

# A case of severe recurrent hypoglycemia after traumatic brain injury

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#### Case

A 14-year-old male had a <u>severe traumatic</u> brain injury (TBI) after a motorcycle accident 11 months ago.

He underwent craniotomy and was admitted in ICU. Two months later, he was diagnosed as refractory epilepsy. Three episodes of seizure were related to <u>low blood sugar</u> (<u>blood sugar</u> <u>10-20 mg/dL</u>). He had inadequate intake due to fatigue and loss of appetite. Liquidized food via the enteral feeding route every 4 hours was used to prevent hypoglycemia. Levetiracetam (Keppra) 250 mg twice a day was used to treat seizures.

However, epilepsy was not controlled by medication. He was referred to a Pediatric Neurologist. Electroencephalography revealed focal epileptic disorder arising from the left frontopolar area.

**Laboratory** showed panhypothyroidism. (Table)

## Figures of MRI





MRI of the brain demonstrated gliotic and encephalomalacia changed at anterior left frontal lobe and left temporal lobe. Severe atrophic changed of the pituitary gland, without mass lesion.

## **Table of laboratory**

	Results	Reference range	Units
TSH	2.49	0.20 to 4.00	mIU/L
FT3	1.47	1.60 to 4.00	pg/mL
FT4	0.18	0.8 to 2.3	ng/dL
GH	<0.05	0–1	ng/mL
IGF1	26.8	187-510	ng/mL
ACTH	13.0	0-46	pg/mL
Cortisol	<1	4-20	ug%
FSH	0.2	1-8.4	IU/L
LH	<0.10	1–10.5	IU/L
Testosterone	<0.087	5.9-24.7	nmol/L
Na	135	135–145	mmol/L

#### Clinical course

At our hospital, the patient was being bedridden. His weight was 39.5 kg, his height was 156.8 cm. The laboratory test revealed panhypopituitarism; central hypothyroidism, GHD, central adrenal insufficiency, hypogonadotropic hypogonadism. (Table). He had no symptom of central DI.

**Prednisone and thyroxine** were started. The anti-epileptic drug was continued. Later, he had a normal appetite and his weight increased 5 kilograms in a month. He had no episode of hypoglycemia and seizure.

### Discussion

Signs and symptoms associated with hypopituitarism often mimic the sequelae of TBI. Therefore, hypopituitarism is likely to be underdiagnosed.

In our case, we considered recurrent severe hypoglycemia as the result of central adrenal insufficiency and GHD which are in counter-regulation mechanisms of hypoglycemia.

Moreover, central adrenal insufficiency and central hypothyroidism caused the consequences of appetite and body weight.

## Conclusion

We reported the case of panhypopituitarism following the severe TBI with an unusual presentation as hypoglycemia. In the case of severe TBI, the hormonal testing should be conducted, and then routine hormonal screening tests should be evaluated.









