Paediatrics E-Poster

EP_P012

BASELINE ASSESSMENT OF SELF-CARE PRACTICES AND PSYCHOLOGICAL WELL BEING AMONG YOUNG ADULTS WITH TYPE 1 DIABETES IN A WARRIOR CAMP SETTING

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INTRODUCTION

Psychological well-being and effective self-care are critical in the management of young adults with Type 1 Diabetes (T1DM). The T1DM Warrior Camp conducted under the MEMS-CD1C initiative is a unique focused camp to empower young adults with T1DM on advocacy and peer leadership. This study aimed to evaluate baseline levels of depression, anxiety and stress in young adults attending a T1DM camp using the Depression Anxiety Stress Scales-21 (DASS-21) and the self-care practices using the Summary of Diabetes Self-Care Activities (SDSCA) questionnaire with a follow-up evaluation planned six months post intervention.

METHODOLOGY

Fourteen young adults with T1DM (aged 18–25) attended a structured 3-day Warrior Camp focused on diabetes education, lifestyle management and peer engagement. Baseline assessment was done using the DASS-21 and SDSCA questionnaire.

RESULT

Mean haemoglobin A1c and SDSCA score were 9.34 (± SD 2.43)%. Participants showed the highest adherence in blood glucose monitoring (5.93 ± 1.2), while foot care had the lowest adherence (3.1 ± 1.1). Dietary and exercise behaviours showed moderate adherence. The average stress score was 7.9 ± 1.2, corresponding to mild stress. Participants also reported moderate anxiety and mild depression, based on mean subscale scores.

CONCLUSION

This initial assessment highlights key areas of strength and opportunity in self-care practices among young adults with T1DM. Notably, blood glucose monitoring was a strong domain, whereas foot care requires greater emphasis. These young adults on assessment report mild to moderate symptoms of psychological distress, highlighting the importance of mental health support in this population. Camps may serve as a valuable setting for monitoring and addressing psychosocial needs in young adults with T1DM. The impact of the Warrior Camp intervention on these two critical areas will be reassessed after six months to evaluate long-term changes.

EP_P013

FAMILIAL MIDFACIAL HYPOPLASIA WITH CONGENITAL HYPOPITUITARISM – A CASE REPORT

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INTRODUCTION

Congenital hypopituitarism is defined as deficiency of one or more pituitary hormones due to abnormal pituitary gland development. Manifestations can be nonspecific such as poor weight gain, short stature, hypoglycemia or they may be associated with midline defects.

CASE

A 10-year-6-month-old female was diagnosed with congenital hypopituitarism at the age of 5 years whereby she presented with septicemic shock secondary to bronchopneumonia with hypernatremic dehydration. Detailed physical examinations showed that she was dysmorphic with short stature, poor muscle bulk, global developmental delay and features of midfacial hypoplasia such as right cleft lip/palate and septo-optic dysplasia. Investigations of the pituitary hormones revealed hypothyroidism, hypocortisolism, growth hormone deficiency and diabetes insipidus. Cranial MRI showed hypoplastic corpus callosum, absent septum pellucidum and thickened pituitary stalk with absence of bright spots of the posterior pituitary. She was started on pituitary hormone replacement including L-thyroxine, oral desmopressin, oral hydrocortisone and somatotropic injection. Clinical response to treatment was satisfactory in which she had gained 6 cm of height for the past year with normalized thyroid hormone and cortisol levels. Analyzing her family history, we noticed that her mother also had features of midfacial hypoplasia. Her elder sister is having a learning disability attending special school. This raised the possibility of genetic mutation in familial congenital

hypopituitarism such as PROP1 gene. Even with similar mutation, individuals can have different levels of hormone deficiencies and be affected differently.

CONCLUSION

Careful evaluation of a dysmorphic child with features of midfacial hypoplasia is crucial to avoid missing congenital hypopituitarism. Early identification with comprehensive hormonal work-up is important to initiate hormonal therapy.

EP_P014

IT IS NOT WHAT IT SEEMS

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INTRODUCTION

Elevated thyrotropin-releasing hormone (TRH) in response to very low thyroxine (T4) level can lead to pituitary gland hyperplasia. This condition can mimic a pituitary adenoma, making it radiographically difficult to differentiate from hyperplasia.

CASE

A 10-year-8-month-old male presented to medical attention due to poor growth and learning difficulties. He was noted to be small since he was 5 years old. There were no significant medical or dietary issues. He was not dysmorphic, but growth parameters corresponded to a 4-year old male. He was prepubertal and there was no goiter noted. Investigations initially showed a normal thyroid function, low IGF-1 and a bone age which corresponds to a 6-month-old. An MRI of the brain was arranged several months later which revealed a pituitary macroadenoma compressing / indenting the optic chiasm. A repeat pituitary panel showed elevated prolactin which did not change post PEG. There was also new evidence of primary hypothyroidism. As the biochemical results exclude a macroadenoma, it was postulated that high TRH as a response to a low FT4 leads to the stimulation of the pituitary thyrotroph and lactotroph cells resulting in pituitary gland enlargement. This is thought to be rare in children but documented to occur in those with severe primary hypothyroidism with a TSH >50 mIU/L. This could be misdiagnosed as a macro-adenoma especially when thyroid function test was not performed prior to an MRI of the pituitary gland.

CONCLUSION

This case illustrates the importance of differentiating a macroadenoma from pituitary hyperplasia. The treatment differs with invasive surgery for macroadenoma and thyroxine replacement in pituitary hyperplasia.

EP_P015

A CASE OF FAMILIAL GLUCOCORTICOID RESISTANCE SYNDROME PRESENTING WITH HYPOKALEMIC PARALYSIS AND HYPERTENSION

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INTRODUCTION

Familial glucocorticoid resistance syndrome (FGRS) is a rare condition leading to compensatory ACTH hypersecretion and excess adrenal steroid production. Patients often present with mineralocorticoid and androgen excess but without features of Cushing's syndrome.

CASE

A 17-year-old, male, Malay was referred for recurrent episodes of acute paralysis secondary to hypokalaemia and concomitant hypertension since the age of 7 years. Investigations showed persistent hypokalaemia with metabolic alkalosis. Clinically, he was not dysmorphic, taller for his age, increased skin pigmentation and was in pubertal with testicular volume of 6 ml and stretched penile length of 7 cm. Laboratory investigations showed a very marked increase in random serum cortisol of more than 2000 with elevated ACTH level. Luteinizing hormone-releasing hormone (LHRH) test confirmed a diagnosis of peripheral precocious puberty. Adrenal ultrasound did not show any suspicion of malignancy. He was started on oral dexamethasone and anti-hypertensive. He showed some improvement clinically and biochemically with no further history of paralysis and improvement in serum cortisol and potassium levels.

This patient presentation is consistent with FGRS where impaired cortisol signaling leads to compensatory increase in ACTH causing excess mineralocorticoid and sex hormones. Management focuses on reducing ACTH stimulation using high-dose dexamethasone and addressing complications such as hypertension and electrolyte imbalances.

CONCLUSION

Careful evaluation of a child presenting with unexplained hypertension, hypokalaemia and hyperpigmentation is