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ORIGINAL ARTICLE

Hydranencephaly complicated by central diabetes insipidus: report of two cases and systematic review of literature

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Abstract

Purpose Hydranencephaly is a congenital condition characterized by the complete or near-complete absence of the cerebral cortex and basal ganglia, while central diabetes insipidus (CDI) is a condition characterized by the inability to concentrate urine due to a deficiency in antidiuretic hormone (ADH). CDI is known to occur in midline congenital malformations such as holoprosencephaly and septo-optic dysplasia, but its association with hydranencephaly is less well-established.

Methods We reported two cases of hydranencephaly complicated by CDI. We also performed a systematic review of the SCOPUS and PubMed databases for case reports and case series of patients with hydranencephaly and CDI, and compiled data on the clinical features and treatment options.

Results Seven cases of hydranencephaly complicated by CDI were identified from the systematic review in addition to the two cases reported here, resulting in a total of nine cases. The patients' age ranged from 4 days to 4 years, and there was a female sex predilection (3.5:1). Patients most commonly presented with macrocephaly, developmental delay, and seizures, with dysmorphic features noted in 33%. In addition to CDI, other endocrinologic derangements included hypothyroidism (22%), hypocortisolemia (22%), and panhypopituitarism (22%). CDI was treated using sublingual or oral desmopressin while hypopituitarism was treated with the appropriate hormone replacement therapy. Insertion of a ventriculoperitoneal (VP) shunt was reported in 44% of cases.

Conclusion The case reports and systematic review suggest a previously unknown association between hydranencephaly and CDI. Clinicians managing cases of hydranencephaly are advised to have a high index of suspicion for CDI in patients presenting with the characteristic signs and symptoms.

Keywords Hydranencephaly · Central diabetes insipidus · Congenital malformations · Shunt surgery

Introduction

Hydranencephaly is a rare congenital central nervous system disorder occurring in one in 10,000 births and reported in 0.2% of infant autopsies [1, 2]. It is characterized by the complete or near-complete absence of the cerebral cortex and basal ganglia with preservation of the thalami and brainstem [3, 4]. This condition is thought to arise from the intra-uterine occlusion of the bilateral supraclinoid internal carotid arteries, leading to resorption of cerebral tissue typically supplied by the anterior circulation [3–6].

Diabetes insipidus is a condition characterized by the inability to concentrate urine secondary to either deficiency of antidiuretic hormone (ADH) in the central type, or resistance of the kidneys to ADH in the peripheral type [7, 8]. It is rare, occurring in 1 in 25,000 births, with central diabetes insipidus (CDI) accounting for greater than 90% of cases [7, 9]. Causes of CDI include mechanical obstruction or infiltration of the pituitary gland by tumors, hypoxic injury, trauma, infections, or medications [7, 9]. Congenital malformations such as holoprosencephaly and septo-optic dysplasia are also known to be associated with CDI, but an association between CDI and hydranencephaly is less well-established [7, 8, 10–12].

We report two cases of hydranencephaly complicated by central diabetes insipidus. We also performed a systematic

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review of the literature to identify cases of hydranencephaly with CDI and discuss management options and outcomes.

Case reports

Case 1

Case 1 is a 4-month old female, born full term to a 29-year old G3P2 (2002) mother via primary low segment cesarean section due to an antenatal diagnosis of fetal hydrocephalus. The mother had regular pre-natal medical care and took folate and iron supplementation, and had an unremarkable pregnancy. The patient's head circumference at birth was 38 cm ($z > 2$). She was also noted to have episodes of increased urine output.

On consult, the patient was awake, active, with downward primary gaze but no other focal neurologic deficits. The head circumference was 51 cm ($z > 2$). She was able to turn her head toward sounds but did not have a social smile. Cranial CT scan showed severe hydrocephalus and absent bilateral cerebral cortices, with intact falx, thalami, cerebelli, and brainstem, consistent with hydranencephaly (Fig. 1). On admission, a diagnosis of central diabetes insipidus was made on the basis of increased urine output (7.9 mL/kg/h), elevated serum sodium (162 mmol/L), high serum osmolarity (335 meqs/L), low urine osmolarity (160 meqs/L), and low urine specific gravity (1.004). She was thus treated with desmopressin 50 μg/tab ½ tab once daily and fluid replacement. Hormonal work-up also revealed secondary hypothyroidism for which the patient was started on levothyroxine 25 μg/tab once daily.

The patient subsequently underwent VP shunt insertion. She was discharged well and maintained on desmopressin and levothyroxine. On follow-up at 11 months of age, the patient was seen awake, with fair activity and spastic bilateral lower extremities. Her head circumference was 48.5 cm ($z > 2$), decreased compared to the preoperative value but still above normal for age. She had no visual threat but had an intact auditopalpebral reflex.

Case 2

Case 2 is a 5-month old female, born pre-term to a 19-year old primigravid via low segment cesarean section performed due to an antenatal diagnosis of fetal hydrocephalus and onset of preterm labor. The mother had a history of cigarette smoking and alcoholic beverage intake during the first two trimesters of pregnancy. The head circumference at birth was 33 cm ($z < 0$). The patient was admitted at the neonatal ICU for a month where she was managed as a case of prematurity and neonatal pneumonia. At 2 months of age, the parents noted gradually progressive increase in head circumference, prompting consult.

On consult, the patient was awake, with good activity and no focal neurologic deficits. She had no social smile and had poor head control. Her head circumference was 45 cm ($z > 2$). She was also noted to have constriction bands on the second and third digits of the left hand and congenital amputation of the first and second digits of the right foot. Cranial CT scan showed severe hydrocephalus and absence of bilateral frontotemporoparietal lobes and thalami with intact brainstem, cerebellum, and part of the occipital lobes, consistent with hydranencephaly (Fig. 2).

On admission, the patient's serum sodium was 155 mmol/L, urine specific gravity was 1.006, and urine output was 0.2 mL/kg/h. She underwent an unremarkable VP shunt insertion the following day. However, on the first postoperative day, serum sodium increased to 172 mmol/L, urine specific gravity decreased to 1.003, while the urine output

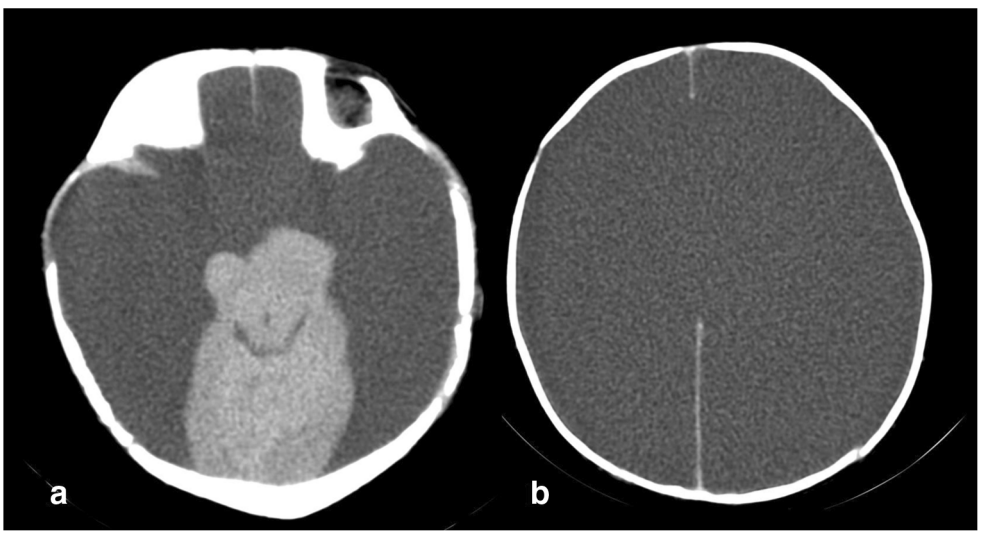

Fig. 1 **a** Axial cut of the plain cranial CT scan shows absence of bilateral frontotemporoparietal lobes with intact thalami and brainstem. **b** A superior axial cut shows absence of bilateral cerebral cortices with intact falx cerebri, consistent with hydranencephaly

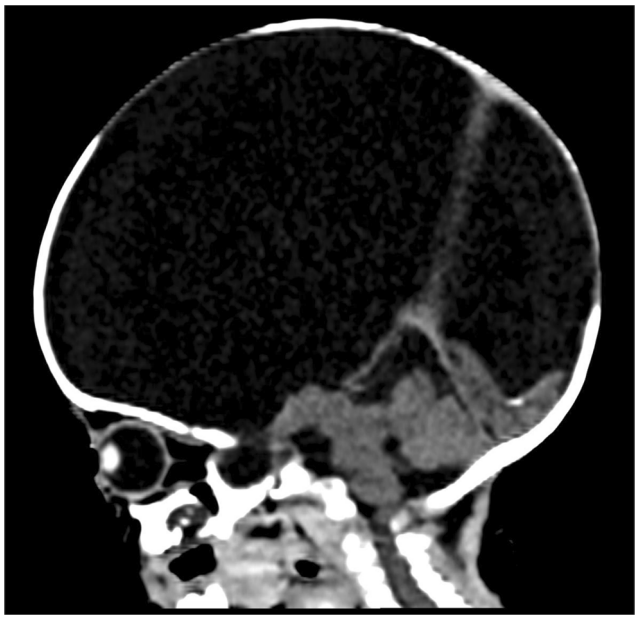

Fig. 2 Mid-sagittal cut of the plain cranial CT scan shows absence of the frontotemporoparietal lobes with intact brainstem, cerebellum, and inferior portion of the occipital lobes

increased to 4.3 mL/kg/h. The patient was diagnosed to have diabetes insipidus due to polyuria supported by the characteristic laboratory findings. She was started on oral desmopressin 50 μg/tab ½ tab once daily, resulting in normalization of the urine specific gravity, serum sodium, and urine output after several days.

The patient's clinical course was complicated by nosocomial pneumonia, sepsis, and fungal UTI. After 6 weeks of antibiotic treatment, the patient was discharged, improved, and maintained on oral desmopressin. On follow-up at 10 months of age, the patient was seen awake with fair activity, with an intact auditopalpebral reflex and no visual threat. The anterior fontanelle was flat and head circumference was 46 cm ($z > 2$).

Literature search strategy

A systematic search of the PubMed and SCOPUS databases was performed using the keywords "hydranencephaly" and "diabetes insipidus." The reference lists of the assessed articles were also searched for relevant studies.

All English- and Spanish-language case reports and case series on hydranencephaly patients with concomitant central diabetes insipidus were collected and analyzed. Articles were excluded if the diagnosis of hydranencephaly was not confirmed radiographically or if there was no breakdown of cases to allow analysis of individual data. Data collected included patient demographics, clinical features, imaging results, medical and surgical management employed, and outcomes.

A total of 41 records were identified through a database search. Of these, 28 articles were excluded after screening the titles and abstracts for relevance to the study. The full text of 13 articles were assessed; of which, 7 articles were excluded for the reasons stated above. Six studies were included in the qualitative analysis (Fig. 3).

Results

In addition to the two cases we are reporting, seven cases of hydranencephaly complicated by central diabetes insipidus were identified from the systematic review, resulting in a total of nine cases (Table 1). The patients' age ranged from 4 days to 4 years at the time of the report, with a female sex predilection (3.5:1). The patients most commonly presented with macrocephaly, developmental delay, and seizures. Dysmorphic features were seen in 33%. In addition to diabetes insipidus, other endocrinologic derangements included hypothyroidism (22%), hypocortisolemia (22%), and panhypopituitarism (22%). Diabetes insipidus was treated using sublingual or oral desmopressin, while panhypopituitarism was treated with the appropriate hormone replacement therapy. Insertion of a VP shunt was reported in 44% of cases. The longest survivor in the series was a 4-year and 10-month old patient who died due to respiratory infection.

Discussion

The findings in our case reports and systematic review suggest a previously unknown association between hydranencephaly and central diabetes insipidus.

The diagnosis of CDI was suggested in our patients due to the presence of characteristic signs and symptoms and confirmed by laboratory investigations. The signs and symptoms vary with age and may often be difficult to discern because they are relatively non-specific [17, 18]. Neonates often present with hypernatremic dehydration due to their limited access to free water [18]. The diagnosis is considered in the setting of significant weight loss, hypernatremia, and increased urinary output as evidenced by excessively wet diapers [17]. Infants, on the other hand, may present with vigorous suck, recurrent fever, irritability, failure to thrive, constipation, and increased urination. The diagnostic criteria for CDI includes the following: elevated plasma osmolality (> 300 mOsm/kg), low urine osmolality (< 300 mOsm/kg), urine/plasma osmolality ratio < 1, and polyuria (urine output > 4–5 mL/kg/h for 2 h in children, and > 6 mL/kg/h in neonates) [18].

Hydranencephaly is thought to arise from intrauterine insults affecting the bilateral supraclinoid internal carotid arteries (ICA), resulting in resorption of brain tissue that is

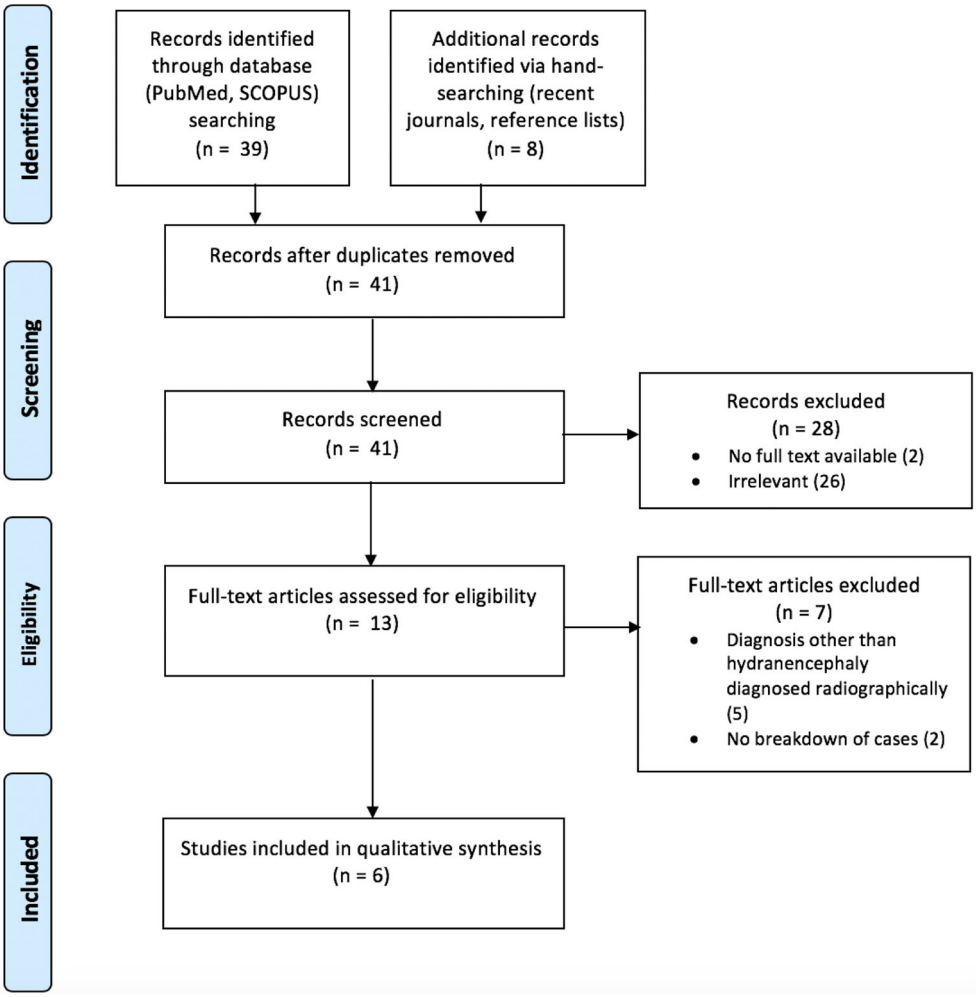

Fig. 3 PRISMA flow diagram of the systematic review

normally supplied by the anterior circulation [3–6]. This theory is supported by animal models of intrauterine bilateral ICA occlusion leading to abnormalities resembling hydranencephaly, and the findings of bilateral supraclinoid ICA hypoplasia in vascular imaging done for hydranencephaly patients [4–6]. Branches of the ICA form the portal capillary system that supplies the pituitary gland [19]; thus, it stands to reason that the same embryonic events leading to in utero bilateral ICA damage can also cause the aberrant development of the pituitary gland due to vascular insults. This in turn may result in central diabetes insipidus and varying degrees of hypopituitarism, with the latter present in six of the nine patients (67%) in this review series.

An alternative theory for the pathogenesis of hydranencephaly suggests that encephaloclastic processes occurring in utero may lead to resorption of normal brain tissue. In this regard, the disease can be considered a form of porencephaly arising from various pathologies, including toxic effects of drugs, diffuse hypoxic ischemic damage, and infection [3, 5]. The infectious agents implicated in the development of hydranencephaly include cytomegalovirus (CMV), *Toxoplasma gondii*, herpes simplex virus, Epstein-Barr virus, adenovirus, parvovirus, and respiratory syncytial virus [2, 3, 5]. In the same manner, central diabetes insipidus has been known to occur as a result of injury to the pituitary gland from various mechanisms, including mechanical destruction by trauma, severe central nervous system infection causing inflammation, and trauma or hypoxic injury causing disruption of the blood supply [7]. In multiple case reports, CDI occurred as a sequela of congenital toxoplasmosis infection, enteroviral meningoencephalitis, herpes simplex infection, *Streptococcus* B meningitis, cryptococcal meningitis, intracranial tuberculomas, and intracranial abscesses [17, 20–26]. In the series by Kasim et al., the prevalence of intracranial abnormalities was significantly higher in the series of patients with CDI than in the general population [8]. This includes a higher incidence of meningitis (13%), traumatic brain injury (9%), and isolated ventricular hemorrhage (9%) [8]. The destructive

Table 1 Cases of concomitant hydranencephaly and diabetes insipidus reported in the literature, including the current case reports

Author, Year	Age/ sex	Clinical features	Imaging	Medical/surgical management	Outcome
Poe 1989 [13]	6 W/F	Irritability Delayed motor development Macrocephaly DI	MRI: fluid-filled supratentorial space with persistence of the falx and relative sparing of posterior fossa structures	NR	NR
Segawa 2007 [3]	3/F	Dysmorphism (cleft lip and palate, flat nasal bridge, lack of nasal bone and septum, hypotelorism, microphthalmus) Seizure Disorder of body temperature regulation DI and hypothyroidism	CT: cerebral hemispheres nearly completely replaced by CSF; intact diencephalon; intact tentorium; and partially intact falx cerebri	NR	Died at 4 Y due to respiratory infection
Negi 2010 [14]	4/M	Macrocephaly Dysmorphism (vertical elongation of head, flat nasal root, micropenis, cryptorchidism) Recurrent infection and dehydration DI and panhypopituitarism	MRI: fluid-filled supratentorial space; thin layer of cerebral hemisphere beneath skull; intact falx in the occipital area	VPS insertion at 7 m of age; Gastrostomy and tracheostomy at 2 Y of age	Died at 4 Y and 10 mos. due to infection
Kelly et al. 2011 [5]	NB/F	Seizures DI	MRI: absence of cerebrum, anterior midline cyst, hypoplastic cerebellum; absent falx	NR	Died at 4 days
Chinsky 2012 [1]	2/F	Seizures Macrocephaly Multiple skin hemangiomas Dysmorphism (hyperteloric upslanting palpebral fissures, right hand transverse crease, and bilateral 5th finger clinodactyly) DI and panhypopituitarism (diagnosed post-VPS)	MRI: absence of cerebral hemispheres but preservation of cerebellar and midbrain tissues. Heterogeneous T2 hyperintense signal suggests proteinaceous CSF	VPS insertion at 8 W of age; Hormone replacement therapy (medications and doses not reported)	Alive at 2 Y (on home hospice care)
Marin 2018 [15]	9 m/M	Developmental delay DI and hypocortisolemia	MRI: CSF-filled supratentorial space; intact parieto-occipital lobe and brainstem; possible hypoplastic pituitary	Sublingual desmopressin (12 μg BID)	Alive at 9 m
Marin 2018 [15]	3 m/F	DI and hypocortisolemia	MRI: CSF-filled supratentorial space; intact temporal region; possible hypoplastic pituitary	Sublingual and oral desmopressin (12 μg BID)	Alive at 3 m
Omar 2019 [16]	4 m/F	Macrocephaly Developmental delay DI and hypothyroidism	CT: absence of bilateral cerebral cortices with intact falx, thalami, cerebelli, and brainstem	VPS insertion at 4 m; oral desmopressin (25 μg OD), levothyroxine (25 μg OD)	Alive at 11 m
Omar 2019 [16]	5 m/F	Macrocephaly Developmental delay DI	CT: absence of bilateral frontotemporoparietal lobes; intact brainstem, cerebellum, and occipital lobes	VPS insertion at 5 m; oral desmopressin (25 μg OD)	Alive at 10 m

NR not reported, *W* weeks, *m* months, *Y* years *DI* diabetes insipidus, *CT* computed tomography, *MRI* magnetic resonance imaging

mechanisms leading to the development of hydranencephaly may have also resulted in damage to the magnocellular neurons in the neurohypophysis, causing CDI.

Central diabetes insipidus has been known to occur in other congenital malformations, particularly in congenital midline defects arising from aberrant segmentation and cleavage [12]. In Wang's series of 35 patients with CDI, congenital malformations were seen in 20%. The most common associated congenital anomalies were callosal agenesis, midline facial defects, microcephaly, and holoprosencephaly [12]. In a larger series of 67 patients, Santiprabhob et al. noted a concomitant central nervous system malformation in 29.8%, which included septo-optic dysplasia, holoprosencephaly, schizencephaly, porencephaly, and nasoethmoidal meningocele. The series also included eight patients with hydranencephaly [11]. However, there was no breakdown of the individual cases in their series and was thus excluded from the current analysis. Kasim et al. also noted three cases of CDI occurring with hydranencephaly, but there was no breakdown of cases [8].

Central diabetes insipidus occurring as a presumed complication of hydrocephalus has also been sparsely reported in the literature. Borenstein-Levin reported two cases of CDI occurring in preterm-infants with posthemorrhagic hydrocephalus that improved after resolution of the hydrocephalus [18]. In one of these cases, insertion of a VP shunt led to resolution of the CDI [18]. Dribin and McAdams also reported a case of congenital hydrocephalus from aqueductal stenosis presenting with CDI, whose symptoms improved with endoscopic third ventriculostomy and bilateral choroid plexus cauterization [27]. Menzies et al. postulated that the hydrocephalus may cause distension of the third ventricle and compression of the neuropophysis or the paraventricular nuclei of the thalamus, resulting in CDI [28]. In our two cases, the insertion of a VP shunt did not improve the patients' CDI.

Krebs et al. noted that the presence of CDI worsened the prognosis of the initial pathology if there was a concurrent congenital malformation, as the consequent hyperosmolality may induce permanent neurologic sequelae [17]. However, for hydranencephaly, in which survivors are expected to have severe neurocognitive defects, the prognostic significance of concomitant CDI remains unknown. Quek et al. note that an intact hypothalamic-pituitary-adrenal axis is critical to the prolonged survival of these patients [2]. However, the lack of formal survivorship data for this rare condition limits prognostication [29].

The possible association between hydranencephaly and central diabetes insipidus should prompt clinicians managing patients diagnosed with hydranencephaly to have a high index of suspicion for CDI and hypopituitarism. While the prognosis for cognitive improvement remains poor for all patients with hydranencephaly, prompt recognition and management of medical comorbidities should intuitively improve survival. This possible association should also be among the points to be included in the counseling of families and caregivers on the prognosis of this congenital condition.

Conclusion

The two cases we described, as well as a systematic review of the reported literature, suggest a previously unknown association between hydranencephaly and central diabetes insipidus. Clinicians managing cases of hydranencephaly are advised to have a high index of suspicion for central diabetes insipidus in patients presenting with the characteristic signs and symptoms.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Chinsky JM (2012) Hydranencephaly: transillumination may not illuminate diagnosis. Neoreviews 13:e233–e240. https://doi.org/10.1542/neo.13-4-e233
2. Quek Y-W, Su P-H, Tsao T-F, Chen JY, Ng YY, Hu JM, Chen SJ (2008) Hydranencephaly associated with interruption of bilateral internal carotid arteries. Pediatr Neonatol 49:43–47. https://doi.org/10.1016/S1875-9572(08)60011-X
3. Segawa Y, Itokazu N, Hirose A, Nakagawa S, Takashima S (2007) A case of partial 14q- with facial features of holoprosencephaly and hydranencephaly. Pediatr Neurol 37:51–54. https://doi.org/10.1016/j.pediatrneurol.2007.02.010
4. Dublin AB, French BN (1980) Diagnostic image evaluation of hydranencephaly and pictorially similar entities, with emphasis on computed tomography. Radiology 137:81–91. https://doi.org/10.1148/radiology.137.1.7422865
5. Kelly TG, Sharif UM, Southern JF, Gururajan K, Segall HD (2011) An unusual case of hydranencephaly presenting with an anterior midline cyst, a posterior calcified mass, cerebellar hypoplasia and occlusion of the posterior cerebral arteries. Pediatr Radiol 41:274–277. https://doi.org/10.1007/s00247-010-1894-1
6. Cecchetto G, Milanese L, Giordano R, Viero A, Suma V, Manara R (2013) Looking at the missing brain: hydranencephaly case series and literature review. Pediatr Neurol 48:152–158. https://doi.org/10.1016/j.pediatrneurol.2012.10.009
7. Dabrowski E, Kadakia R, Zimmerman D (2016) Diabetes insipidus in infants and children. Best Pract Res Clin Endocrinol Metab 30:317–328. https://doi.org/10.1016/j.beem.2016.02.006
8. Kasim N, Bagga B, Diaz-Thomas A (2018) Intracranial pathologies associated with central diabetes insipidus in infants. J Pediatr Endocrinol Metab 31:951–958. https://doi.org/10.1515/jpem-2017-0300

9. Di Iorgi N, Napoli F, Allegri AEM et al (2012) Diabetes insipidus: diagnosis and management. Horm Res Paediatr 77:69–84. https://doi.org/10.1159/000336333
10. Pavone P, Praticò AD, Vitaliti G, Ruggieri M, Rizzo R, Parano E, Pavone L, Pero G, Falsaperla R (2014) Hydranencephaly: cerebral spinal fluid instead of cerebral mantles. Ital J Pediatr 40:79. https://doi.org/10.1186/s13052-014-0079-1
11. Santiprabhob J, Likitmaskul S, Boonyasiri A, Boonsathorn S, Buddawong T (2005) Etiologies of central diabetes insipidus in Thai children. J Pediatr Endocrinol Metab 18:653–661
12. Wang LC, Cohen ME, Duffner PK (1994) Etiologies of central diabetes insipidus in children. Pediatr Neurol 11:273–277. https://doi.org/10.1016/0887-8994(94)90001-9
13. Poe LB, Coleman L (1989) MR of hydranencephaly. Am J Neuroradiol 10:S61
14. Negi M, Kobayashi D, Kumagai J, Kuroiwa T, Shiraishi J, Takemura T, Irahara K, Hayashi M, Eishi Y (2010) An autopsy case of congenital hydrocephalus and severe thinning of the cerebral cortex in a 4-year-old boy. Neuropathology 30(5):559–563. https://doi.org/10.1111/j.1440-1789.2010.01138.x
15. Marin G, Baspineiro B, Vilca I (2018) Treatment with sublingual desmopressin in two infants with hydranencephaly and central diabetes insipidus. Arch Argent Pediatr 116:e93–e97. https://doi.org/10.5546/aap.2018.e93
16. Omar AT, Khu KO (2019) Hydranencephaly complicated by central diabetes insipidus: report of two cases and systematic review of literature. Childs Nerv Syst (in review)
17. Krebs VLJ, Damiani D, Albuquerque Diniz EM et al (1998) Central diabetes insipidus as a complication of neonatal pathology: report of three cases. Pediatr Int 40:146–149. https://doi.org/10.1111/j.1442-200X.1998.tb01900.x
18. Borenstein-Levin L, Koren I, Kugelman A, Bader D, Toropine A, Riskin A (2014) Post-hemorrhagic hydrocephalus and diabetes insipidus in preterm infants. J Pediatr Endocrinol Metab 27(11-12):1261–1263. https://doi.org/10.1515/jpem-2014-0098
19. Antunes JL, Muraszko K (1990) The vascular supply of the hypothalamus-pituitary axis. In: Neuroendocrinological aspects of neurosurgery. Springer Vienna, Vienna, pp 42–47
20. Karadag A, Erdeve O, Atasay B, Arsan S, Deda G, Ince E, Ocal G, Berberoglu M (2006) Isolated central diabetes insipidus in a newborn with congenital toxoplasmosis. J Pediatr Endocrinol Metab 19(2):173–175. https://doi.org/10.1515/JPEM.2006.19.2.173
21. Jones G, Muriello M, Patel A, Logan L (2015) Enteroviral meningoencephalitis complicated by central diabetes insipidus in a neonate: a case report and review of the literature. J Pediatric Infect Dis Soc 4:155–158. https://doi.org/10.1093/jpids/pit055
22. Torres MDAM, Kazee MDAM, Simon MDH et al (2000) Central diabetes insipidus due to herpes simplex in a patient immunosuppressed by Cushing's syndrome. Endocr Pract 6:26–28. https://doi.org/10.4158/EP.6.1.26
23. Ribeiro S, Domingues V, Faria RM, Mendonca T (2013) Invasive pneumococcal disease complicated by cerebral vasculitis, transient diabetes insipidus and spondylodiscitis. Case Reports 2013: bcr2013010336–bcr2013010336. https://doi.org/10.1136/bcr-2013-010336
24. Juffermans NP, Verbon A, Van der Poll T (2002) Diabetes insipidus as a complication of cryptococcal meningitis in an HIV-infected patient. Scand J Infect Dis 34:397–398
25. Chellen S, Whittaker E, Eisenhut M, Grandjean L (2018) Cerebral tuberculomas in a 6-year-old girl causing central diabetes insipidus. BMJ Case Rep 2018:bcr-2018-226590. https://doi.org/10.1136/bcr-2018-226590
26. Malhotra A, Bajpai A, Hunt RW (2010) Central diabetes insipidus in association with neonatal brain abscess. J Pediatr Endocrinol Metab 23(3):235–236. https://doi.org/10.1515/JPEM.2010.23.3.235
27. Dribin T, McAdams RM (2014) A neonate with gastroschisis and hydrocephalus complicated by central diabetes insipidus. J Pediatr Surg Case Rep 2:366–368. https://doi.org/10.1016/j.epsc.2014.07.011
28. Menzies DG, Shaw JF, Kean DM, Campbell IW (1988) Cranial diabetes insipidus secondary to arrested hydrocephalus. Postgrad Med J 64:300–302. https://doi.org/10.1136/pgmj.64.750.300
29. Merker B (2008) Life expectancy in hydranencephaly. Clin Neurol Neurosurg 110:213–214. https://doi.org/10.1016/j.clineuro.2007.11.011