

Sheehan's syndrome with cardiac arrest: A case report and review of the literature

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Submitted: 2014-02-19 Accepted: 2014-03-11 Published online: 2014-09-28

Key words: Sheehan's syndrome; hypoglycaemia; serum potassium; cardiac arrest

Neuroendocrinol Lett 2014; **35**(5):352-354 PMID: 25275267 NEL350514C01 © 2014 Neuroendocrinology Letters • www.nel.edu

Abstract

A 62-year-old woman was admitted to our hospital because of unconsciousness and hypoglycaemia. She had a history of weakness and fatigue after postpartum haemorrhage in 1983. Unfortunately, she was not diagnosed with Sheehan's syndrome and did not receive sufficient professional treatment due to the limited medical resources in her hometown. A laboratory examination at the local hospital revealed severe hypoglycaemia (1.8 mmol/L) with relatively low serum potassium (2.9 mmol/L). The woman appeared twice in the hospital with cardiac arrest, and her medical history, and the later laboratory investigations were consistent with Sheehan's syndrome. Empty sella was also found by magnetic resonance imaging (MRI). We conclude that Sheehan's syndrome may lead to cardiac arrest with the complication of hypokalaemia and deserves the vigilance of clinicians.

INTRODUCTION

Sheehan's syndrome, which is defined by pituitary necrosis after severe postpartum haemorrhage and hypovolaemia, may cause hypopituitarism. A variety of stresses, such as infection, diarrhea, vomiting, dehydration, hunger, cold, acute myocardial infarction, cerebrovascular accident, surgery, trauma, anaesthesia and the use of sedatives, hypnotics and hypoglycaemic agents can induce pituitary crisis. The prevalence of Sheehan's syndrome was estimated to be 5.1 per 100,000 women in a retrospective nationwide investigation (Kristjansdottir *et al.* 2011). However, the pathogenesis of Sheehan's syndrome is not completely clear. Although anterior pituitary necrosis occurs rapidly, some clinical symptoms of pituitary dysfunction may appear and intensify in the years after necrosis. Clinical manifestations in women with Sheehan's syndrome have been reported to develop

at a variety of intervals ranging from one month to 47 years after delivery (Slee & Rensma 1990). Most patients present with either complete or partial hypofunction of the anterior pituitary (Laway *et al.* 2009; Kelestimur 2003). The clinical manifestation of Sheehan's syndrome can be expressed as weakness, nausea, vomiting, diarrhoea, oedema, bradycardia, dizziness and fatigue. The severity of the disease depends on the degree of tissue destruction (Laway *et al.* 2011; Gokalp *et al.* 2011; Sert *et al.* 2003). A medical history of postpartum haemorrhage and laboratory tests are helpful clues for the diagnosis. Here, we report a case of Sheehan's syndrome that presented with cardiac arrest.

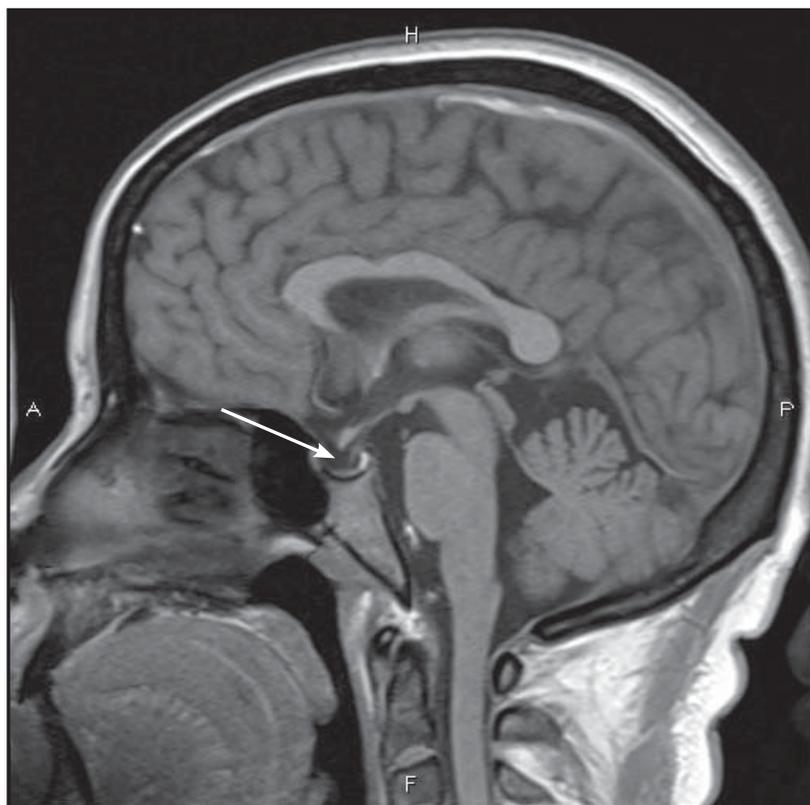
CASE REPORT

The patient, a 62-year-old woman, 150 cm in height and 50 kg in weight, was hospitalised at a local hospital for cough, expectoration and vomiting on October 1, 2013. She lost consciousness on October 12, 2013. Examination revealed random

blood glucose 1.8 mmol/L, serum sodium 127.0 mmol/L and serum potassium 2.9 mmol/L. Replacement therapy was instituted with glucose and glucocorticoid; however, as the patient did not recover consciousness, she was admitted to our hospital for coma on October 13, 2013. The general physical examination showed that her blood pressure, heart rate and temperature were within normal limits. She was comatose and appeared pale, with dryness and furfuration of the skin. Cardiac arrest suddenly occurred within 4 hours of admission. Chest compressions were immediately administered, and spontaneous cardiac rhythm recovered approximately two minutes later. Shortness of breath, tachycardia occurred 1 hour later; lidocaine 50 mg via intravenous bolus was immediately administered. Her heartbeat dropped gradually, and sighing respiration appeared; as a pulse could not be detected by touch, chest compressions were given at once, and autonomic heart rhythm was restored one minute later. She was referred to the ICU for further treatment five hours after arrival at our hospital. Routine laboratory findings demonstrated a red blood cell count $3.69 \times 10^{12}/\mu\text{L}$, haemoglobin 109 g/L, random blood glucose 9.8 mmol/L, serum sodium 139.0 mmol/L, uric acid 581 $\mu\text{mol/L}$, aspartate transaminase (AST) 4704 U/L, total protein (TP) 60.6 g/L, albumin (ALB) 35.8 g/L, creatinine 168 $\mu\text{mol/L}$ and brain natriuretic peptide (BNP) 3070 ng/L. Routine urine and routine stool were normal. Electrocardiogram (ECG): T wave change. Transthoracic echocardiographic findings showed mild increase in the left ventricular and right atrium, moderate mitral and tri-

cuspid regurgitation and a left ventricle ejection fraction (LVEF) of 50%. The patient lost consciousness suddenly due to ventricular fibrillation at 5 hours after arrival in the ICU. Chest compressions and defibrillation were given immediately. The heart rhythm converted into ventricular tachycardia first and then sinus rhythm; consequently, consciousness and pupillary light reflex were recovered. The patient had no past history of any heart illness. Retrospective questioning of close family members revealed that the patient appeared weak and fatigued after postpartum haemorrhage in 1983. Unfortunately, she was not diagnosed with Sheehan's syndrome and did not receive sufficient professional treatment due to the limited medical resources in her hometown. Her hormonal profile revealed low levels of serum T3 (0.554 nmol/L), FT3 (1.74 pmol/L), T4 (24.94 nmol/L) and FT4 (4.56 pmol/L), normal levels of cortisol (60 nmol/L), pituitary prolactin (3.33 ng/mL) and ACTH (<5 pg/mL) and a high level of serum TSH (8.22 mIU/L). Magnetic resonance imaging (MRI) of the pituitary revealed evidence of empty sella (Figure 1). Thus, the patient was diagnosed with pituitary crisis (low blood sugar type) related to Sheehan's syndrome. The patient's condition gradually improved after glucocorticoid and sodium levothyroxine replacement therapy. Her combined pituitary stimulation test (TRH, GnRH, insulin hypoglycaemia) results were below normal. She was transferred to the endocrinology ward on October 16, 2013 and was discharged on October 30, 2013.

Fig. 1. MRI pituitary sagittal view showing pituitary fossa filled with cerebrospinal fluid and the stalk touching the atrophic pituitary at the base (white arrow) suggestive of partially empty sella.



DISCUSSION

The entity known as Sheehan's syndrome was first proposed by Sheehan in 1937 to be caused by postpartum haemorrhage (Pasa *et al.* 2010; Kaplun *et al.* 2008; Tessnow & Wilson 2010; Lee & Moon 2011). Sheehan's syndrome remains a serious health problem in developing countries and is characterised by varying degrees of anterior pituitary dysfunction. The diagnosis of Sheehan's syndrome is established with the help of medical history and physical findings consisting of a puffy face, fine wrinkles around the eyes, sparse hair in the armpit and pubic regions, breast atrophy, hypotensive vascular collapse, stupor or myxoedema coma. Early clinical manifestations, such as fatigue, hair loss, dizziness and other symptoms, are not typical; later-appearing symptoms include amenorrhoea, achalasia and other signs of gonadal axis impairment. Sudden death and fatal arrhythmia are occasionally reported (Iga *et al.* 1992), but cardiac arrest has rarely been reported. Köpp *et al.* (2010) reported a patient with Sheehan's syndrome who refused any intensive-care treatment and died from toxic cardiac arrest in 2010. The patient in our case underwent cardiac arrest twice in the course of the disease, with restored sinus rhythm after chest compressions and defibrillation, and then recovered and was discharged with hormone replacement therapy. Severe hypoglycaemia can cause numerous cardiac arrhythmias, including premature ventricular contractions, tachycardia and high-degree heart block, and potassium supplementation can limit hypoglycaemia-associated death (Reno *et al.* 2013). However, as the patient's blood glucose levels were always in the normal range after admission, it was considered that the hypokalaemia might have been related to the cardiac arrest. The significant clinical manifestation of nausea, vomiting, diarrhoea and other gastrointestinal symptoms before admission can cause hypokalaemia, and glucose supplementation can increase the severity of hypokalaemia because it can cause the influx of potassium ions into cells. The changes in T wave on the electrocardiogram and serum potassium level (K 2.86 mmol/L) confirmed the assumption that it was hypokalaemia that led to the occurrence of cardiac arrest. Groth KA reported that diarrhoea could cause a life-threatening situation due to the excretion of potassium, ultimately causing cardiac arrest due to hypokalaemia (Groth *et al.* 2012). The patient no longer exhibited malignant arrhythmia and cardiac arrest after the serum potassium level was reversed by rapid bolus potassium injection. Although Ruisz *et al.* (2013) reported one case of severe hypokalaemia (1.1 mmol/L) without cardiac arrest, this trend in serum potassium (2.86 mmol/L) level would not be sufficient to cause fatal arrhythmia. It is possible that there might be a more serious low extracellular serum potassium level at some point in the course of the disease, along with structural and functional changes in the heart that promoted the occurrence of

cardiac arrest at the low serum potassium level. Struck *et al.* (2011) reported one case of perioperative cardiac arrest (ventricular fibrillation) of a patient undergoing elective orthopaedic surgery due to moderate hypokalaemia (serum potassium 2.8 mmol/L).

In conclusion, although Sheehan's syndrome leading to cardiac arrest is very rare, clinicians should be aware that severe hypokalaemia due to vomiting, diarrhoea and glucose supplementation for hypoglycaemia can lead to cardiac arrest. Potassium chloride should be given in a timely manner by rapid bolus injection to avoid the occurrence of arrhythmia.

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