

Desvenlafaxine Overdose-induced Toxic Cardiomyopathy and Acute Left Ventricular Failure: A Case Report

Sir,

Desvenlafaxine, a serotonin and norepinephrine reuptake inhibitor (SNRI), is indicated for the treatment of major depressive disorder. Desvenlafaxine is the major active metabolite of venlafaxine. There is limited clinical trial experience with desvenlafaxine succinate overdosage in humans. The most commonly reported events in overdosage include tachycardia, changes in level of consciousness (ranging from somnolence to coma), mydriasis, seizures, and vomiting. Electrocardiogram changes (e.g., prolongation of QT interval, bundle branch block, and QRS prolongation), sinus and ventricular tachycardia, bradycardia, hypotension, rhabdomyolysis, vertigo, liver necrosis, with serotonin syndrome, and death have been reported.^[1]

We report a patient who developed toxic cardiomyopathy with left ventricular (LV) dysfunction and acute LV failure (LVF), with overdose of desvenlafaxine.

CASE REPORT

A 27-year-old male presented to casualty of our hospital with an alleged history of overdose of desvenlafaxine and risperidone. He ingested approximately 1 week's medicine (he was taking desvenlafaxine 150 mg/day and risperidone 1 mg/day). He was admitted in medicine ward for 12 days with diagnosis of toxic cardiomyopathy with LV dysfunction, acute LVF, cardiogenic shock and Acute Kidney Injury (AKI). In the emergency department, his blood pressure was 80/60 mmHg with a pulse of 88 beats/min. The patient had no pallor, pedal edema, cyanosis, lymph node enlargement, or clubbing. He was conscious and oriented to time, place, and person. The patient had no focal neurological deficit. The physical examination including cardiovascular examination was normal. Electrocardiogram (ECG) at admission was normal but on day 2 ECG showed generalized ST-T changes.

Nasogastric tube was inserted and gastric lavage was given. He was given fluid boluses and was started on noradrenaline infusion as his blood pressure remained low. On day 2 his urine output also gradually

started decreasing. An echocardiogram was done in view of the persistence of low blood pressure. Echo showed global hypokinesia with ejection fraction of 30%. He was shifted to medical intensive care unit for further monitoring. The patient was put on noninvasive ventilation as the patient had tachypnea, hypotension and fall in saturation. A possibility of toxic cardiomyopathy was kept. Noradrenaline infusion was stopped and he was started on dopamine and lasix infusion and injection hydrocortisone. The patient was intubated as he was not maintaining saturation on non invasive ventilation. He was extubated the next day. Inotropic support was gradually tapered. Repeat echocardiogram on day 8 showed global hypokinesia with ejection fraction of 40%.

His urea and creatinine were 17 and 0.84 on admission. His creatinine increased to 1.6 on 10/2/17 (day 2). On Day three Urea and Creatinine were 2.1 and 1.31 respectively and increased to 8.2 and 1.75 respectively on February 12, 2017 (Day 4). On February 18, 2017 (Day 10), patient's urea and creatinine returned to 4.8 and 1.28. On day 2, his creatine kinase MB was 50 (Normal Value 0-25 U/L) and Troponin T was 0.119 ng/ml (Positive as normal value is less than 0.1 ng/ml). Creatine phosphokinase was 118.

The patient was discharged in satisfactory condition.

DISCUSSION

Cardiomyopathy is reported with regular doses of venlafaxine (parent molecule of desvenlafaxine). Although the etiological mechanism of Takotsubo cardiomyopathy (TTC) remains unclear, a dramatic increase in catecholamines, such as epinephrine and norepinephrine, has been recognized as a possible cause.^[2,3]

Neil *et al.* reported a case series of six patients who had TTC in association with therapeutic ingestion or overdose of the SNRI venlafaxine or its metabolite desvenlafaxine. Five patients had TTC with venlafaxine, and one patient had TTC with desvenlafaxine.^[4]

Rajapakse *et al.* reported a case of venlafaxine-induced rhabdomyolysis and acute renal failure.^[5]

Risperidone, when taken alone in overdose, causes minimal adverse effects. Tachycardia and dystonic reactions are the main features of risperidone overdose. Significant cardiac and other neurological features seem to be uncommon.^[6] In the outpatient setting, vomiting, diarrhea, poor fluid intake, fever, use of diuretics, and heart failure are all common causes of acute renal failure.^[7]

Our patient had Toxic cardiomyopathy, Acute LVF, Cardiogenic Shock and AKI. It is difficult to comment whether AKI was due to overdose of desvenlafaxine or secondary to cardiac events .

Until now, no case report has reported toxic cardiomyopathy, acute LVF and cardiogenic shock in the same patient with desvenlafaxine overdose.

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Conflicts of interest

There are no conflicts of interest.

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