

childhood obesity, in low- and middle-income countries, which are facing an epidemic of chronic non-communicable diseases in the near future.

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Pitfalls in Investigating for Diabetes Insipidus

Many pediatricians will remember a clinical case or scenario that highlights the potential pitfalls when investigating patients with suspected diabetes insipidus(1). Water deprivation tests, like tests of anterior pituitary function, can be misleading with false positives and false negatives. For example, patients who fail deprivation tests by developing a plasma osmolality greater than 300 mOsm/kg, may ultimately be found to have primary polydipsia. Conversely, patients who 'pass' a deprivation test one week can fail the test in the next week. In the case of patients with primary polydipsia, the raised plasma osmolality may reflect an impaired urinary concentrating ability in a child with polydipsia. The

fact that a child can 'fail' a test shortly after being found to have apparently normal posterior pituitary function may reflect a varying capacity of a diseased hypothalamo-pituitary axis to produce appropriate amounts of vasopressin.

Factors such as the pattern of fluid intake, baseline biochemistry, the response to desmopressin and a period of observation in hospital may all prove to be extremely useful parameters when assessing a child with polydipsia. Hence, there is much more to the assessment of these children than deprivation testing alone. In our center, we have 'hijacked' the language of our local genetics team and talk about some children 'growing' into a diagnosis. This makes the point that one test does not necessarily provide an answer and information may need to be gathered over a period of time before a diagnosis is reached.

Bajpai and colleagues highlight a number of important points in their article about the presentation of young people with idiopathic and organic cranial diabetes insipidus (CDI)(1). Perhaps the key lesson is the central role of intracranial imaging in the investigation of patients with CDI. The authors discuss the importance of considering repeat intracranial imaging in children with what might appear to be 'idiopathic' cranial DI on the first set of images. It is well known that children with CDI secondary to an underlying germinoma may have no evidence of the malignant process on MR scanning until months or even years later(2).

The more profound phenotype in those patients with CDI secondary to organic pathology will partly reflect the impact of the disease process on anterior pituitary function. Unfortunately, the two groups of children (idiopathic and organic) in Bajpai and colleagues study do not segregate neatly into distinct groups in terms of age or height. This reinforces the importance of ongoing vigilance as well as the need to re-image all patients with supposedly idiopathic DI at a later date. Although many familial cases of cranial DI do not result in a profound phenotype in early life, presentation in infancy with failure to thrive is well recognized(3). Children can, therefore, grow poorly in the absence of anterior pituitary disease. Furthermore, growth can be surprisingly well preserved in patients with central nervous system disease as part of the phenomenon of 'growth without growth hormone'(4).

Patients with 'idiopathic' CDI were also relatively short in Bajpai and colleagues study (-1.0 SDS) which could be interpreted as meaning that they may not have 'idiopathic' CDI after all. The proportion of patients with idiopathic disease in case series like this will tend to fall as our understanding of diseases and disease evolution evolves. Hence, some of the idiopathic groups in this study may in fact have underlying autoimmune destruction(5) or vascular impairment(6).

A pediatrician should remember that there are few rules or tests that have 100% sensitivity and specificity for diagnosing DI in a short child. Furthermore, what is simply 'idiopathic' CDI in one year may not prove to be idiopathic the next.

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