Case report

Late magnetic resonance imaging findings in trauma-induced central diabetes insipidus: Case report and review of literature✩✩✩

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Abstract

We presented the late magnetic resonance imaging characteristics in a 47-year-old male who diagnosed with a permanent trauma-induced diabetes insipidus. The patient developed polyuria following a deceleration injury which has been diagnosed as central diabetes insipidus based on the water deprivation test. Computed tomography or magnetic resonance evaluation of the pituitary gland is usually normal in such cases. Therefore, negative imaging studies do not exclude the diagnosis. However, MRI is more sensitive and can depict subtle injuries of the hypothalamus-pituitary axis in acute and late phases. The late MR imaging findings are not well established. To the best of our knowledge, this will be the first report to describe the late MR imaging features in a permanent case of trauma-induced diabetes insipidus.

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Introduction

Trauma-induced central diabetes insipidus is a complication associated with traumatic brain injury (TBI) [1]. Central diabetes insipidus may pass unnoticed in patients presented with severe TBI [2]. Central diabetes insipidus is a clinical diagnosis that present with polyuria and hypernatremia [2,3]. Water deprivation test is helpful to distinguish diabetes insipidus (DI) from other causes of polyuria and even differentiate central from peripheral DI types [4]. In central DI, the patients will have reduced urinary osmolality < 300 mOsm/kg which increases > 50% after exogenous 1-deamino-8-D-arginine vasopressin (DDAVP) administration [5]. Majority of cases do not require imaging for diagnosis of DI [3]. Hence, normal imaging scans e.g. computed tomography (CT) or magnetic resonance imaging (MRI) do not exclude this possibility. However, MRI provides high resolution imaging of the pituitary gland and might be helpful in the acute phase and demonstrate acute injury of the hypothalamic-pituitary axis [1,2,6].

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Fig. 1 – Trauma-induced central diabetes insipidus in a 47-year-old male. Sagittal T1 pre (A), and post IV contrast administration (B) MR images demonstrate loss of PPBS and posterior translation of the pituitary infundibulum (arrows). (C) High-resolution sagittal T1 non-contrast enhanced MPRAGE image shows focal kinking and partial transection of the pituitary stalk (arrow). (D) Coronal T2 weighted image at the level of hypothalamus shows parenchymal volume loss of the hypothalamus (arrowheads), mild dilatation of the third ventricle with mild global cerebral atrophy.

Case presentation

A 47-year-old Caucasian gentleman presented for evaluation of long history of hyponatremia. The patient had a remote history of motocross accident (deceleration injury), 8-years before time of presentation, with multiple compression fractures of the vertebrae. After the accident, the patient noticed that he had to go to the bathroom every hour and drink 8-10 bottles of water about 12 ounces daily. He also mentioned that he had to use the bathroom at least 5 times during the night. He complained of fatigue, tiredness, and mild erectile dysfunction that he has noticed two years earlier. Patient stated that he normally drinks 6-7 beers/night. Physical exam was normal. Differential diagnosis at this time included diabetes insipidus with dilutional hyponatremia owing to liberal intake of water and hypotonic fluid. Other possibility included cerebral salt wasting syndrome in which patients have increased urine osmolality > 100 mOsm/kg, increased urine sodium concentration > 40 mmol/L and decreased extracellular fluid volume (hypovolemia) [10,11]. Other less likely possibilities included central hypothyroidism or secondary adrenal insufficiency. Evaluation of pituitary function with measurement of cortisol level, testosterone, free thyroxin level and thyroid stimulating hormones was done which turned back as normal. The water deprivation test was ordered and revealed low urine osmolality which significantly increased from 147 to 515 following DDAVP injection, post water deprivation for 17 hours. These findings were consistent with central diabetes insipidus. The patient underwent MRI brain with pituitary protocol for evaluation of structural abnormality of the hypothalamic-pituitary axis. MRI showed loss of the posterior pituitary bright spot (PPBS) and posterior displacement of the pituitary infundibulum with partial transection of its fibers (Fig. 1). No evidence of hemorrhagic foci identified. The patient had prescribed DDAVP 0.1mg bid with significant improvement in polyuria. The patient was encouraged for liberal salt intake and beverages with electrolytes and to limit beer to 3/day maximum.
Discussion

We presented the long-term imaging findings in a permanent trauma-induced central diabetes insipidus. Diabetes insipidus following traumatic brain injury is the most commonly encountered immediate complication with salt and water disturbances [1]. The vast majority of cases are transient and reversible but can be permanent in rare occasions [1]. DI is an uncommon with an estimate of 1/25000 [12,13]. It is characterized by antidiuretic hormone (ADH) deficiency with clinical finding of polyuria (> 30 ml/Kg/day) and polydipsia [13]. Due to increase loss of water in urine with retention of electrolytes such as sodium, this lead into dilute urine (< 250 mmol/L), dehydration and hyponatremia [13]. However some patients with polydipsia and huge intake of hypotonic fluids can manifest with hyponatremia as in our case.

Traumatic brain injury is a leading cause of mortality and morbidity in young adults in the developed world with 10 million hospitalization each year [14]. According to the center for disease control, 2.8 million TBI-related emergency department visits, hospitalization and deaths were reported in the united states in 2013 [15]. The estimated number of TBI-related deaths is over 50000 annually [14]. The incidence rate increases significantly with adolescents, young adults, and military service members (approximately 1,763.6 per 100,000) [9]. Pituitary dysfunction is one of the common immediate and late complication of traumatic brain injury with variable prevalence (5%-70%) among studies [9]. This large variation might be attributed to patient selection, different mechanisms of injury, various time-points for evaluation, and different study designs [9]. Trauma-induced pituitary dysfunction is usually associated with moderate to severe TBI. Hence, patients who present with intracranial hemorrhage such as intraparenchymal hemorrhage or subarachnoid hemorrhage and seizure are vulnerable to develop post-traumatic pituitary dysfunction and central diabetes insipidus [16,17]. However trauma-induced pituitary dysfunction has been developed in a substantial number of patients with mild TBI. Close monitoring for symptoms of hypopituitarism and hormone testing should be performed in patients with TBI [16].

Though majority of cases are transient and reversible, incidence rate of permanent pituitary dysfunction is higher than previously reported [3,18]. Trauma-induced central diabetes insipidus occurs due to direct injury of the hypothalamus and posterior pituitary gland. Post-traumatic edema of the hypothalamus and posterior gland results in transient cases that resolves after subsidence of the swelling [1]. The permanent cases typically develop due to direct injury to the hypothalamic nuclei i.e. paraventricular and supra-optic nuclei, pituitary infundibulum and neurohypophysis [19]. Associated intracranial hypertension can contribute to the injury of pituitary axis through occlusion of the long hypophyseal vessels crossing the diaphragma sellae. Short portal veins arise below the diaphragma sellae, supplying the medial and anterior adenohypophysis are typically spared giving chance for long term recovery [3]. In severe trauma cases, transection of the pituitary infundibulum could occur and result in disruption of hypothalamic outflow into the posterior pituitary gland [3].

Trauma-induced central diabetes insipidus manifests with polyuria and loss of large amount of dilute urine with increased plasma osmolality. Partial or subclinical form of diabetes insipidus has mild symptoms and is usually overlooked by severity of neurological and cognitive comorbidities [20]. Diagnostic workup includes thorough evaluation of serum electrolytes, serum ADH, urinary and plasma osmolality, and 24 h urine collection [12]. Water deprivation test is helpful to diagnose diabetes insipidus and distinguish between central and nephrogenic subtypes [4]. The classic findings in central DI include urine osmolality < 300 mOsm/kg which increases > 50% after exogenous DDAVP administration [5]. As the patient presented with hyponatremia, the possibility of cerebral wasting syndrome, hypothyroidism and adrenal insufficiency should be excluded. In contrast to DI, cerebral wasting syndrome is characterized by high urine osmolality (> 100 mOsm/kg) and sodium concentration (> 40 mmol/L) with hypovolemia [10,11]. Normal levels of cortisol level, free thyroxin level and thyroid stimulating potentially exclude the possibility of anterior pituitary dysfunction with hypothyroidism or adrenal insufficiency. The complete details on clinical diagnostic workup to differentiate these conditions is beyond the scope of this report. Our patient also presented with dilutional hyponatremia due to consumption of large amount of hypotonic fluid.

CT is the initial imaging modality to evaluate for TBI. However, MRI is more sensitive and could depict subtle injury of the hypothalamic-pituitary axis. The imaging features in the immediate post-traumatic stage may be normal. The manifestation of traumatic brain injury as intracranial hemorrhage and skull fractures could be present [1,13]. The pituitary gland characteristically has normal T1 hyperintense focus of the neurohypophysis, also known as posterior pituitary bright spot (PPBS) in 60%-90% of individuals [13,21]. Many hypotheses are postulated about the reason of T1 hyperintensity of neurohypophysis which may be attributed to the presence of high percentage of neurophysins (ADH carrier protein), phospholipid vesicles, or granules containing oxytocin and ADH [13,21]. Injury of the neurohypophysis is the hallmark of acute and early development of trauma-induced central diabetes insipidus (CDI) [6]. The key imaging finding is loss of normal T1 hyperintensity of neurohypophysis, posterior pituitary bright spot (PPBS) [6-8,13,21]. Other imaging features include focal hemorrhage or infarction within the hypothalamus or pituitary gland, thickening and focal hematoma of the pituitary infundibulum [1,22]. Acute transection of the pituitary infundibulum has also been described however it leads into delayed onset CDI [6,7,13,22]. Ectopic and displacement of bright spot or neurohypophysis secondary to stalk transection and retraction could be observed [1,13,23]. The imaging findings in late stages are not well established. To the best of our knowledge this is the first report of long-term imaging criteria in permanent trauma-induced central diabetes insipidus. The acute findings of hemorrhage and edema of the hypothalamus and pituitary infundibulum typically subside with restoration of normal function of the pituitary axis in transient cases. In contrast, the permanent cases could manifest radiologically with parenchymal volume loss of hypothalamus and pituitary gland, attenuated or loss of neurohypophysis bright spot, and pituitary infundibulum changes (Fig. 1).
These changes include posterior translation of the pituitary infundibulum with partial or complete transection of its fibers and atrophy as observed in our case.

Since majority of the cases are transient, administration of single dose of DDAVP parenterally is the treatment of choice [1,2,16]. Careful observation and monitoring of the patient symptoms is recommended as DDAVP should be administered if polyuria recurred [2,20]. In contrast, patients with permanent pituitary dysfunction and central diabetes insipidus require long-life DDAVP replacement [1,2,16]. The recommended dose is 0.2 mg once at night or twice daily [2]. The expected complication of treatment is the development of hyponatraemia which should be monitored carefully [13,16].

Conclusion

Late MR imaging findings in clinically suspected trauma-induced central diabetes insipidus could include loss of posterior pituitary bright spot (PPBS), parenchymal volume loss of the hypothalamus, partial or complete transection of the pituitary infundibulum with subsequent ectopic neurohypophysis.

Ethics approval and consent to participate

Not applicable.

Data sharing

Data used in this study are not shared publicly.

Patient consent

Patient consent was waived by the IRB for the study design (case report).

REFERENCES

[1] Capatina C, Paluzzi A, Mitchell R, Karavitaki N. Diabetes insipidus after traumatic brain injury. J Clin Med. 2015;4(7):1448–62. doi:10.3390/jcm4071448.
[2] Tudor RM, Christopher, Thompson J. Posterior pituitary dysfunction following traumatic brain injury: review. Pituitary 2019;22:296–304. doi:10.1007/s11202-018-0917-z.
[3] Scranton R, Baskin D. Impaired pituitary axes following traumatic brain injury. J Clin Med 2015;4(7):1463–79. doi:10.3390/jcm4071463.
[4] Gubbi S, Hannah-Shmouni F, Koch CA, Verbalis JG. Diagnostic testing for diabetes insipidus. MDtext.com, Inc; 2019. Feingold KR, Anawalt B, Boyce A, al, Ed. Published online https://www.ncbi.nlm.nih.gov/books/NBK537591/.
[5] Christ-Crain M. At the cutting edge diabetes insipidus: new concepts for diagnosis; 2020. Published online. doi:10.1159/000505548.
[6] Shin JH, Lee HK, Choi CG, et al. MR imaging of central diabetes insipidus: A pictorial essay. Vol 2; 2001. Available at: www.amc.seoul.kr. Accessed February 4, 2021.
[7] Ma L, Gao Y, Cai Y, Li T, Liang Y. MR evaluation of the brain in central diabetes insipidus. Chin Med J (Eng) 1996;109(9):724–9.
[8] Tien R, Kucharczyk J, Kucharczyk W. MR imaging of the brain in patients with diabetes insipidus. AJNR 12533-542 1991;12:533–42.
[9] Silva PPB, Bhattachar J, Herman SD, et al. Predictors of hypothopituitarism in patients with traumatic brain injury. J Neurotrauma 2015;32(22):1789–95. doi:10.1089/neu.2015.3998.
[10] Kiyama H, Tolunay O, Training AN, et al. Inapropriate antidiuretic hormone secretion and cerebral salt-wasting syndromes in neuroendocrine patients. Front Neurosci 2019. Available at: www.frontiersin.org 2019;13:1170. doi:10.3389/fnins.2019.01170.
[11] Yee AH, Burns JD, Wijdicks FM Cerebral salt wasting: Pathophysiology, diagnosis, and treatment. Neurosurg Clin NA. 21:339–352. doi:10.1016/j.nec.2009.10.011.
[12] Di Iorgi N, Napoli F, Allegri AF, et al. Diabetes insipidus - diagnosis and management. Horm Res Paediatr 2012 Published online. doi:10.1159/000336333.
[13] Adams NC, Farrell TP, O’Shea A, et al. Neuroimaging of central diabetes insipidus-when, how and findings. Neuroradiol 2018;60:995–1012. doi:10.1007/s00234-018-2072-7.
[14] Sulhan S, Iyon KA, Shapiro LA, Huang JH, Author C. Neuroinflammation and blood-brain barrier disruption following traumatic brain injury: Pathophysiology and potential therapeutic targets HHS public access. J Neurosurg 2020;98(1):19–28. doi:10.1016/j.jnrs.2020.04.011.
[15] Taylor CA, Bell JM, Breiding MJ, Xu L. Traumatic brain injury related emergency department visits, hospitalizations, and deaths — United States, 2007 and 2013. MMWR Surveill Summ 2017;66(SS-9):1–16. doi:10.15585/mmwr.ss6609a1.
[16] Gilis-Januszewska A, Kluczynski L, Hubalewska-Dydejczyk A. Traumatic brain injuries induced pituitary dysfunction: A call for algorithms. Endocr Connect 2020;9(S):R112–23. doi:10.1530/EC-20-0117.
[17] Gray S, Bilski T, Diedonne B, Saeed S Hypopituitarism after traumatic brain injury: Published online 2019. doi:10.7759/cureus.4163.
[18] Chou Y-C, Wang T-Y, Yang P-Y, Meng N-H, Chou L-W. Brain Injury Permanent central diabetes insipidus after mild traumatic brain injury) Permanent central diabetes insipidus after mild traumatic brain injury. Brain Inj 2009;23:1095–8. doi:10.3109/02699050903379396.
[19] Agha A, Thornton E, O’Kelly P, Tormey W, Phillips J, Thompson CJ. Posterior pituitary dysfunction after traumatic brain injury. J Clin Endocrinol Metab 2004;89(12):5987–92. doi:10.1210/jc.2004-1058.
[20] Bellastella A, Bizzarro A, Coletta C, Bellastella G, Sinisi AA, De Bellis A. Subclinical diabetes insipidus. Best Pract Res Clin Endocrinol Metab 2012,26(4):471–83. doi:10.1016/j.beem.2011.11.008.
[21] Elster AD. Modern imaging of the pituitary. Radiology 1993;187(1):1–14. doi:10.1148/radiology.187.1.8451394.
[22] Roguski M, Morel B, Sweeney M, et al. Magnetic resonance imaging as an alternative to computed tomography in select patients with traumatic brain injury: A retrospective comparison. J Neurosurg Pediatr 2015;15(5):529–34. doi:10.3171/2014.10.PEDS14128.
[23] Su D-H, Chang Y-C, Chang C-C. Post-traumatic anterior and posterior pituitary dysfunction. J Formos Med Assoc 2005;104(7):463–467 Available at: http://europemc.org/abstract/MED/16091821.