

Pheochromocytoma Causing Acute Pulmonary Edema in Association with Cardiogenic Shock: Case Reports and Brief Overview

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Abstract— The article reports cardiogenic shock in association with clinical features of acute pulmonary edema caused by pheochromocytoma.

Keywords— Pheochromocytoma, acute left ventricle, acute pulmonary edema, cardiogenic shock.

I. INTRODUCTION

Pheochromocytoma rare and late-diagnosed catecholamine secreting tumors derived from chromaffin tissue tropism, which may be associated with 25-50% of catecholamine's cardiomyopathies that accounts for about 0.04% cases of hypertension, causing persistent or paroxysmal hypertension through secretion and release large amounts of catecholamine's. Pheochromocytoma associated with acute heart failure and pulmonary edema is poor prognostic sign. It will be more critical condition if the patient goes into shock at the same time. We successfully rescued a patient with pheochromocytoma in association with acute pulmonary edema and cardiogenic shock.

II. CASE REPORT

A previously healthy 48 year old male with chest pain after short exercise with abdominal pain suggestive of abdominal obstruction and dyspnea for an hour, accompanied by palpitation, sweating, nausea, vomiting but no history of headache was admitted to the emergency department our hospital, suddenly after ten minute of admission patient vomited 200ml of blood (hemoptysis) in association with pain over bilateral waist region. The patient also suffered from palpitation after drinking white wine and symptoms relieved after 20 minutes of rest. He also has history of UGI bleeding. On physical examination temperature 38.6⁰, pulse 150bpm, respiration rate 26bpm, BP 210/125mm of Hg. He was in orthopnea, cool clammy extremities with cyanosed skin, lips and acrocyanosis. Lungs fields were dry and moist rales present bilaterally with normal heart borders. On auscultation of heart sound were strong and pathologic S3 gallop sounds with no pathological murmurs heard. Palpation of abdomen were flat and soft, tenderness over epigastrium and right upper quadrant region, on percussion tenderness and pain over flank (renal) region present

Laboratory data are as follow:
Myocardium Enzymogram

| Time | CPK | LDH | CK-MB |
|-----------------|--------|--------|-------|
| At presentation | 115U/L | 325U/L | 52U/L |
| After 6hrs | 150U/L | 300U/L | 43U/L |

Renal function test

| Date | BUN | Cr | UA |
|------------------|------------|-----------|-----------|
| On admission day | 9.35mmol/L | 128umol/L | 350umol/L |

Blood routine examination

| Date | WBC | Neutrophil | L | RBC | HB | PLT |
|------------------|-----------|------------|-----|--------|------------|--------|
| On admission day | 12.0mg/dl | 74% | 23% | 5.4T/L | 17.0 gm/dl | 145G/L |

B-type natriuretic peptide was 10 pg./ml [1.0 ng/l] (< 100)
Random blood glucose: - 250g/dl moderate hyperglycemia
Blood electrolytes were normal in range
EKG showed nodal tachycardia and another three times ECG done no any ST-T segment changes was seen.

The patient was diagnosed to have hypertension with acute left heart failure and pulmonary edema. The patient was given 20mg of furosemide twice intravenously, 0.4mg of cedilanid and added up to 0.2mg in 7hr, Morphine 6mg via Murphy's tube with 50ml of normal saline and intravenous nitrates given via drop adjusted according to blood pressure, aminophylline 0.25mg with 50ml normal saline via intravenous infusion pump set, after 30 minutes intravenous dexamethasone of 10mg given while the systolic blood pressure gradually decreased to 120mmHg and there was no significant improvement of heart failure even after 3hr, the pain intensified over bilateral waist and the right side of the abdominal muscles became a little tensile with pain on percussion, tenderness along with psoas muscle and rebound tenderness over right kidney region. Now the blood pressure dropped to 90/60mm of Hg, HR is 150 to 100 bpm with sweating, restlessness. Suddenly blood pressure 30/0 mm of Hg and nitroglycerin stop using, dopamine, metaraminol and rehydration with normal saline given and blood pressure improved and became 90/60mm of Hg after 6hr, acute heart failure symptoms were improved slightly. Abdominal computed tomography confirmed the presence of a heterogeneous right renal sub-capsular hemorrhage flare up into retro peritoneum, contrast-enhanced right adrenal mass measuring 5.9× 4.0× 6.8 cm in size with focal tumor. Echocardiography revealed normal with slightly impaired left ventricular ejection fraction of 48%. With all the findings

clinically diagnosed as right adrenal pheochromocytoma with spontaneous bleeding and rupturing, catecholamine crisis, acute pulmonary edema and cardiogenic shock. Administration of continuous intravenous infusion with dopamine and metaraminol, the patient had significant improvement of heart failure after nine hours achieved as his heart rate 100 bpm, blood pressure in between 100/60_120/70mm of Hg. Dopamine and metaraminol dosage gradually reduced after three days and blood pressure stabilized. Patient was transferred to urosurgery department, after a week perfect preparation made for surgery. . Successful open adrenalectomy was performed under protective extracorporeal life support; the patient could be weaned off mechanical support rapidly and made a full cardiopulmonary recovery. Postoperative pathological diagnosis showed: adrenal pheochromocytoma in association with hemorrhage and necrosis. The patient was followed up for 2 years without recurrence.

III. DISCUSSION

Catecholamine-secreting tumors may rarely cause an acute adrenergic cardiomyopathy at initial presentation [1], although classic symptoms consist of episodic headache, sweating, palpitation, tachycardia, and paroxysmal hypertension. And the specificity and sensitivity of this diagnostic method are about 90%. In patients with suspected pheochromocytoma fractionated metanephrines and catecholamine's in a 24-hour urinary specimen or occasionally fractionated plasma free metanephrinesshould be measured [3], [4]. The patient found to have acute heart failure with acute pulmonary edema at first, because of spontaneous rupture and bleeding leads to shock. The ruptured tumor leads to release of catecholamine plummeted and induced catecholamine crisis. At an early stage of hemorrhagic tumor stimulates a number of catecholamine to release into the blood, but when it ruptured into retro peritoneum the amount of catecholamine's declines. Catecholamine has a direct harmful effect on myocardium, leading to partial myocardial necrosis and left ventricular dysfunction [5] suggestive of catecholamine cardiomyopathy, clinically manifested as acute left heart failure and pulmonary edema. Mechanism analysis: catecholamine's in high concentration caused coronary microcirculation dysfunction, increased coronary spasm and myocardial oxygen consumption, induced myocardial ischemia and hypoxia. Y. J. Akashi et al [6] reported that catecholamine's 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. (S. H. Sardesai, 1990) Overloaded intracellular calcium ion and catecholamine metabolites caused myocardium injury; (3) Catecholamine's directly damage lung tissue leading to pulmonary edema. K. Suga et al reported that catecholamine's cause postcapillaryvenule 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 catecholamine's 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. 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 Continuous 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 retro peritoneum, 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 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.If we cure pheochromocytoma at early stage, the secondary hypertension or myocardial damage can be fully restored. Those measures included using alpha blockers and beta blockers and raising blood volume are prepared for operation, but if the tumor ruptured and bleeds, it is difficult to maintain circulation stability, and we should do surgery timely.

IV. CONCLUSION

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.

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