้ผู้ป่วยได้เข้ารับการรักษาและผ่าตัดได้อย่างเหมาะสม ทำให้ผู้ป่วยกลับมามีคุณภาพชีวิตได้ปกติและปลอดภัย รายงานกรณีศึกษานี้ให้ข้อมูลเชิงลึกที่มีคุณค่าในการวินิจฉัยและจัดการนำเสนอผู้ป่วยโรคหลอดเลือดแดงใหญ่ฉีกขาดที่มีอาการแสดงที่พบได้น้อย

คำสำคัญ: หลอดเลือดแดงใหญ่ฉีกขาดโดยที่ไม่มีอาการเจ็บหน้าอก, โรคหลอดเลือดสมอง, โรคหัวใจล้มเหลว

Introduction

Aortic dissection (AD) is uncommon but can be challenging for any physicians, considering its catastrophic high mortality rate if not diagnosed or managed promptly. The initial presenting symptoms and signs of acute aortic dissection are so diverse; hallmark feature of AD is a sudden onset of 'tearing' chest pain radiating to the back. However, 5-15% of cases are painless dissections which may present with symptoms secondary to complications of AD[1], including cerebral infarction or congestive heart failure (CHF), making early and accurate diagnosis arduous. The Standford classification is divided into two groups, A and B, depending on whether the ascending aorta is involved. The Standford type A involves the ascending aorta and often has high a mortality rate, requiring primary surgical treatment[2]. While, type B involves the descending aorta without involving the ascending aorta, which mostly managed medically first. Here, we present a case of Standford type A aortic dissection presented with left-side weakness and numbness without chest pain.

Case report

A 53-year-old Thai woman denied prior medical history, presented with left side weakness 3 hours prior to the admission. She developed sudden left side weakness and numbness, left facial palsy, dysarthria without loss of consciousness or chest pain. She denied any traumatic event. On admission her vital signs were as follows: blood pressure 110/70 mmHg; pulse regularly 74 beats/minute; respirations 18 breaths/minute; temperature 37 °C. No cardiac murmurs were heard. Lung and abdominal examinations were normal. Neurological examinations show mild dysarthria and left facial palsy. Manual muscle testing revealed muscle strength in left upper and lower extremities were grade III and IV consequently and right sides were grade V. Sensory tests showed a decrease in pinprick sensation on left side. The electrocardiogram demonstrated normal sinus rhythm. The patient was proceeded to CT brain for presumptive acute stroke (stroke fast track) which showed lacunar infarction at right basal ganglia. After re-evaluation her NIH stroke score decrease from 9 to 2, thus the patient was not eligible for thrombolytic therapy, she was admitted and treated as acute ischemic stroke with dual antiplatelet therapy (aspirin 300 mg and clopidogrel 75 mg daily). Electrolyte levels, renal function tests, transaminases and complete blood count were normal but fasting blood sugar were 128 mg/dl. The patient was first diagnosed diabetic. Two days after admission, monitor EKG showed atrial fibrillation (AF), so her medication was switched from antiplatelets to anticoagulants (enoxaparin 1 mg/kg/day and warfarin) to treat as cardioembolic stroke. Two days later, the patient developed acute dyspnea, orthopnea, restlessness but did not complain of any pain at the time. Her vital signs were as follows: blood pressure 199/97 mmHg;pulse totally irregularly 120 beats/minute; respirations 30 breaths/minute; temperature 37 °C. Cardiovascular examination revealed a diastolic murmur at right upper parasternal boarder and jugular venous pressure 5 cm above sternal angle. Rales were heard in the lower lung fields bilaterally. Pulses were present and equal in all four extremities. Chest X-ray showed wide mediastinum 8.6 cm and pulmonary congestion (Figure 1). Transthoracic echocardiogram showed dilatation of ascending aorta measuring 4.9 cm, aortic regurgitation, hemopericardium and dissection flap at ascending aorta extending to descending aorta (Figure 2). Computed Tomographic Angiography (CTA) whole aorta reveals intimal flap entry site at aortic root, involved thoracic aorta, brachiocephalic trunk, right common carotid artery, right subclavian artery, abdominal aorta and exit site at right common illac artery with evidence of hemopericardium (Figure 3). The diagnosis was ruptured Standford A aortic dissection, acute CHF with respiratory

failure. The acute right lacunar infarction could be explained by the dissection of ipsilateral right common carotid artery. She was then intubated, held anticoagulant and given Intravenous (IV) diuretic, IV Amiodarone (control AF rate) and IV Nicardipine (IV beta-blocker was unavailable). The patient was referred and had emergency open cardiac surgery by the thoracic surgeons and had an uneventful postoperative course. She was later discharged with regained left side motor grade V.

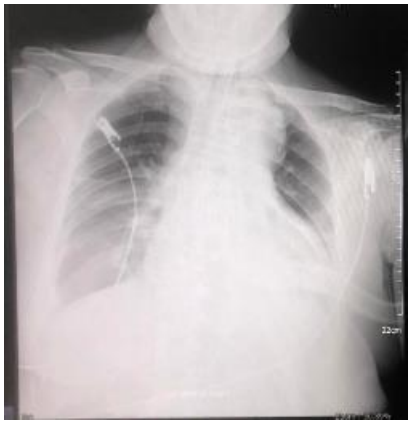

Fig.1 Anteroposterior (AP) view of portable chest radiograph showed a mediastinum width of 8.6 cm. and pulmonary congestion

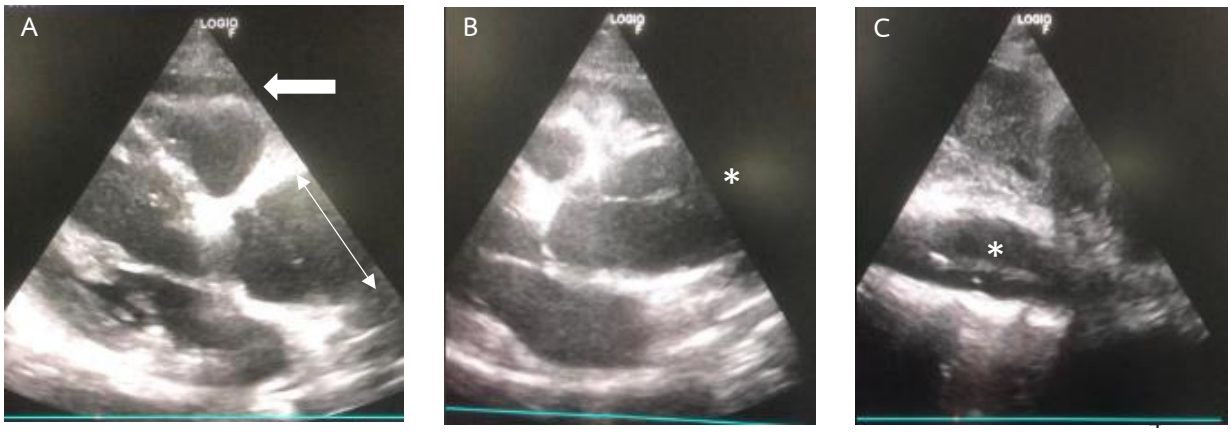

Fig.2a Parasternal long axis view showed dilatation of ascending aorta 4.9 cm. and hemopericardium (arrow)
Fig.2b Showed mobile dissection flap in the ascending aorta (star) and aortic regurgitation
Fig.2c Subcostal abdominal aorta view showed dissection flap in the descending thoracic/abdominal aorta (star)

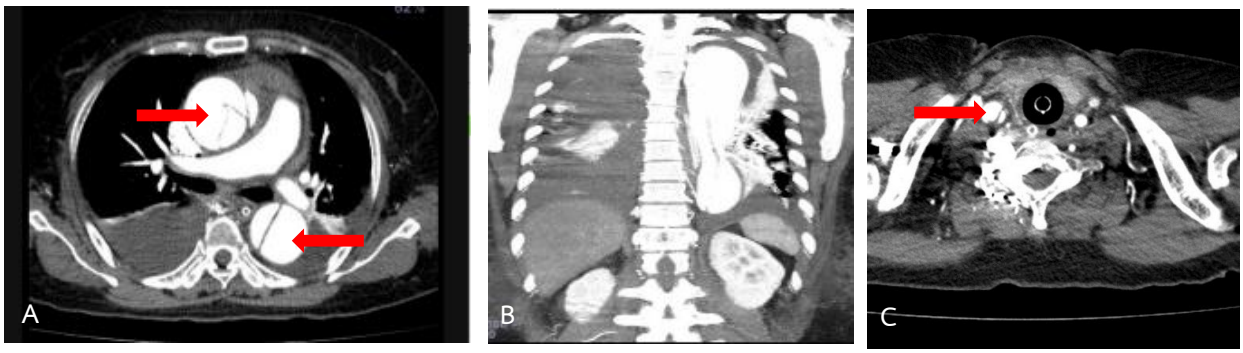

Fig.3 CT angiography whole aorta shows (A) Axial view (B) Sagittal view. Red arrows indicate intimal flap at ascending and descending aorta (C) Right carotid artery dissection

Discussion

Aortic dissection refers to an aortic disease in which the primary event is a tear in the intima, allowing blood to pass through the tear, forming a dissecting flap that can propagate further in an anterior or retrograde fashion, leading to numerous fatal complications. If left untreated, AD has a mortality rate as high as 1% per hour during

the first 48 hours after onset of symptoms[3]; thus, early and accurate diagnosis is essential for survival. The clinical presentations depend on localization of the dissection, its classic presentation is a sudden onset of a tearing, stabbing chest or back pain that radiates to the shoulder, neck, or jaw. Atypical presentations of AD make prompt diagnosis difficult and subsequently increase morbidity and mortality. Painless AD accounts for up to 5-15%[1], mostly type A dissection. Its common presentations include syncope, stroke, hypotension, and CHF[4]. The mechanism of painless AD may be explained from syncope and ischemic stroke may attenuate pain perception, and hypotension may diminish wall stress. Neurological manifestations (accounting for 17-40% of AD) can be classified to a) persistent or transient ischemic stroke b) spinal cord ischemia c) ischemic neuropathy and d) hypoxic encephalopathy[5]. The most common is acute ischemic stroke predominantly right-sided, which frequently evanescent and fully remitted[6]. Otherwise, thrombolytic therapy is an absolute contraindication; if administered, it could extend the dissection into the pericardium and result in death. Moreover, only 6% of AD cases present with acute congestive cardiac failure, which may lead to a delay in surgical intervention[7]. In type A AD, aortic regurgitation and cardiac tamponade are the main causes of acute heart failure, while in type B, it is usually due to myocardial ischemia or hypertensive crisis[8].

This is another case of painless type A AD presented with rapid recovery of left hemiplegia and later developed CHF. Fortunately, she did not receive rt-PA due to an improve NIH stroke score but still received anticoagulants for a presumptive cardioembolic stroke. From the aforementioned, painless AD could be presented with stroke or CHF, even the patient did not complain any syncope or disturbance of conscious, but in the setting of unexplained cardiac failure, aortic dissection should be considered, especially when an aortic regurgitant murmur has been detected as found in this patient. In such setting, the physical examination and transthoracic echocardiogram (TTE) are helpful tools, Positive pertinent findings in AD include asymmetrical pulses, asymmetrical blood pressure, or a new aortic murmur. Transthoracic echocardiography can provide information on the ascending aorta especially in type A AD (diagnostic rate up to 75%)[9]. To establish definitive diagnosis, better characterize the AD and decide on therapeutic option, CT angiography is performed. Pathologically CHF in this patient could be a result of aortic regurgitation or cardiac tamponade, with further conviction from previous anticoagulants use and delayed diagnosis. The initial treatment of AD is the same, regardless of the type. The patient should be kept pain-free and blood pressure should be controlled with IV beta-blockers. IV calcium-channel blockers can be utilized if IV beta-blockers are contraindicated or unavailable. Still, emergency open surgery is the mainstay treatment for type A aortic dissection.

Conclusion

To summarize, this is a case report of painless AD presented with stroke and CHF. Chest pain is not an obligatory symptom of aortic dissection especially in the context of clinical malperfusion syndrome in multiple organ systems such as cardiovascular and neurological systems. Although, such condition is rare but has a high mortality rate. Prompt diagnosis in right clinical settings and appropriate urgent treatment along with participation of multidisciplinary team are required in order to achieve good outcome.

Statement of Ethics

Verbal informed consent was obtained prior to the interview.

Conflict of Interest Statement

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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