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Case Report

A case of fatal heart and liver failure accompanied by thyroid storm treated with prompt plasma exchange

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ABSTRACT

A 36-year-old man with a history of Graves' disease was admitted complaining of dyspnea. He was diagnosed with acute heart failure and severe liver dysfunction accompanied by thyroid storm. Left ventricular ejection fraction was 19%, and liver enzyme levels were markedly elevated followed with coagulation disorders. In addition to the conventional therapy, we performed plasma exchange emergently. Thyroid hormone levels promptly normalized, then his clinical condition improved. Finally, his cardiac and liver function almost normalized from a fatal condition without serious complications. Hyperthyroidism can cause myocardial and liver injury, hence thyroid hormone removal in acute phase is important. Prompt plasma exchange is effective in the acute phase for heart and liver failure accompanied by thyroid storm.

<**Learning objective:** Thyroid storm is a life-threatening condition. Prompt reduction of serum free thyroid hormone is important in fatal conditions. Because plasma exchange (PE) can decrease serum thyroid hormone and improve critical condition, PE should be conducted emergently. In the present case, we promptly performed PE for the patient with potentially fatal heart and liver failure. We could treat him without any complication. We wish to emphasize the importance of prompt PE in acute phase of thyroid storm.>

Introduction

Thyroid storm is a morbid state, characterized by elevated circulating thyroid hormones levels leading to multiple organ failure [1]. It sometimes occurs in patients with untreated or inadequately treated hyperthyroidism and is usually triggered by physical stress. The frequent manifestations are severe circulatory, hepatic, gastrointestinal, and neurological dysfunction. As one or more organ systems are decompensated, it becomes a life-threatening condition. Therapy should not be delayed because of the high mortality rate (10–30%) [2]. Conventional therapy for thyroid storm consists of beta-blockers, antithyroid drugs, iodine, and glucocorticoids. Therapeutic plasma exchange (PE) is an alternative treatment that was proposed in the 1970s for hyperthyroidism including thyroid storm, regardless of etiology [3]. With threatening health conditions, PE should be conducted early without confirming the conventional treatment to be effective, because it is the fastest known method to improve the critical condition [1]. In our case, we started PE just after the transfer to our hospital, and could save his life.

Case report

A 36-year-old man who was previously diagnosed with Graves' disease and atrial fibrillation two years previously discontinued his medication. Several days earlier, he had caught a cold and was admitted to a local hospital due to dyspnea. He also had fever, edema, and diarrhea. On physical examination, his height was

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171 cm, and his weight was 95.5 kg. His Japan coma scale level was I-2. Body temperature was 37.8 °C, blood pressure was 84/66 mmHg, and heart rate was 112 beats/min. Coarse crackles were audible, and cardiac extra sounds and systolic murmur (Levine 3/6) at the apex were confirmed. The thyroid gland was diffusely enlarged without pain, and jaundice and leg edema were observed. The endocrinological examination showed elevated serum free thyroxine and triiodothyronine levels with low serum thyroid stimulating hormone (TSH) levels and high TSH receptor antibody titers. Transthoracic echocardiography showed left ventricular dilatation and a left ventricular ejection fraction (LVEF) of 19%. He was diagnosed with heart failure accompanied by thyroid storm and treated with diuretics, methimazole 80 mg divided over 4 times/day, potassium iodide 200 mg/day, and prednisolone 30 mg/day. Despite initiation of medical treatment, his condition deteriorated, and he became anuric. Then, he was transferred to our hospital on the second hospital day. Laboratory evaluation indicated multiple organ failure including heart failure, severe liver dysfunction, acute renal failure, and respiratory failure (Table 1). Marked elevation of liver enzyme levels and coagulation disorders were confirmed. Negative hepatitis virus and autoantibody test results indicated that severe acute liver failure was accompanied by thyroid storm. An electrocardiogram showed atrial fibrillation, at 150 beats/min (Fig. 1A). Chest radiography showed bilateral pleural effusions and pulmonary congestion (Fig. 1A). The cardio-thoracic ratio was 67%. His disturbed consciousness worsened, and he was intubated. We performed direct current cardioversion, and his rhythm recovered to sinus tachycardia, but immediately returned to atrial fibrillation. He was treated with landiolol and inotropic agents in addition to the drugs already mentioned (Fig. 2). Then, we emergently performed PE to remove the thyroid hormones and improve the hemodynamics and organ failure. To compensate for metabolic acidosis and remove congestion, continuous hemodiafiltration (CHDF) was simultaneously performed. On the next day, both serum aspartate aminotransferase and alanine aminotransferase levels were markedly decreased (Fig. 2). The serum free thyroxine levels decreased from 2.40 ng/dL to 2.14 ng/dL, with elevated TSH levels (from <0.004 μIU/mL to 0.259 μIU/mL). His clinical condition had also improved, and we stopped the PE with no side effects. CHDF was continued until the improvement of metabolic acidosis and fluid retention with adequate amount of urine excretion (on hospital day 6). Inotropic agents were ceased, and he was extubated two days after the termination of CHDF. Although his condition was improving, total bilirubin levels increased gradually. Although we discontinued methimazole which is known to induce hyperbilirubinemia, his consciousness had not fully recovered. Electroencephalography showed a slow wave, which was consistent with metabolic encephalopathy. Total bilirubin levels peaked at 28.0 mg/dL (direct bilirubin of 23.0 mg/dL) on hospital day 16; then, it gradually decreased. The serum free triiodothyronine and thyroxine levels were also normalized. His consciousness finally recovered. After confirmation of no thrombosis at the left atrium appendage via a transesophageal echocardiogram, we performed direct current cardioversion again. Then, he recovered to sinus rhythm (Fig. 1B). Chest radiography showed no bilateral pleural effusion and pulmonary congestion (Fig. 1B), and the LVEF recovered to 50%. We did not find delayed myocardial enhancement in cardiac magnetic resonance imaging. We could treat him without any major complication except muscle weakness due to the long intensive care unit stay. He was transferred to the previous hospital for physical rehabilitation on hospital day 61.

Discussion

Triiodpthyronine increases cardiac inotropy and chronotropy, and decreases systemic vascular resistance. While hyperthyroidism is known to cause high output cardiac failure, it also reduces LVEF at a rate of 3% [4]. Because left ventricular systolic dysfunction associated with thyrotoxicosis improves after getting euthyroid, prompt reduction of thyroid hormone is important in potentially fatal conditions. PE is a short-term treatment that could reduce plasma protein-bound thyroid hormones and TSH receptor antibodies [5]. Each PE session is reported to decrease serum free triiodothyronine and free thyroxine concentrations by 30–50% [6]. However, because the thyroid hormone is present not

Table 1 Laboratory data on admission.

WBC	30,900	/μl	Na	131	mEq/l
RBC	509	$\times 10^4$/μl	K	5.3	mEq/l
Hb	14.2	g/dl	Cl	94	mEq/l
Ht	46.3	%	BUN	36.8	mg/dl
Plt	17.5	$\times 10^4$/μl	Cr	3.95	mg/dl
APTT	47.5	sec	TC	69	mg/dl
PT	<5	%	HDL-C	32	mg/dl
PT-INR	8.26		TG	25	mg/dl
D-dimer	25.5	μg/ml	CK	277	IU/l
TP	5.7	g/dl	CK-MB	34	IU/l
Alb	3.7	g/dl	PG	163	mg/dl
T-Bil	5.9	mg/dl	CRP	3.89	mg/dl
AST	9258	IU/l	BNP	314.2	pg/ml
ALT	3550	IU/l	Tn-I	0.16	ng/ml
LDH	10,812	IU/l	TSH	<0.004	(0.35–4.94; μIU/ml)[a]
ALP	385	IU/l	FT3	2.6	(1.71–3.71; pg/ml)[a]
γ-GTP	48	IU/l	FT4	2.4	(0.70–1.48; ng/dl)[a]
			TRAb	7	(<1.0; IU/l)[a]
Blood gas analysis (nasal O_2 3L)					
pH	7.27		HCO_3^-	6.9	mmol/l
pCO_2	15.6	mmHg	BE	−18.6	mmol/l
pO_2	104	mmHg	Lac	14.5	mmol/l

δGTP, δ-glutamyl transpeptidase; Alb, albumin; ALP, alkaline phosphatase; ALT, alanine-aminotransferase; APTT, activated partial thromboplastin time; AST, aspartate-aminotransferase; BE, base excess; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; CK, creatinine kinase; CK-MB, creatinine kinase-muscle-brain isozyme; Cr, creatinine; CRP, C-reactive protein; freeT3, free triiodothyronine; freeT4, free thyroxine; Hb, hemoglobin; HDL-C, high-density lipoprotein cholesterol; Ht, hematocrit; Lac, lactic acid; LDH, lactate dehydrogenase; PG, plasma glucose; Plt, platelets; PT, prothrombin time; PT-INR, prothrombin time international normalized ratio; RBC, red blood cells; T-Bil, total bilirubin; TC, total cholesterol; TG, triglycerides; Tn-I, troponin I; TP, total protein; TRAb, thyrotrophin receptor antibody; TSH, thyroid-stimulating hormone; WBC, white blood cells;

[a] Numerals in parentheses are normal values.

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