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ETIOLOGICAL YIELD AND TREATMENT PATTERNS IN PAEDIATRIC ARGININE VASOPRESSIN DEFICIENCY: A SINGLE-CENTRE COHORT STUDY

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INTRODUCTION
Arginine vasopressin deficiency (AVP-D), previously termed central diabetes insipidus, is a rare paediatric endocrine disorder that may present as an early manifestation of diverse hypothalamic–pituitary conditions. Identifying the underlying etiology is crucial for guiding management and surveillance; however, in some children, the cause remains unresolved, leading to primarily symptomatic treatment. In Malaysia, published literature on paediatric AVP-D has been limited to isolated case reports, with no cohort-level data available. This study aims to describe the clinical features, etiological spectrum, and treatment patterns of paediatric AVP-D in a single-centre Malaysian cohort, emphasizing diagnostic yield, unresolved causes, and the central role of neuroimaging.

METHODOLOGY
A retrospective review was conducted of children diagnosed with AVP-D. Data collected included age at symptom onset, age at presentation, clinical features, etiological classification, neuroimaging findings, comorbidities, treatment modalities, and follow-up outcomes. Descriptive statistics were used for analysis.

RESULTS
Twelve children were included, with a median age of 9.8 years; two-thirds were male. Children were referred at a median age of 3.4 years, with presentation ranging from the neonatal period to 10.4 years. Diagnostic delay was minimal overall, although 25% experienced delays exceeding 1 year. The water deprivation test was performed in 33.3% of patients. Magnetic resonance imaging (MRI) was completed in all children and served as the principal determinant of etiology. Structural abnormalities were identified in 50% of the cohort, including semilobar holoprosencephaly (33.3%), Arnold–Chiari I malformation (8.3%), and pituitary stalk interruption syndrome (8.3%). The posterior pituitary bright spot was absent in most patients, and 50% required repeat MRI to clarify evolving neurohypophyseal features. Tumor-related AVP-D accounted for 8.3%, while one-third had no definitive etiology despite imaging. Initial therapy included desmopressin (58.3%), hydrochlorothiazide (25%), and diluted sublingual desmopressin (8.3%), with most requiring dose escalation. Growth impairment occurred in 66.7% of patients, and delayed puberty in 16.7%.

CONCLUSION
MRI played a central role in defining etiology, with half of the cohort demonstrating congenital structural abnormalities. Persistent unresolved cases highlight the diagnostic challenges of AVP-D and underscore the importance of early MRI and longitudinal endocrine follow-up.