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Case Report

A Case of Acute Type A Aortic Dissection Complicated with High-Altitude Pulmonary Edema

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Abstract: We report a case of a 55-year-old middle-aged male patient who developed dyspnea while traveling in a high-altitude region. After further investigations, he was diagnosed with high-altitude pulmonary edema (HAPE). During the treatment process, he was urgently transferred to our hospital due to chest pain, and a computed tomography angiography (CTA) confirmed acute type A aortic dissection (ATAAD). The patient underwent an emergency Bentall procedure, total arch replacement, and descending aortic stent placement. The surgery was uneventful. The postoperative recovery was smooth and the patient was discharged in good condition. ATAAD is a critical condition in cardiac surgery, with a low incidence rate but an increasing trend. It is associated with high mortality rates. Acute pulmonary edema is one of the complications of aortic dissection, possibly related to acute aortic valve insufficiency and the release of inflammatory factors leading to pulmonary capillary damage. Acute pulmonary edema will seriously affect lung function, and the resulting hypoxemia will also lead to hypoxia in tissue cells of organs throughout the body, leading to disorder of the body's internal environment. HAPE refers to the patient who has recently arrived at a plateau (generally above 3,000 meters above sea level) and has difficulty breathing at rest, chest tightness, cough, white or pink foamy sputum, and the patient feels general weakness or reduced mobility. The main pathological change of high-altitude pulmonary edema is extensive alveolar edema distributed in patches, and occasionally the formation of hyaline membranes can be seen (this is caused by fibrin deposition in the alveolar edema fluid). HAPE is a non-cardiogenic pulmonary edema specific to high-altitude regions, with severe conditions and high mortality rates. The current understanding suggests that the mechanism of HAPE involves excessive elevation of pulmonary arterial pressure due to hypoxia, increased pulmonary vascular permeability, impaired pulmonary fluid clearance, fluid retention, and fluid transport imbalance. Acute pulmonary edema is a relatively rare presentation of aortic dissection, and the combination of ATAAD and HAPE is extremely rare, posing additional challenges to surgical treatment and anesthesia management.

Keywords: Acute Type A Aortic Dissection, High-Altitude Pulmonary Edema, Acute Pulmonary Edema

1. Introduction

Acute aortic dissection is a rare and life-threatening aortic disease associated with significant morbidity and mortality. The most lethal type is dissection involving the ascending aorta, often referred to as ATAAD. Without immediate surgical repair, patients often die from a range of complications associated with dissection, including aortic rupture, cardiac tamponade, aortic regurgitation, end-organ malperfusion or acute heart failure. ATAAD is a critical condition in cardiac surgery and a cardiovascular emergency that poses a serious threat to human health. It is characterized by a high mortality rate, with a 50%-68% mortality rate within 48 hours if not promptly treated and a 90% mortality rate within three months [1]. Immediate surgical intervention is necessary once ATAAD is diagnosed, with a surgical mortality rate ranging from 12% to 20%. Acute pulmonary edema is one of the complications of aortic dissection and is

a relatively rare manifestation. Its mechanism may be associated with acute aortic valve insufficiency [2].

HAPE is a severe acute mountain sickness, mainly characterized by severe dyspnea and cyanosis. HAPE is a disease caused by a small number of people who have just arrived or returned to plateau. Due to sudden exposure to the hypoxic environment of the plateau, pulmonary artery pressure suddenly increases, pulmonary blood volume increases, pulmonary circulation disorders, and body fluids in the microcirculation leak into the pulmonary interstitium and alveoli. A plateau-specific disease, it is a high-protein, high-exudation edema, characterized by acute onset and rapid disease progression, and the incidence rate increases significantly with the increase in altitude. A major clinical feature of HAPE is the progression from exertional dyspnea to resting dyspnea within a relatively short period of time. Chest CT usually shows characteristic patchy alveolar infiltrates, mainly in the middle right side of the chest, which become more confluent and bilateral as the disease progresses. HAPE is a high-altitude non-cardiogenic pulmonary edema with a severe clinical course and high mortality rates. It is believed that the mechanisms underlying HAPE involve excessive elevation of pulmonary arterial pressure due to hypoxia, increased pulmonary vascular permeability, impaired pulmonary fluid clearance, fluid retention, and fluid transport imbalance.

The combination of ATAAD and HAPE is extremely rare and significantly increases the complexity of treatment.

2. Case Presentation

A 55-year-old middle-aged male patient traveled to a high-altitude region and experienced dyspnea. He was diagnosed with HAPE at a local hospital and received symptomatic treatment such as oxygen therapy. After experiencing chest pain for 5 hours, he was urgently transferred to our hospital. Computed tomography angiography (CTA) of aortic dissection revealed ATAAD (Figure 1A), and chest CT showed diffuse patchy opacities in both lungs, suggestive of lung infection combined with HAPE (Figure 1B). Bedside echocardiography indicated aortic dissection, severe aortic regurgitation, mild mitral regurgitation, and reduced left ventricular systolic function. The patient had a history of hypertension for the past 10 years and was on regular medication. The patient underwent an emergency Bentall procedure, total arch replacement, and descending aortic stent placement. During the surgery, the patient became restless with a blood pressure of 90/52 mmHg, heart rate of 110 beats per minute, and oxygen saturation of 90% under a 3L/min mask oxygen inhalation. Lung auscultation revealed decreased breath sounds with moist rales. Intraoperatively, approximately 100ml of dark red blood was found in the pericardial cavity. The aortic dissection involved the aortic arch, with severe aortic regurgitation and involvement of the ascending aorta, aortic arch, and descending aorta, including the branches of the arch. The surgery lasted for 7.5 hours, with a cardiopulmonary bypass time of 3 hours and 45 minutes. The patient was subsequently transferred to the intensive care unit for further treatment. On postoperative day 10, computed tomography angiography (CTA) of the aortic dissection showed patency of the artificial graft and stent in the aorta (Figure 1C). Postoperative chest CT shows that the diffuse patchy opacities in both lungs are improved compared with before and there is encapsulated pleural effusion in the right chest cavity (Figure 1D). The chest CT indicated improvement of lung infection and pulmonary edema compared to previous images. The echocardiogram report indicates no apparent abnormalities in the functioning of the mechanical aortic valve and smooth blood flow through the artificial ascending aorta. The patient had a smooth recovery and was discharged 15 days after surgery.

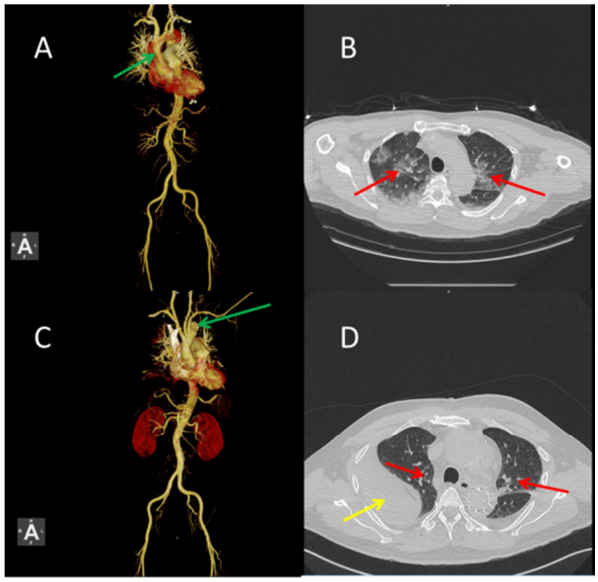

Figure 1. *A: CTA of aortic dissection shows aortic dissection involving the root of the thoracic aorta, thoracic aorta, abdominal aorta, proximal mesenteric artery, and origin of the left common iliac artery (green arrow); B: Chest CT demonstrates diffuse patchy opacities in both lungs with a "butterfly-wing" pattern of changes (red arrow); C: CTA after aortic dissection shows patency of the artificial graft and stent in the aorta (green arrow); D: Postoperative chest CT shows that the diffuse patchy opacities in both lungs are improved compared with before (red arrow), and there is encapsulated pleural effusion in the right chest cavity (yellow arrow).*

3. Discussion

Aortic dissection refers to the tearing of the intimal layer and partial medial layer of the aortic wall, creating a separation in the inner lining. Blood from the aortic lumen enters the medial layer through this intimal tear, subjected to strong hemodynamic forces. As a result, the intima gradually peels off and expands, leading to varying degrees and extents of medial layer dissection and the formation of true and false lumens within the artery. The annual incidence of aortic dissection is approximately 2.6 to 3.5 cases per 100,000 individuals [3, 4]. Before hospital admission, the mortality rate is about 20%, with an overall in-hospital mortality rate that can reach up to 30% [5]. The commonly used

classification system for aortic dissection is the Stanford classification, which divides it into Stanford Type A and Stanford Type B based on whether the ascending aorta is involved. Stanford Type A is the most common form. Without surgical intervention, the mortality rate for ATAAD increases by 1%-2% per hour within the first 24 hours, with a mortality rate exceeding 70% within one week. The main manifestations of aortic dissection are acute chest or back pain [6]. Other symptoms may include syncope, shock, hematemesis, hemiplegia, paraplegia, limb ischemia, or ischemia of vital organs [7-9]. Aortic dissection is an acute aortic disease characterized by rapid onset, high risk, and rapid progression. If left untreated, there is a high risk of rupture and increased mortality rate [10]. Complications of aortic dissection include rupture and fatal bleeding of dissected arterial aneurysms, organ dysfunction, stroke, aortic valve damage or rupture, and cardiac tamponade. Surgery is the main treatment method for aortic dissection. Once diagnosed with Type A aortic dissection, immediate surgical intervention is necessary, with a surgical mortality rate ranging from 12% to 20%.

HAPE is a non-cardiogenic pulmonary edema that occurs as a result of exposure to high altitudes. Its clinical manifestations primarily include dyspnea, cough, pink or white frothy sputum, and cyanosis. Wet crackles can be heard upon auscultation of the lungs. HAPE is a potentially life-threatening condition [11]. The current understanding of the pathogenesis of HAPE involves excessive elevation of pulmonary artery pressure due to hypoxia, increased pulmonary vascular permeability, impaired clearance of lung fluid, fluid retention, and imbalance in fluid transport [12-15]. Diagnosis of HAPE can typically be made based on the patient's medical history and relevant examinations. Treatment mainly involves oxygen therapy and descent to a lower altitude.

The mechanism by which ATAAD causes pulmonary edema can be attributed to various factors. The tearing of the aortic intima leads to the release of a large amount of inflammatory factors, thereby causing damage to pulmonary capillaries and interstitial edema [16]. Acute valvular regurgitation associated with ATAAD may be an important mechanism for the development of acute pulmonary edema. Mitral valve regurgitation directly affects the left atrial and pulmonary venous pressures [17]. Additionally, severe newly developed aortic regurgitation can result in acute left ventricular volume overload, rapid elevation of left atrial pressure, pulmonary venous congestion, and subsequently lead to acute pulmonary edema [2].

In this case, the patient had a history of high-altitude travel and was diagnosed with HAPE. During the treatment process, he also developed ATAAD, which increased the complexity of his treatment. Fortunately, the prognosis turned out to be favorable.

4. Conclusion

ATAAD is a critical illness in cardiac surgery. Although it has a low incidence rate, the mortality rate is extremely high. On the other hand, acute pulmonary edema is a relatively rare manifestation of aortic dissection. The combination of ATAAD and HAPE is exceedingly rare, further adding to the complexity of treatment. Here, we report a rare case of ATAAD complicated by HAPE.

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Conflicts of Interests

The authors declare no conflicts of interest.

References

[1] Apostolakis E, Akinosoglou K. What' s new in the biochemical diagnosis of acute aortic dissection dissection: Problems and perspectives [J]. Med Sci Monit, 2007, 13(8): RA154-158.

[2] Bekeredjian R, Grayburn PA. Valvular heart disease: aortic regurgitation. Circulation. 2005 Jul 5; 112(1): 125-34. doi: 10.1161/CIRCULATIONAHA.104.488825.

[3] Clouse WD, Hallett JW, Schaf HV, et al. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clin Proc. 2004 Feb; 79(2): 176-80. doi: 10.4065/79.2.176.

[4] Melvinsdottir IH, Lund SH, Agnarsson BA, et al. The incidence and mortality of acute thoracic aortic dissection: results from a whole nation study. Eur J Cardiothorac Surg. 2016 Dec; 50(6): 1111-1117. doi: 10.1093/ejcts/ezw235.

[5] Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA. 2000 Feb 16; 283(7): 897-903. doi: 10.1001/jama.283.7.897.

[6] Siddiqui WJ, Arif A, Khan MH, Khan M, et al. An Atypical Case of Silent Aortic Dissection in a Peritoneal Dialysis Patient: A Case Report and Review of Literature. Am J Case Rep. 2018 Jul 27: 19: 880-883. doi: 10.12659/AJCR.909966.

[7] Ohle R, Kareemi HK, Wells G, Perry JJ. Clinical Examination for Acute Aortic Dissection: A Systematic Review and Meta-analysis. Acad Emerg Med. 2018 Apr; 25(4): 397-412. doi: 10.1111/acem.13360.

[8] Khan IA, Nair CK. Clinical, diagnostic, and management perspectives of aortic dissection. Chest. 2002 Jul; 122(1): 311-28. doi: 10.1378/chest.122.1.311.

[9] Mussa FF, Horton JD, Moridzadeh R, Nicholson J, Trimarchi S, Eagle KA. Acute Aortic Dissection and Intramural Hematoma: A Systematic Review. JAMA. 2016 Aug 16; 316(7): 754-63. doi: 10.1001/jama.2016.10026.

[10] Lien WC, Wang CH, Chang WT, Hsu RB, Chen WJ. Aortic dissection presenting with acute pulmonary edema. Am J Emerg Med. 2018 Jul; 36(7): 1323. e7-1323. e9. doi: 10.1016/j.ajem.2018.04.001. Epub 2018 Apr 3.

[11] Pei C, Jia N, Wang Y, Zhao S, et al. Notoginsenoside R1 protects against hypobaric hypoxia-induced high-altitude pulmonary edema by inhibiting apoptosis via ERK1/2-P90rsk-BAD ignaling pathway. Eur J Pharmacol. 2023 Nov 15: 959: 176065. doi: 10.1016/j.ejphar.2023.176065. Epub 2023 Sep 27.

[12] Khan M, Bordes SJ, Murray IV, Sharma S. Physiology, Pulmonary Vasoconstriction. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan. 2023 Apr 17.

[13] Gojkovic M, Darmasaputra GS., Veliça P, Rundqvist H, et al. Deregulated hypoxic response in myeloid cells: A model for high-altitude pulmonary oedema (HAPE). Acta Physiol (Oxf) 2020 Jun; 229(2): e13461. doi: 10.1111/apha.13461. Epub 2020 Mar 16.

[14] Zong HF, Guo G, Liu J, Yang, CZ, et al. Influence of alveolar fluid on aquaporins and Na+/K+-ATPase and its possible theoretical or clinical significance [J]. Am J Perinatol. 2022 Oct; 29(14): 1586-1595. doi: 10.1055/s-0041-1724001. Epub 2021 Feb 21.

[15] Hudalla H, Michael Z, Christodoulou N, Willis G, et al. Carbonic Anhydrase Inhibition Ameliorates Inflammation and Experimental Pulmonary Hypertension [J]. Am J Respir Cell Mol Biol. 2019 Oct; 61(4): 512-524. doi: 10.1165/rcmb.2018-0232OC.

[16] Oz K, Iyigun T, Karaman Z, et al. Prognostic value of neutrophil to lymphocyte ratio and risk factors for mortality in patients with Stanford type A aortic dissection. Heart Surg Forum. 2017 Jun 30; 20(3): E119-E123. doi: 10.1532/hsf.1736.

[17] Koerner MM, Loebe M, Lisman KA, et al. New strategies for the management of acute decompensated heart failure. Curr Opin Cardiol. 2001 May; 16(3): 164-73. doi: 10.1097/00001573-200105000-00002.