Case Report

A Case of Severe Dehydration with Marked Rhabdomyolysis

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A 23-year-old male was admitted to hospital with severe dehydration and hypokalemic myopathy due to secondary aldosteronism. On admission serum sodium and chloride were markedly elevated to 198 mEq/l and 169 mEq/l, respectively, and serum potassium was down to 2.3 mEq/l. Serum electrolytes were normalized by transfusion therapy, but subsequently rhabdomyolysis grew worse due to metabolic abnormalities such as dehydration, hypothermia, oppressive ischemia and metabolic acidosis, at the same time transient polyuria and the elevation of serum myoglobin and enzymes originating in muscle tissue were observed. Serum CPK went up to 26,532 IU/l on the sixth day and other enzymes reached a peak following CPK. Dexamethasone was administered when the increase in enzyme levels caused the patient to fall into a stupor. He rapidly regained consciousness from the 15th day after admission, and he was able to stand up on the 29th day. Serum enzymes originating in muscle tissue decreased gradually to the normal range by the 30th day and no renal failure occurred.

Key Words: Heat exhaustion, Hypernatremia, Hypokalemia, Secondary aldosteronism, Myoglobinuria, Myoglobinemia.

Dehydration occurs in various diseases. Heat syndrome which occurs when a person is subjected to high temperatures is often accompanied by dehydration. We treated a 23-year-old male, a member of the Ground Self Defense Force of Japan, who collapsed from heat exhaustion during field exercises in summer. Severe dehydration and hypokalemic myopathy by the secondary aldosteronism were observed on admission. Subsequently, rhabdomyolysis became worse due to metabolic abnormalities such as dehydration and hypothermia which occurred after hospitalization.

CASE PRESENTATION

Actual history: A 23-year-old male, a member of the Self Defense Force of Japan, was admitted to the First Department of Internal Medicine of

Ehime University Hospital on Aug. 1, 1983, with disturbance of consciousness and muscular weakness. He had a record of good health, and had been a member of the Ground Self Defense Force since the age of eighteen. Since the beginning of July in 1983, he had been participating in field exercises in extremely hot weather. Although he began to suffer from severe thirst, muscular weakness and fatigue from the middle of July, he continued to participate in field training and ignored his thirst. On July 30, 1983, he complained of a headache and others noticed an unnatural gait, slurred speech and irritability. The next day he developed a middle grade fever and was admitted to the hospital. He had lost 8 kg since the beginning of July.

Physical examination: On admission to the hospital, the patient appeared sluggish and drow-

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sy. His height was 160 cm, and he weighed 44 kg. His blood pressure was 110/76 mmHg, his pulse was 84/min and irregular. His axillary temperature was 37.2°C. Palpebral conjunctiva was hyperemic and bulbar conjunctiva was not icteric. The pupils were isocoric and reacted to direct and consensual light. The results of the ocular funduscopic examination were within normal limits. The tongue was reddened and markedly wrinkled. Lymph nodes, in general, and struma were not palpable. There were no abnormal findings in the chest or the abdomen. The extremities showed no edema and the skin was dry. Neurological examination

disclosed a drowsy patient with the lack of response of the patellar tendon reflex. He had muscle grasp pain and his muscle strength had decreased.

Laboratory findings: The results on admission are shown in Table 1. Erythrocyte sedimentation rate was slower than usual. In urinalysis, the specific gravity increased strikingly and proteinuria and positive occult blood reaction were found, but the urine sediment contained only a few erythrocytes and a few hyaline and granular casts. Stool examination was positive for the occult blood test. Hematological examination showed polycythemia resulting from hemoconcentration. Although the white blood cell count was within normal range, a shift to the left was observed. In blood chemistry, GOT and LDH were slightly above normal levels. Serum sodium and chloride were markedly elevated to 198 mEq/l and 169 mEq/l, respectively. Potassium was decreased to 2.3 mEq/l. BUN and serum creatinine were elevated too. The findings of sodium, chloride, BUN and creatinine indicated severe hypertonic dehydration. The blood sugar count showed hyperglycemia. The rises in the levels of CPK, which went up to 1,181 IU/l, aldolase, GOT and LDH indicated rhabdomyolysis. High levels of both renin and aldosterone indicated the presence of secondary aldosteronism. The level of myoglobin in the serum and urine was high. Hemostasis was within normal range, but blood gas analysis showed metabolic acidosis.

Although there were no abnormal findings in the CT scan of the head, an electroencephalogram (EEG) taken on the fifth day after admission revealed moderate disturbance of consciousness, such as severe low voltage, no change by opening and closing eyes, existence of driving to the photostimulation. The electrocardiogram showed supraventricular premature systoles.

Clinical course: The patient was diagnosed for heat exhaustion accompanied by hypertonic dehydration resulting from heavy perspiration and low liquid intake after participating in field exercises in extreme heat.

Muscle weakness and the increase of various

Table 1. Laboratory Findings

ESR	1 hr	1 mm	Blood chemistry	
	2 hr	2 mm	GOT	45 IU/l
Urine			GPT	47 IU/l
Specific gravity	1.047		LDH	208 IU/l
pH	6		Isozyme LDH ₁	11.2%
Protein	(2+)		LDH ₂	22.3%
Sugar	(-)		LDH ₃	18.3%
Occult blood	(2+)		LDH ₄	11.6%
Urobilinogen	(N)		LDH ₅	36.4%
Sediment RBC	1/HPF		AIP	84 IU/l
WBC	4/HPF		γGTP	6 IU/l
Hyaline & granular casts	4/HPF		LAP	61 IU/l
Bacteria rods	(3+)		Cholesterol	176 mg/dl
			P. lipid	211 mg/dl
			Triglyceride	159 mg/dl
			T. protein	6.6 g/dl
Stool			albumin	68.3%
Occult blood	(+)		α ₁ globulin	2.4%
Ova	(-)		α ₂ globulin	10.5%
Hemogram			β globulin	7.1%
RBC	543×10 ⁴ /μl		γ globulin	11.7%
Hb	17.6 g/dl		Na	198 mEq/l
Ht	45%		K	2.3 mEq/l
WBC	5900/μl		Cl	169 mEq/l
Meta	1.0%		BUN	71 mg/dl
Stab	24.5%		Uric acid	5.6 mg/dl
Seg	62.0%		Creatinine	2.7 mg/dl
Eo	0%		Creatine	1.7 mg/dl
Ba	0%		Ca	9.0 mg/dl
Mon	0%		T. bilirubin	1.1 mg/dl
Ly	12.5%		CPK	1188 IU/l
Platelet	13.3×10 ⁴ /μl		Isozyme MM	100%
Serological test			MB	0%
TPHA	(-)		BB	0%
VDRL	(-)		Aldolase	26.8 IU/l
HBsAg	(-)		Blood sugar	197 mg/dl
HBsAb	(-)		Amylase	152 Somo.U
CRP	(-)		Radioimmunoassay	
Blood gas analysis			Renin	4.892 ng/ml/hr
pH	7.264		Aldosterone	181.5 pg/ml
Pco ₂	41.2 mmHg		Serum	
Po ₂	99.0 mmHg		myoglobin	>500 ng/ml
HCO ₃	18.1 mEq/l		Urine	
BE	-8.5 mEq/l		myoglobin	189.6 ng/ml

enzymes originating in muscle tissue were considered to be symptoms of hypokalemic myopathy which resulted from an increase of aldosterone and a decrease of serum potassium. After admission, isotonic glucose and potassium were transfused immediately. Urine volume was 1,100 ml on the first day. Sodium and chloride were 188 mEq/l and 154 mEq/l, respectively, 12 hours after admission in spite of a transfusion of 4,500 ml of isotonic glucose solution and 110 mEq potassium. On the third day electrolytes in the serum had improved with therapy. Sodium, chloride and potassium levels improved to 160 mEq/l, 126 mEq/l and 3.5 mEq/l, respectively. Transfusion volume per day was reduced to 3,500 ml the following day. Despite a reduction of the transfusion amount, the urine volume reached 7,450 ml on the third day and its specific gravity was 1.009. Potassium in the urine was 246 mEq on that day. During this period, he remained drowsy and was unable to change his position himself because of muscle weakness. Although the hematocrit improved to 36% on the third day, it began to increase again with hypothermia. The color of the urine turned dark brown and the level of enzymes originating in muscle tissue such as CPK, GOT, GPT and LDH, began to rise and myoglobin in the serum and urine increased to over 500 ng/ml. Serum CPK reached 26,532 IU/l on the sixth day and then began to decrease. Other enzymes reached their peaks following CPK. They decreased gradually to the normal range by the 30th day and no renal failure occurred.

Eight to sixteen mg of dexamethasone per day were needed because he began to fall into a stupor with the rise of enzymes. Both the urine volume and the excretion of potassium were increased by dexamethasone. He began to regain consciousness rapidly from the 15th day with a decrease of enzymes originating in muscle tissue and was almost completely conscious on the 17th day (Figs. 1, 2).

Although the EEG results indicated a moderate disturbance of consciousness in spite of normalization of serum electrolytes on the fifth day, by the 37th day the EEG reading indicated that the patient was back to normal.

Fig. 1. After admission electrolytes in the serum improved with therapy. But on the third day a transient polyuria was observed accompanied by hypothermia and an increase of the hematocrit. From 6 to 8 days after hospitalization both the urine volume and the excretion of potassium were increased with the use of dexamethasone.

Renin and aldosterone returned to normal levels with the improvement of dehydration state (Fig. 3). However, while mental state and laboratory findings improved rather quickly, muscle strength returned slowly. He was finally able to stand up on the 29th day and walk after the 40th day.

A rectus femoris muscle biopsy was carried out

Fig. 2. On admission the rises in the levels of CPK, aldolase, GOT and LDH indicated rhabdomyolysis. On the third day the levels of enzymes originating in muscle tissue began to rise and myoglobin in the serum and urine increased to over 500 ng/ml. Serum CPK reached 26,532 IU/l on the sixth day and other enzymes reached their peaks following CPK.

Fig. 3. High levels of both renin and aldosterone indicated the presence of secondary aldosteronism. They returned to normal levels with the improvement of dehydration state.

on the 12th day, but no abnormal findings, such as degeneration, were noticed.

DISCUSSION

Heat syndrome is classified into heat cramp, heat exhaustion and heat stroke. Among them, heat exhaustion, which shows anomalous circu-

latory adaptation to high temperatures, is most common.

The present case was considered to have been heat exhaustion accompanied by severe hypertonic dehydration resulting from an excess of perspiring during field exercises in extreme heat for a month. Because of the dehydration of cerebral cells by hyperosmolarity the central nervous symptoms, such as impairment of consciousness, generally appear when the sodium concentration in the serum is more than 155 mEq/l^{1, 2)}. It was also reported that the sense of thirst was impaired in patients with hypernatremia³⁾. This case showed marked elevation of serum sodium concentration to 198 mEq/l. It appeared that the reason for this increase was not only due to dehydration but also to a decrease in liquid intake as result of thirst center impairment in the hypothalamus caused by hypernatremia and the impairment of consciousness. It was supposed moreover, that secondary aldosteronism, caused by dehydration, maintained serum sodium and induced hypokalemia. It is known that the sodium, potassium and chloride values in the cerebrospinal fluid (CSF) are elevated with an increase of the sodium concentration in the serum³⁾. It was also reported that a transient suppression of anti-diuretic hormone (ADH) secretion brought about water diuresis when the sodium concentration in CSF was lowered by the intraventricular infusion of isotonic solution without electrolytes⁴⁾. It was supposed accordingly that the decrease of the sodium concentration in CSF with a large transfusion of isotonic glucose caused polyuria on the third day. In addition, the occurrence of polyuria from 6 to 8 days after hospitalization is considered to be due to the diuretic action of dexamethasone because polyuria occurred with the use of the drug.

It is well known that rhabdomyolysis is caused by various conditions such as all kinds of muscle diseases, severe exercises^{5, 6)}, hypokalemia^{7, 8)}, various drugs⁹⁾, infections¹⁰⁾ and poisoning, and sometimes brings about acute renal failure. Subjective symptoms of muscle pain and weakness and abnormal findings of leakage of myoglobin and intracellular enzymes resulting from the

breakdown of muscle cell membrane are induced by rhabdomyolysis. In this case the existence of rhabdomyolysis was assumed because of the high values of CPK in the serum and myoglobinuria on admission. Rhabdomyolysis was considered to have resulted from severe exercise and hypokalemia. It is accepted that the increase in number and size of mitochondria of the skeletal muscle tissue by physical training protects the cell membrane through an increase of ATP production¹¹⁾. Moreover, it should be emphasized that rhabdomyolysis caused by exertion is mostly observed in amateurs and military recruits who have not undergone sufficient physical training^{5, 6)}. On the third day after admission, rhabdomyolysis became worse with marked myoglobinuria and an elevation of the serum enzymes originating in muscle tissue. This is considered to be caused by metabolic abnormalities such as oppressive ischemia caused by a decrease of body motion (bed rest), dehydration resulting from polyuria, hypothermia and metabolic acidosis¹²⁾.

The fact that no evidence of degeneration indicating rhabdomyolysis was found in this case does not necessarily negate the existence of rhabdomyolysis because muscle biopsies sometimes reveal normal histological figures after recovery from the disease^{13, 14)}. It may be possible that the reason the findings of the muscle biopsy in this case revealed no abnormalities was perhaps because it was performed during the convalescent stage or taken from an uninjured site.

Heat syndrome is often accompanied by dehydration and hypernatremia, which sometimes brings about fatal intracranial bleeding³⁾. It has also been reported that rhabdomyolysis induced fatal acute renal failure in some cases⁹⁾. The polyuria on the third day in this case possibly resulted from the rapid and large isotonic glucose transfusion, therefore serum sodium concentration should be corrected more slowly and care-

fully. It is important that heat syndrome and rhabdomyolysis, which are sometimes fatal, should be diagnosed at an early stage and should not be confused with mere fatigue, and care should be taken to insure complete recovery.

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