They are at a risk of developing recurrent UTI, VUR and hydronephrosis. Renal damage or failure can occur as a result of repeated infections with progressive renal scarring or as a result of obstruction caused by inability to empty the bladder. During follow up visits, each patient should have a physical examination with blood pressure measurement and growth monitoring, a urine analysis for proteinuria for early detection of deteriorating renal functions, a urine culture and ultrasound examination of the upper urinary tract. The best way to assess the bladder function is urodynamic studies which are not available in Sri Lanka.

Clean intermittent catheterization is the mainstay of therapy and it should be done 3-4 times per day along with antibiotic prophylaxis to prevent UTI [1]. The other treatment options include pharmacological and surgical interventions. Pharmacotherapy includes anticholinergics for dysynergistic bladder. In the surgical approach lower urinary tract is manipulated by diversion, augmentation, bladder neck surgery or using artificial sphincter [1]. Ureteral reimplantation can be performed in patients with recurrent symptomatic UTIs despite adequate bladder drainage and antibiotic prophylaxis or in patients with persistent high-grade reflux with demonstrated renal scarring.

It is important to counsel regarding future social and psychological consequences of neuropathic bladder. The paediatricians can act as advocates to ensure adequate and smooth transition to adult services later [2].

References

A patient with generalised warty lesions

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Case report
A 36 year-old male, presented with intermittent low-grade fever, night sweats, productive cough with haemoptysis and diarrhoea of two month duration. Additionally, he complained of anorexia and weight loss for the past six months. He also suffered from generalised warty skin lesions for the past five years and recurrent respiratory tract infections for the past two years. He has a past history of recurrent cellulites from the age of twenty.

On examination, he was febrile, emaciated and depressed. There were warts covering all parts of the body and oral mucosa which showed evidence of Candida infection. He did not have jaundice, lymphadenopathy, clubbing, peripheral stigmata of endocarditis or features suggestive of chronic liver disease. Abdominal examination revealed an enlarged liver (liver span of 16 cm) and spleen. In addition, he had coarse crepitations in apical region of the right lung.

After the initial clinical evaluation, the following possibilities were considered; disseminated tuberculosis or lymphoreticular malignancy in the setting of immune deficiency state.

His full blood count showed low white cells (<1000/mm³), anaemia (Hb 8.2 g/dL) and
thrombocytopenia (90×10⁹/L). ESR was 120 mm in 1" hour while blood picture showed normochromic normocytic red blood cells with marked leucopenia and low platelet count. Excessive rouleaux formation was noted. His chest X-ray showed right apical non-confluent shadow suggestive of consolidation. Examination of sputum for Acid Fast Bacilli was negative on three consecutive days and Mantoux test was also negative.

Ultrasound scan of the abdomen confirmed mildly enlarged spleen and there were multiple circumscribed hypoechogenic lesions within the spleen. Some lesions showed central hyperechogenicity. There was moderate hepatomegaly. No ascites or enlargement of para-aortic lymph nodes were seen.

His liver and renal functions were normal while liver biopsy showed normal liver tissue. Bone marrow examination was normal and bone marrow culture was negative for bacteria or Acid Fast Bacilli. His blood, urine and stool cultures were sterile. HIV screening also was negative.

Since the above investigations did not point to a definitive diagnosis and there were features of immunodeficiency the following immunological studies were done.

Serum immunoglobulin concentrations

<table>
<thead>
<tr>
<th>Immunoglobulin</th>
<th>Concentration</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG</td>
<td>190 mg/dL</td>
<td>(200 – 1300)</td>
</tr>
<tr>
<td>IgM</td>
<td>70 mg/dL</td>
<td>(60 – 280)</td>
</tr>
<tr>
<td>IgA</td>
<td>100 mg/dL</td>
<td>(90 – 450)</td>
</tr>
</tbody>
</table>

Lymphocyte subsets

<table>
<thead>
<tr>
<th>Subset</th>
<th>Count /L</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>CD4</td>
<td>95</td>
<td>(300 – 1400)</td>
</tr>
<tr>
<td>CD8</td>
<td>86</td>
<td>(200 – 900)</td>
</tr>
<tr>
<td>CD3</td>
<td>194</td>
<td>(700 – 2100)</td>
</tr>
<tr>
<td>CD20</td>
<td>2</td>
<td>(100 – 500)</td>
</tr>
</tbody>
</table>

The above findings were suggestive of common variable immunodeficiency and patient was treated with a combination of anti-tuberculosis therapy, broad-spectrum antibiotics and antifungal agents. While on treatment patient failed to show an improvement and subsequently died. Postmortem findings including histology confirmed disseminated Aspergillosis involving the liver, spleen, kidneys and lungs.

**Discussion**

Common variable immunodeficiency is a heterogeneous disorder characterised by the following [1, 2]:

- markedly reduced serum levels of IgG (<400 mg/dL) and variably low IgA or IgM.
- recurrent bacterial infections.
- impaired antibody response despite the presence of B cells.
- normal or near-normal T-cell immunity in 60% of patients [1-3].

We arrived at the diagnosis based on reduced IgG, IgA and IgM with the

(a) onset of immunodeficiency state after 2 years of age and
(b) exclusion of defined causes of hypogammaglobulinaemia.

Fulfilling the third criteria which include the “absent or poor response to vaccines” was not practical in this patient.

Treatment includes intravenous immunoglobulin for life and antibiotics for the possible infections. Interleukin-2 (IL-2), Tumour necrosis factor (TNF) inhibitor and B lymphocyte stimulator (BlyS) therapy are been used as experimental therapies [2, 4].

**References**


A 69 year-old diagnosed patient with hypertension and ischaemic heart disease was admitted to hospital with a four day history of dysurea, vomiting, reduced urine output and altered behaviour for one day.

He was well until one month prior to the hospital admission when he developed an upper respiratory tract infection which lasted for three days and on the fourth day of the illness he became confused together with an unsteady gait. He was admitted to a district general hospital where he was infused with intravenous fluids to which there was a dramatic response. He was also found to be anaemic and the blood picture was normochromic, normocytic but a cause for the anaemia was not established. He was transfused with a pint of blood and discharged.

He was well for three weeks until he was admitted to the Teaching Hospital, Karapitiya with symptoms suggestive of a urinary tract infection. On examination he was afebrile, confused, pale and hypotensive. Reduced body hair was also noted. There were no focal neurological signs.

On admission the following differential diagnoses were considered; urinary tract infection, electrolyte imbalance secondary to vomiting and chronic subdural haemorrhage.

Treatment was commenced empirically with intravenous Co-amoxyclav. Investigations revealed the following: UFR - normal, Urine culture - sterile, Na⁺ - 125 mmol/L, K⁺ - 6 mmol/L, RBS - 3.3 mmol/L, Hb - 11 g/dL and

Chest X-Ray - normal. Patient remained confused despite treatment. There was persistent hyperkalaemia with hyponatraemia and the blood pressure remained low which prompted us to investigate for a possible hypoadrenalism. He was started on intravenous dexamethasone and normal saline for which he showed a marked improvement. Subsequent investigations revealed a low ACTH concentration (< 10 pg/mL - mean 24 pg/mL) with a low 6.00 a.m. cortisol concentration (35.8 nmol/L - Normal range - 100 -600 nmol/L) confirming our tentative diagnosis of secondary adrenal insufficiency. CT scan of the brain revealed an intrasellar pituitary tumour. On further evaluation of the pituitary hormone profile a low concentration of LH (0.79 u/L - Normal range1-10 u/L) and a low normal concentration of GH (4.35 u/L - Normal range > 4u/L) was found. Rest of the anterior pituitary hormone concentrations remained within normal range. Since the tumour showed mass effect he was referred for neurosurgical management.

Discussion

Pituitary tumours are uncommon (1-2 /100,000 patients per year) [1]. They account for 10% - 15% of intracranial tumours and 75% of them secrete inappropriate levels of pituitary hormones [2]. Almost all the pituitary tumours are benign [1]. Typical presenting features include hormonal hypersecretion, visual field defects, headache, hypopituitarism, pituitary apoplexy, hydrocephalus, cranial nerve palsies and temporal lobe epilepsy.