

Cardiogenic Shock and Acute Pulmonary Edema Due To a Pheochromocytoma: A Case Report and Possible Mechanism

M. Zhang Xiao¹, R. Lei²

^{1,2}Department of Cardiology, First Affiliated Hospital of Yangtze University, Jingzhou, Hubei PR China
Email address: hxp270@163.com

Abstract—This article analyzed the clinical features and treatment of acute pulmonary edema and cardiogenic shock in association with pheochromocytoma. This case illustrates the treatment for pheochromocytoma spontaneous rupturing and bleeding in association with acute pulmonary edema and cardiogenic shock and possible mechanism. Catecholamine cardiomyopathy and catecholamine crisis is a major cause of acute pulmonary edema, shock. Diagnosis and treatment at early phase is the key for first-aid. Alpha blockers should be added to block the toxic effects of catecholamine and catecholamine should be to correct hypotension or shock, in order to buy time for surgery. Surgery is the most effective and fundamental way to cure pheochromocytoma.

Keywords— Pheochromocytoma; catecholamine; cardiomyopathy; pulmonary edema; shock.

I. INTRODUCTION

Pheochromocytoma is a tumor derived from chromaffin tissue tropism, causing paroxysmal or persistent high blood pressure through secretion and release large amounts of catecholamines, and 25% -50% of pheochromocytoma patients suffered from catecholamine cardiomyopathy [1]. Pheochromocytoma associated with acute heart failure and pulmonary edema is poor prognosis [2]. It will be more critical condition if the patient go into shock at the same time .we have successful rescued a patient who have pheochromocytoma in association with acute pulmonary edema and shock as following.

II. CASE REPORT

A 46-years-old man complained of abdominal obstruction and dyspnea for 1 hour, accompanied by palpitation, sweating, nausea, vomiting but no headache, and we admitted the patient to our department. After ten minutes, the man had one episode of hemoptysis. The amount of hemoptysis was about 200ml/d and at the same time he felt persistent pain in bilateral waist region. Vital signs at presentation were blood pressure 200/120mmHg, heart rate 148bpm He had orthopnea, clammy skin, lips cyanosis and acrocyanosis. On auscultation, inspiratory crackles were heard at both lung bases and pathologic S3 gallop sounds. Abdomen examination revealed tenderness and percussion pain over renal region. There were no abnormal findings in laboratory Data including myocardium enzymogram test and renal function test. ECG revealed tachycardia. According to the clinical manifestations, the patient was diagnosed as hypertension, acute left heart failure and pneumonedema. We take measures included 20mg furosemide and 0.4mg cedilanid intravenoust injection; morphine 6mg dripping into Murphy's tube; nitrate by intravenous drop, adjusted according to blood pressure; dexamethasone 10mg intravenous. After 30 minutes, the blood pressure gradually decreased to 140 /90mmhg, and there is no

significant improvement of heart failure. After 3 hours, the pain intensified over bilateral waist and the right side abdominal muscles became a little tensed, he felt percussion pain and tenderness along the psoas muscle and rebound tenderness over right kidney area. Then blood pressure dropped to 90 / 60mmHg and heart rate is 140-200 beats / min with sweating, restlessness, and stop using nitroglycerin. The blood pressure decreased continually, started using dopamine, metaraminol and rehydration, blood pressure increased to 90/60mmhg after 6 hours, heart failure improved slightly. CT showed: space-occupying lesions in size of 6.1 cm×6.7cm on right adrenal region. Focal tumor and right renal sub capsular hemorrhage and broken into the retro peritoneum. Clinical diagnosis: right adrenal pheochromocytoma spontaneous bleeding and rupturing, catecholamine crisis, acute pulmonary edema, and shock. With continuous intravenous infusion of dopamine and metaraminol, the patient heart failure had a significant improvement after nine hours of admission, his heart rate and blood pressure were normal. The dosage of dopamine and metaraminol gradually reduced after 3 days, and the blood pressure was stable. We transfered the patient to urology surgery department when the patient maintained steady after 1 week and made perfect preparation for surgery. Postoperative pathological diagnosis showed adrenal pheochromocytoma in association with hemorrhage and necrosis. The patient was followed up for 2 years without recurrence.

III. DISCUSSION

Headaches, palpitations and sweating are typical clinical symptoms of pheochromocytoma, and the specificity and sensitivity of this diagnostic method are about 90% [3]. This patients found to have acute heart failure and pulmonary edema at first, then shock because of the tumor spontaneous rupturing and bleeding. And the ruptured tumor led to the release of catecholamine plummeted and induced catecholamine crisis. Catecholamine has a direct harmful

effect on myocardial, leading to partial myocardial necrosis and left ventricular dysfunction [4], and we called it catecholamine cardiomyopathy, clinically manifested as acute left heart failure and pulmonary edema. Mechanism analysis: catecholamines in high concentration caused coronary microcirculation dysfunction, increased coronary spasm and myocardial oxygen consumption, induced myocardial ischemia and hypoxia. Akashi YJ et al [5] reported that catecholamines may cause transient left ventricular dysfunction, weaken apical movement and enhance the movement of the bottom of heart; ECG shows ST-T segment dynamic evolution, and coronary angiography is normal. Those features are in line with the performance of coronary spasm; (2) Overloaded intracellular calcium ion and catecholamine metabolites caused myocardium injury; (3) Catecholamines directly damage lung tissue leading to pulmonary edema. Suga Ket al reported that catecholamines cause postcapillary venule and lymphatic contraction, elevated capillary hydrostatic pressure, increased capillary permeability, leading to alveolar exudate and pulmonary edema; (4) High blood pressure resulted in cardiac afterload overloading; (5) Catecholamines make heart rate fast and shortened the diastolic period of left ventricular. The main reasons of shock: (1) After the tumor rupture, the amount of catecholamines dropped or arrest. Catecholamines has a more long-acting on β -receptor than α -receptor. When vasodilatation caused by α -receptor agonist disappeared, the action of β receptor agonists exist. (2) Catecholamine cardiomyopathy leads to heart failure. (3) High permeability of vascular wall increased plasma extravasation leading to circulating blood volume decline. (4) Catecholamines have long-term effects on β -receptor and α -receptor, resulting in receptor down-regulation. When the release of catecholamines reducing, it caused a longer time to use vasopressor and positive inotropic agents to maintain hemodynamic stability. (5) Tumors had rupture and bleed. It emphasized that we should diagnose early, adjust treatment plan timely. Early diagnosis mainly depends on those phenomenon included blood pressure significantly fluctuating, high blood pressure and shock changing alternately, acute left heart failure and no improvement of pulmonary edema. If those phenomenon cannot account for heart disease, we should be highly suspected pheochromocytoma, and do related examinations [8]. Using alpha blockers to block the toxic effects of catecholamine, supplementing catecholamine to correct hypotension or shock and raising blood volume were the key for successful rescue. We had better use central venous pressure or pulmonary artery wedge pressure as monitoring indicators for blood volume [9], and radial artery pressure as monitoring indicators for arterial pressure we should continuously use phentolamine and dopamine in intravenous to decrease blood pressure fluctuations during peak and valley values in order to remain hemodynamics stable [10]. The patients cannot use alpha blockers for hemorrhage due to the tumors ruptured into the retroperitoneum, so that catecholamine can't release into the blood resulting in a lower blood pressure and we need

continuous supplement catecholamine in intravenous. If the patient have persistent tachycardia (> 120 beats / min) β -receptor blocking agents should be added. We banned to use β -receptor alone without α -blockers or add it earlier than α -blockers [11], emphasizing the use of selective β_1 receptor blocking agents [12]. Surgery is the most effective and fundamental way to cure pheochromocytoma and complications of the disease and reduce mortality from it [13]. If we cure pheochromocytoma at early stage, the secondary hypertension or myocardial damage can be fully restored [14]. Those measures included using alpha blockers and beta blockers and raising blood volume are prepared for operation, but if the tumor ruptured and bleed, it is difficult to maintain circulation stability, and we should do surgery timely [15].

REFERENCES

- [1] P. K. Panda, J. Mokta, K. Mokta, B. Bhardwaj, "Catecholamine-induced cardiomyopathy in pheochromocytoma," *Indian journal of endocrinology and metabolism*, vol. 18, issue 3, pp. 432-433, 2014.
- [2] S. H. Sardesai, A. J. Mourant, Y. Sivathandon, et al., "Pheochromocytoma and catecholamine induced cardiomyopathy presenting as heart failure," *British Heart Journal*, vol. 63, issue 4, pp. 234-237, 1990.
- [3] C. Contargyris, L. Nee, C. Saby, F. Kerbaul, E. Peytel, "Pheochromocytoma revealed by a cardiogenic shock treated by extracorporeal life support," *Annales francaises d'anesthesie et de reanimation*, 2012.
- [4] C. Richard, Stress-related cardiomyopathies, *Ann Intensive Care*, 2011, 1: 39, Published online 2011 September 20.
- [5] Y. J. Akashi, K. Nakazawa, and M. Sakakibara, "Reversible left ventricular dysfunction "takotsubo" cardiomyopathy related to catecholamine cardiotoxicity," *Journal of Electrocardiology*, vol. 35, issue 4, pp. 351-356, 2002.
- [6] A. Kevin, Bybee, and A. Prasad, "Contemporary reviews in cardiovascular medicine: Stress-related cardiomyopathy syndromes," *Circulation*, vol. 118, issue 4, pp. 397-409, 2008.
- [7] K. Suga, K. Tsukamoto, K. Nishigauchi et al., "Iodine-123-MIBG imaging in pheochromocytoma with cardiomyopathy and pulmonary oedema," *Journal of Nuclear Medicine*, vol. 37, issue 8, pp. 1361-1364, 1996.
- [8] F. Xeridat, V. Vidal, J. B. Puech, V. Gariboldi, F. Kerbaul, "A rare cause of cardiogenic shock treated by extracorporeal life support: catecholamine secreting paraganglioma," *Annales Françaises D'anesthésie et de Reanimation*, vol. 30, issue 4, pp. 363-365, 2011.
- [9] S. L. Chew, "Recent developments in the therapy of pheochromocytoma," *Expert Opinion on Investigational Drugs*, vol. 13, issue 12, pp. 1579-1583, 2004.
- [10] A. Adenekan, A. Faponle, T. Badmus, and A. Sanusi, "Anaesthetic management of giant pheochromocytoma in a patient with chronic renal disease," *Journal of the West African College of Surgeons*, vol. 1, issue 2, pp. 112-122, 2011.
- [11] D. Weismann, D. Liu, T. Bergen, M. Peitzsch, A. Raida, M. Wagner, M. Fassnacht, F. Weidemann, and B. Allolio, "Hypertension and hypertensive cardiomyopathy in patients with a relapse-free history of pheochromocytoma,"
- [12] G. R. Lee, P. C. Johnston, A. B. Atkinson, D. McKillop, P. Auld, and S. J. Hunter, "A comparison of plasma-free metanephrines with plasma catecholamines in the investigation of suspected pheochromocytoma," *Journal of hypertension*, vol. 29, issue 12, pp. 2422-2428, 2011.
- [13] S. Das, S. Kumar, M. Nath, and A. P. Bhalla, "Perioperative management of combined surgery for pheochromocytoma and double outlet right ventricle: A rare combination," *Indian journal of Anaesthesia*, vol. 59, issue 6, pp. 378-80, 2015.
- [14] N. Narin, A. Baykan, S. Sezer, et al., "Catecholamine-induced cardiomyopathy and paragangliouroma in a pediatric patient," *Anadolu Kardiyol Derg*, vol. 11, issue 8, pp. 740-744, 2011.
- [15] P. C. Johnston, K. R. Mullan, A. B. Atkinson, F. C. Eatock, H. Wallace, M. Gray, and S. J. Hunter, "Recurrence of pheochromocytoma and

abdominal paraganglioma after initial surgical intervention,” *Ulster*

Medical Journal, vol. 84, issue 2, pp. 102-106, 2015.