

CR-GE-41**PANHYPOPITUITARISM FROM EMPTY SELLA SYNDROME ASSOCIATED WITH IDIOPATHIC PORTAL GASTROPATHY IN AN ADULT MALE**

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INTRODUCTION

Hormone deficiencies from hypopituitarism have been linked to liver dysfunction in the neonates up to adulthood with a spectrum ranging from cholestasis, elevated transaminases, NAFLD, hepatitis and cirrhosis. The exact mechanisms are still unclear but points to growth- and adrenocorticotrophic hormone deficiencies affecting liver metabolic pathways. This paper presents a rare case of panhypopituitarism associated with portal gastropathy.

CASE

A 19-year-old male presented with hematemesis from bleeding esophageal varices, requiring rubber band ligation. He has no prior or family history of hepatic and metabolic disorders. Diagnostics revealed elevated transaminases, recent normal bilirubins but high alkaline phosphatase, and bicytopenia consistent with hypersplenism. Hepatitis profile and iron overload markers were normal. Work-up for common causes of cirrhosis were negative and a diagnosis of idiopathic portal hypertension was made. He was referred to endocrinology due to eunuchoid body habitus, signs of hypogonadism and symptoms of hypothyroidism and hypocortisolism. Hormonal evaluation revealed anterior pituitary and target-organ hormone deficiencies. Delayed skeletal maturity was seen on skeletal survey. Cranial MRI revealed bilateral hippocampal atrophy with unremarkable sellar structures. Hormone replacement with steroids and Levothyroxine was initiated with improvement of symptoms. He plans to start testosterone therapy and is scheduled for surveillance endoscopies.

CONCLUSION

Case reports have suggested that early hormone replacement therapy in hypopituitarism may still reverse liver dysfunction such as cholestasis but if initiated later, the pathology will most likely be persistent. Though rare, in patients presenting with idiopathic liver dysfunction and signs of hormone deficiencies, a neuroendocrinologic cause such as hypopituitarism should be entertained and addressed.

KEY WORDS

panhypopituitarism, portal gastropathy, cirrhosis

CR-GE-42**HYPOGONADOTROPIC HYPOGONADISM ASSOCIATED WITH CENTRAL HYPOTHYROIDISM AND SECONDARY ADRENAL INSUFFICIENCY IN TWO FILIPINO MALE ADULTS**

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INTRODUCTION

Hypogonadotropic hypogonadism (HH) is a form of gonadal failure secondary to deficient gonadotropin secretion. HH that occurs in association with impaired secretion of other pituitary hormones result from defects in pituitary cell differentiation. We present two adult males with secondary hypogonadism, hypothyroidism and adrenal insufficiency.

CASE

A 32-year-old admitted for viral encephalitis was incidentally found to have absent secondary sex characteristics, micropallus and cryptorchidism. Diagnostics revealed low testosterone levels 0.025 nmol/L (NV=2.8–8), low DHEAS 3.9 ug/dL (NV=120-520) with inappropriately low LH 0.345 mIU/L (NV 1.7–8.6) and FSH 0.421 mIU/mL (NV=1.7–8.6). TSH was normal at 0.837 UIU/ml (NV=0.3–5) but with low FT4 6.96 pmol/L (NV 11–22) and FT3 0.805 pmol/L (NV 3.1–6.5). Morning serum cortisol was decreased at 74.26 nmol/L (NV=171-536). The second patient is a 50-year old male with eunuchoid habitus complaining of low energy. Laboratory results similarly showed normal TSH 3.7 UIU/mL with decreased values of the following: FT4 8.42 pmol/L, morning cortisol 59.94 nmol/L, testosterone 0.020 nmol/L, LH <0.100 mIU/L and FSH 0.276 mIU/mL. Both had unremarkable pituitary imaging and normal male karyotypes. Hormone replacement therapy was given.

CONCLUSION

Detection of low testosterone and gonadotropin levels confirms the diagnosis of male hypogonadotropic hypogonadism. Early recognition and diagnosis of HH including associated hormone deficiencies can prevent negative physical and psychological sequelae and restore fertility in affected patients, thereby improving quality of life.

KEY WORDS

hypogonadotropic, hypogonadism, hypothyroidism, adrenal, insufficiency, central