Endocrinological abnormalities and Takotsubo cardiomyopathy

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Dear Editor,

We read with much excitement in the article "Takotsubo syndrome and pheochromocytoma: an insidious combination" published by Maffé *et al.* in your esteemed journal. The authors have discussed in detail a 53-year-old gentleman who succumbed to Takotsubo syndrome in the background of a catecholaminergic storm secondary to ruptured pheochromocytoma following a fall [1]. We feel this topic is relevant and have the following addendum.

Takotsubo cardiomyopathy (TTC), also known as stress cardiomyopathy is traditionally thought to be treated by physical and emotional events. Over the past few years, there have been increasing reports of various endocrine abnormalities precipitating TTC. Table 1 shows the spectrum of endocrine abnormalities presenting with TTC as reported in Medline [2,3]. While pheochromocytoma and paraganglioma directly cause TTC secondary to catecholaminergic surge, the other endocrine abnormalities result in an augmentation of the catecholaminergic response through various mechanisms like i) exaggerated reactivity to stress during an autonomic surge in absence of cardioprotective peptides like estrogen, ii) potentiation of the effect of the normal level of catecholamine by thyroxine, iii) increased sensitivity to catecholamines in presence of steroid hormones [2].

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We agree with the authors that patients with TTC usually have a good prognosis, with reported recovery in more than 95% of patients. However, in recent literature, various poor prognostic markers have been reported. Male gender, acute physiological,

Table 1. Showing the various reported endocrine causes precipitating Takotsubo cardiomyopathy.

Thyroid disorder	Thyrotoxicosis • Endogenous (graves, Hashimoto) • Exogenous (radio iodine, iatrogenic thyroxine, porcine thyroxine) • Thyroid storm Hypothyroidism Amiodarone induced hyperthyroidism Post total thyroidectomy Obstructive goiter
Adrenal disorders	Deficiency • Primary adrenal insufficiency • Secondary adrenal insufficiency • Addisonian crisis • Excess • Adrenal adenocarcinoma • Conns
Catecholamine producing tumors	Pheochromocytoma Paraganglioma MEN 2 A
Diabetes mellitus	DKA HONCC Hypoglycemia
Pituitary	Pituitary adenoma Pituitary apoplexy Empty sella ACTH deficiency Sheehan syndrome (all hormones) Acromegaly Cushing syndrome TSH secreting adenoma SIADH (Hyponatremia)
Others	APS Menopause Drugs: • Levothyroxine • Terlipressin • Indapamide • Water (Psychogenic polydipsia) • Venlafaxine • Triamterene

MEN2A, Multiple endocrine neoplasias; DKA, Diabetic ketoacidosis; HONC, Hyperosmolar nonketotic coma; ACTH, Adrenocorticotrophic hormone; TSH, Thyroid-stimulating hormone; SIADH, Syndrome of inappropriate ADH secretion; APS, Autoimmune polyglandular syndrome.

psychological stress, severe left ventricular dysfunction (LVEF <45%), acute neurological pathology, and extremely high troponins (>10 times from baseline) are associated with poor in-hospital outcomes, most of which were present in the reported patient [4]. Reported markers for long-term poor prognosis have been older age, reduced blood ventricular ejection fraction (LVEF <45%), presence of cardiogenic shock, presence of atrial fibrillation, or neurological pathologies [4,5].

Finally, nitro derivatives, which were used in the mentioned patient for reducing blood pressure can worsen pre-existing left ventricular outflow tract obstruction and hence requires extreme caution in patients with TTC.

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