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Original Article

Endovascular cooling combined with plasmapheresis to treat thyroid crisis patient: a case report

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Abstract: This article describes a 22-year-old patient with multiple organ dysfunction syndromes due to hyperthyroidism crisis, invalid to conventional drugs and physical cooling treatment. Then the intravascular heat exchange cooling technique combined with plasmapheresis was applied, and the patient's body temperature and elimination of the thyroid hormone was effectively controlled. Finally, the patient was rescued successfully. We conclude that the intravascular heat exchange cooling technique combined with plasmapheresis may be a therapeutic option for hyperthyroidism crisis patients who do not respond to conventional treatment.

Keywords: Thyroid crisis, intravascular heat exchange cooling technique, plasmapheresis

Introduction

Thyroid crisis is a rare, severe and life-threatening exacerbation of thyrotoxicosis, characterized by dysfunction of the thermoregulatory, central nervous, astrointestinal-hepatic and cardiovascular systems [1, 2]. Therefore, thyroid crisis can cause a high death rate, may approach 20-30% [3]. Historically, medical management has focused on supportive measures and medications that act to halt the synthesis, release and peripheral effects of thyroid hormone. Hence, the acute phase must be treated with standard treatments such as thiamazole, prednisolone and nonselective beta-blockers [4]. However, antithyroidal therapy is sometimes limited due to rare and serious side effects or failure to control disease progression, leaving few treatment alternatives to choose.

In recent years, the intravascular heat exchange cooling technique and plasmapheresis are often used in Critical Care. The intravascular heat exchange cooling technique has been effectively and safely used to induce hypothermia following cardiac arrest [5-7] and infants with moderate or severe hypoxic-ischemic encephalopathy [8], and also successfully to treat fever in neurological cases where the person is critically ill [9]. Plasmapheresis has been used as an important treatment modality in various neurological, hematological, and rheumatologic diseases and plasmapheresis were also successful used in patients with thyroid crisis which was reported previously in literature [10, 11].

The following case describes an adult woman with thyroid crisis that was failed to respond conventional medical management. However, the patient responded well to the intravascular heat exchange cooling technique combined with plasmapheresis, which resulted in clinical and biochemical stabilization of her disease processes. The treatment option of the intravascular heat exchange cooling technique combined with plasmapheresis leads to amelioration of symptoms and a significant decline in thyroid hormone levels. We used this system for an individual with multiple organ dysfunction syndromes due to thyroid crisis. To our knowledge, this may be the first time to use such a treatment strategy in the world. Here we introduced this procedure as a new method to treat the patients with thyroid crisis.

Case report

The patient is a 22-year-old young woman, who has a hyperthyroidism history for about 1 year. She started to use antithyroid drugs after diag-

Table 1. Condition assessment during the course of the patient's illness

Test / Day	1	2*	3#,&	4	5	6	7
FT3 (pmol/L)	24.20	12.31	22.74	3.65	3.8	6.62	6.23
FT4 (pmol/L)	>78.13	>78.13	>78.13	45.64	30.28	32.02	27.07
TSH (mIU/L)	0.56	0.37	0.54	0.01	0.06	0.01	0.01
Lactic acid	5.1	8.6	11.6	3.2	0.81	0.30	0.21
Tmax (°C)	38.0	39.0	39.6	35.0	36.1	36.5	37.0
HRmax (beats/min)	160	175	182	78	75	82	86

*2014.12.13 4 pm transferd to ICU. #2014.12.14 11 am intravascular heat exchange cooling technique. &2014.12.14 3 pm plasmapheresis.

nosis, but not regularly during the later period. There is no symptom such as heart palpitations, chest tightness, sweating, nausea, and vomiting, skin yellow dye and so on. In the absence of obvious incentives, she suddenly appeared coma. She was admitted to a progressive care unit for management of coma. On examination, the patient had a temperature at 38°C, heart rate at 160 times/min, respiratory rate at 27 times/min. Computed tomography (CT) of the head showed a cerebral edema without hemorrhage or infarction. Electrocardiogram showed supraventricular tachycardia. The free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH) levels see **Table 1**.

The patient was given mannitol, propranolol (200 mg Q8 h), propylthiouracil (20 mg Q6 h), hydrocortisone sodium succinate (200 mg Q8 h), surface cooling and supportive therapy (day 1). The patient has no improvement, she was still unconsciousness, and the temperature reached 39°C, the heart rate reached 175 times/min. The patient was found to be in thyroid crisis. At this time, the patient became short of breath and was transferred to the intensive care unit for further care (day 2). In the condition of high-flow oxygen treatment (5 L/min), the patient's partial pressure of oxygen is 68 mmHg.

Upon arrival, oral trachea cannula and sedation with Propofol were performed. Breath-holding was performed using a ventilator with Ventilator Intermittent Positive-Pressure Ventilation (IPPV) mode. Ventilator parameters were set at 450 mL tidal volume, 4 cmH_2O positive end-expiratory pressure (PEEP), 45% fraction of inspiration O_2 (FiO_2), 20 times/min frequency. Continual measured oxygen saturation was >or = 98%. And the treatment of glucocorticoid, antithyroid drugs and ice blanket cooling were continued.

The temperature at 8:00 AM was 39.6°C on hospital day 3. In order to control body temperature, decrease persistently high temperature that causes elevated metabolic rate and increased oxygen consumption and prevent deterioration in organ function, the decision was made to initiate the intravascular heat exchange cooling technique. A vascular heat exchange catheter was placed in the patient's right femoral vein and mild systemic hypothermia was immediately started. It took 2 h to reduce the core temperature to below 38°C and a further 5 h to reduce it to the target 35.0°C, where it was effectively maintained.

The concentration of FT3 was 22.74 pmol/L in the same morning. In view of overall deteriorating, a temporary double-lumen catheter was placed in the patient's left femoral vein and plasmapheresis was immediately started to eliminate thyroid hormone which concentrations were very high in plasma. The plasmapheresis treatment was administered using 2.0 litres fresh frozen plasma (FFP).

On the third day of mechanical ventilation (hospital day 4), the patient became conscious. The body temperature was maintained at 36°C. Heart rate was around 60 beats/min (range 50 to 78 beats/min). Repeated thyroid examination showed an evident decrease with FT3 and FT4 (see **Table 1**). On hospital day 5, the patient successfully weaned. On hospital day 6, the use of intravascular heat exchange cooling technique was stopped for a total of 72 h. After cessation of treatment her temperature returned to normal with acceptable fluctuations.

The patient was transferred to general endocrinology wards on hospital day 7 to continue the treatment with glucocorticoid and antithyroid drugs. Subsequently, the thyroid hormone has been controlled in a relatively ideal level. On the 23 day, the patient was discharged from hospital.

Discussion

Thyroid crisis is a rare, severe and life-threatening exacerbation of thyrotoxicosis, characterized by dysfunction of the cardiovascular systems, gastrointestinal-hepatic, central nervous and thermoregulatory [12, 13]. These dysfunctions are probably due to the excess of thyroid hormone. The diagnosis of thyroid crisis was determined on the basis of the Burch-Wartofsky score, which is widely used for evaluation of the severity of thyrotoxicosis [13, 14]. A score of 45 or greater is highly suggestive of thyroid crisis. In this case, clinical findings showed a total score of 85 or more, which is highly suggestive of definite thyroid crisis. The exact mechanisms underlying the development of thyroid crisis from uncomplicated hyperthyroidism are not well understood. A heightened response to thyroid hormone is often incriminated along with increased or abrupt availability of free hormones and enhanced binding to thyroid hormone receptors [3, 12, 15]. In order to eliminate excessive thyroid hormone in the blood plasma, we applied not only the conventional drug treatment but also the plasmapheresis to this case.

Plasmapheresis has been used in a variety of illnesses to remove harmful plasma constituents rapidly or to decrease the concentrations of antibodies, immune complexes and toxins [16]. It has been utilized for treatment of myasthenia gravis [17], hepatic coma [18] and rheumatoid arthritis [19]. During plasma exchange, the patient's plasma is extracted from the components of the blood, and instead of ordinary frozen plasma, which contains abundant albumin is infused back to the patient [20]. During this procedure, thyroxine binding globulin (TBG), with bound thyroid hormones, is removed from the plasma. Then the colloid replacement (eg, albumin) provides new binding sites for circulating free thyroid hormone [11, 20]. Although albumin binds thyroid hormone less avidly than TBG, it provides a much larger capacity for low-affinity binding that may contribute to lower free thyroid hormone levels. Plasmapheresis is a reasonably safe procedure, with a recommendation of grade IIc and category III in the latest American Society for Apheresis document [21]. The overall incidence of adverse effects of this technique, which are largely reversible, is approximately 5%. Notable side effects include transfusion reaction, hypotensive reactions, citrate-related nausea and vomiting, respiratory distress and tetany. Death is rare and usually due to an underlying disease [22].

Clinical features represent manifestations of organ decompensation, with fever seen almost universally. In our patient, the body temperature reached 40°C, and the surface cooling cannot effectively control body temperature. In order to avoid heating caused further damage, we used the vascular internal heat exchange technology.

Hypothermia treatment can help provide neuroprotection in cases of anoxic brain injury and global brain ischemia; hence it has been applied in cardiac arrest patients [8]. The treatment effectiveness in other neurological injuries such as stroke and traumatic brain injury is not established firmly [23-25]. Endovascular therapeutic hypothermia is feasible and avoids the problems encountered in previous studies that used surface cooling, notably poor maintenance of target temperature, uncontrolled rewarming, and overshoot to lower temperatures [26, 27]. In this case, using endovascular therapeutic hypothermia, we successfully controlled the patient's body temperature at 36°C, and stopped using endovascular cooling within 72 hours. The patient had no fever again.

This case illustrates a successful management using plasmapheresis to decrease circulating thyroid hormone levels and endovascular therapeutic hypothermia to control body temperature in a patient with thyroid crisis.

Disclosure of conflict of interest

None.

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