IMAGES IN CARDIOLOGY

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Acute adrenal insufficiency complicated by cardiogenic shock

A 42 year old woman was admitted to the intensive care unit (ICU) for hypotension and confusion. Her medical history comprised autoimmune thyroiditis and Biermer's disease. The patient rapidly developed multiorgan failure requiring mechanical ventilation, fluid loading, and noradrenaline (norepinephrine) infusion. All samples for bacteriological culture were negative. Serum cortisol level was 2 nmol/l (normal range: 170–740). Hydrocortisone and fludrocortisone therapies were initiated. After initial resuscitation, transoesophageal echocardiography was performed because of persistent circulatory failure. It showed diffuse severe left ventricle (LV) hypokinesia (ejection fraction: 35%) without dilatation, low cardiac index (1.3 l/min/m²), and a restrictive mitral profile (panels A and B; supplementary file posted online). Dobutamine was introduced to restore normal blood pressure and stop noradrenaline. On day three, extubation failed, because of severe cardiogenic pulmonary oedema requiring re-intubation. Echocardiography showed persistent left ventricular hypokinesia. A second extubation trial succeeded three days later after large fluid removal by diuretics, and dobutamine was stopped. Myocardial dysfunction had completely reversed on check transthoracic echocardiography performed four days later (panels C and D; supplementary file posted online).

This observation has a significant therapeutic impact. Acute adrenal insufficiency usually involves high output circulatory failure requiring volume

loading and noradrenaline infusion. However, this strategy was deleterious in this case by increasing left ventricular end diastolic pressure and afterload. Normal cardiovascular reactivity to catecholamines requires glucocorticoids for maintenance of vascular tone and cardiac contractility. Glucocorticoids are needed for the synthesis of Na-K adenosine triphosphatase and catecholamines. Furthermore, they induce expression of adrenergic receptors and decrease nitric oxide production, which is involved in myocardial

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contractile impairment. In conclusion, acute adrenal crisis can manifest as reversible cardiogenic shock.

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