Continuous Renal Replacement Therapy for Pheochromocytoma Crisis With Multiple Organ Failure

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Abstract: Pheochromocytoma crisis is a feared and potentially lethal complication associated with excess release of catecholamine from the tumor, which might lead to multiple organ failure (MOF). The definitive treatment for pheochromocytoma is surgical resection. To safely proceed with surgery, hemodynamic stabilization in the acute phase is indispensable, but it might be extremely challenging in case of pheochromocytoma crisis with MOF even if the sufficient pharmacological drugs would be administered. Catecholamine is a dialyzable substance and is removed by renal replacement therapy. In this report, we described 2 cases of pheochromocytoma crisis with MOF, in which we succeeded in controlling acute unstable hemodynamics by means of catecholamine removal with continuous renal replacement therapy. These cases suggest that continuous renal replacement therapy may be effective to manage unstable hemodynamics because of massive catecholamine excretion in patients with pheochromocytoma crisis and MOF.

Key Indexing Terms: Pheochromocytoma crisis; Multiple organ failure; Renal replacement therapy; Catecholamine cardiomyopathy. **[Am J Med Sci 2015;350(6):508–511.]**

Pheochromocytoma crisis is a potentially lethal condition. Acute and rapidly progressive hemodynamic disturbance results from excess release of catecholamine by the tumor, leading to multiple organ failure (MOF).[1] Although the management of patients presenting with pheochromocytoma crisis should include initial stabilization of the acute crisis followed by sufficient alpha blockade before surgery, some patients are resistant to such pharmacological intervention. Catecholamine is a dialyzable substance and can be removed by continuous renal replacement therapy (CRRT).[2] However, there are no reports available on the efficacy of CRRT in patients with pheochromocytoma crisis and MOF. In this report, we described 2 cases of pheochromocytoma crisis with MOF, in which we succeeded in controlling acute unstable hemodynamics by means of catecholamine removal with CRRT.

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CASE REPORTS

Case 1

A 52-year-old man without any significant medical history was emergently admitted to hospital because of palpitations and paroxysmal nocturnal dyspnea over the preceding 3 days. On admission, he was in an extremely activated state with diffuse diaphoresis. His body temperature was 36.0°C. His blood pressure was 150/116 mm Hg, and his heart rate was regular at 140 beats per minute. Blood analysis showed abnormally elevated levels of serum creatinine (Cr; 1.42 mg/dL) and plasma brain natriuretic peptide (BNP; 2,210 pg/mL) without liver dysfunction. Electrocardiography showed sinus tachycardia with nonspecific ST-T changes. Chest x-ray showed cardiomegaly and pulmonary edema. Echocardiography showed diffuse hypokinesis of left ventricular (LV) wall motion (LV end-diastolic dimension, 61 mm; ejection fraction [LVEF], 25%), severe mitral regurgitation and pulmonary hypertension. His condition was diagnosed as congestive heart failure and started continuous infusion of nitroglycerin and furosemide. From the 2nd through the 3rd hospital day, progressive severe renal and liver dysfunction occurred (Cr, 5.6 mg/dL, aspartate aminotransferase, 13,676 IU/L and alanine aminotransferase, 7,090 IU/L). Thereafter, abdominal computer tomography was preformed which showed a left adrenal tumor (76 × 66 mm in diameter) with low-dense areas inside (Figure 1). Endocrine laboratory assessment showed a plasma adrenaline level of 160 (reference range, <0.17) ng/mL, a plasma noradrenaline level of 420 (reference range, 0.15–0.57) ng/mL, a plasma dopamine level of 160 (reference range, <0.03) ng/mL, a urine adrenaline level of 4,480 (reference range, 1–23) μg/d, a urine noradrenaline level of 6,130 (reference range, 29–120) μg/d and a urine dopamine level of 2,300 (reference range, 100–1,000) μg/d ^{123}I-metaiodobenzylguanidine

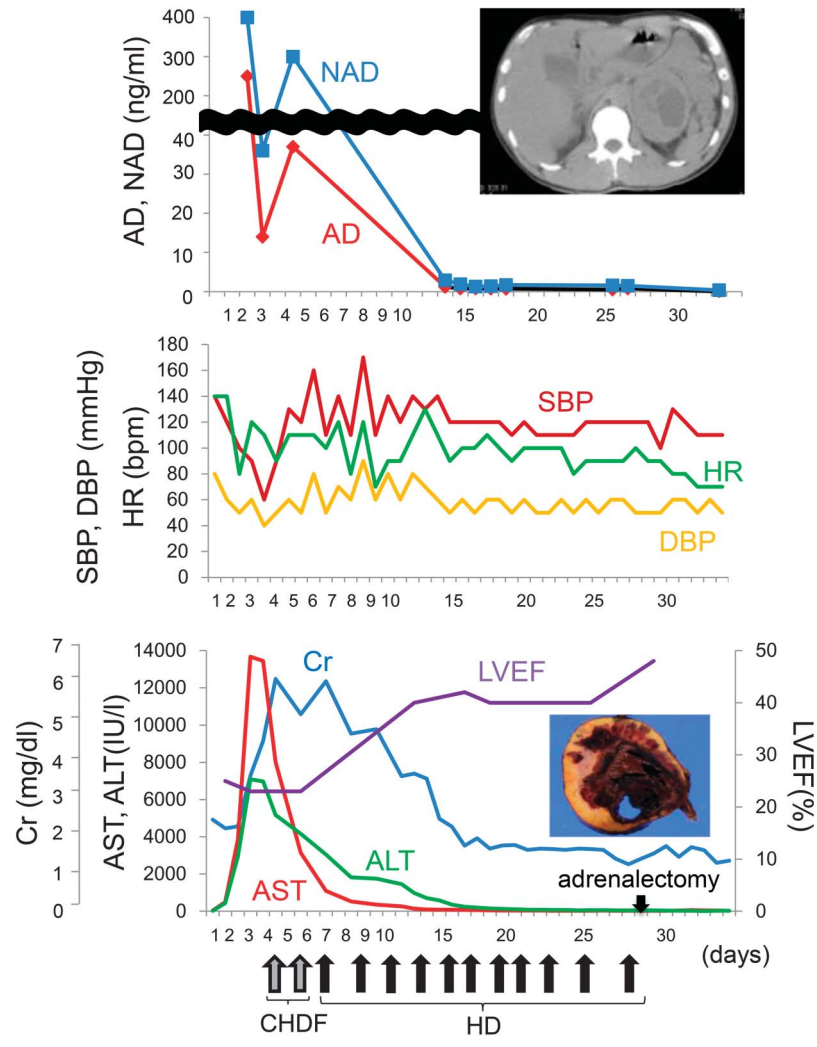

FIGURE 1. The clinical course in case 1 treated with continuous renal replacement therapy for pheochromocytoma crisis with multiple organ failure. AD/NAD, plasma adrenaline/noradrenaline concentration; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CHDF, continuous hemodiafiltration; Cr, serum creatinine level; HD, hemodialysis; HR, heart rate; LVEF, left ventricular ejection fraction; SBP or DBP, systolic or diastolic blood pressure.

scintigraphy showed focal intense radiotracer collection at the left adrenal tumor. His condition was diagnosed as pheochromocytoma and was started on an intravenous phentolamine infusion. Continuous hemodiafiltration (CHDF) and hemodialysis (HD) to manage acute kidney injury (AKI) and remove excessive plasma catecholamine. The postdialyzer catecholamine concentration was decreased when compared with the predialyzer catecholamine concentration (adrenaline, 330–170 ng/mL; noradrenaline, 990–410 ng/mL). His hemodynamic status was stable during HD, but at a few hours after cessation of CHDF/HD, hemodynamic instability relapsed. CHDF/HD was repeated every 2 or 3 days, and he ultimately recovered from MOF and underwent left adrenalectomy on the 29th day. Pathological assessment confirmed the presence of pheochromocytoma with hemorrhagic and necrotic cysts.

Case 2

A previously healthy 30-year-old woman was transferred to hospital because of acute heart failure with suspected pheochromocytoma crisis. One day before admission, she was emergently transferred to the previous hospital because of onset of the sudden abdominal pain. Abdominal computed tomography showed a left adrenal tumor (87 × 77 mm in diameter) with high- and low-dense cystic areas inside. On arrival, her blood pressure was 99/79 mm Hg, and her heart rate was 130 beats per minute and regular. Blood analysis showed renal and liver dysfunction (Cr; 2.35 mg/dL; aspartate aminotransferase, 220 IU/L; alanine aminotransferase, 108 IU/L) and elevated troponin (troponin I, 46.13 (reference range, <0.10) ng/mL) and BNP (2,668 pg/mL). Electrocardiography showed sinus tachycardia with nonspecific ST changes, and echocardiography showed severely depressed LV function (diffuse hypokinesis with LVEF of 15% and LV end-diastolic dimension of 61 mm). The catecholamine concentrations were elevated (plasma adrenaline, 1.90 ng/mL; plasma noradrenaline, 480 ng/mL; urinary adrenaline, 229 μg/d; urine noradrenaline, 9,290 μg/d). It was suspected that her depressed cardiac function was induced by catecholamine cardiomyopathy because of pheochromocytoma crisis and therefore started intravenous phentolamine and propranolol. After a few hours of this treatment, the patient developed frequent nonsustained ventricular tachycardia, and she went into pulseless electrical activity. Cardiopulmonary resuscitation was initiated but spontaneous circulation was not established, so a percutaneous cardiopulmonary support system was used. Emergent coronary angiography showed no abnormality. To remove excessive catecholamine, we performed CHDF, which continued for the subsequent 9 consecutive days (Figure 2). On the 7th day, LVEF improved to 35%, and the