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Review Article

Blake's pouch cyst

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Abstract

Background: In 1900, Joseph Blake described a transient posterior evagination of the tela choroidea of the fourth ventricle in the normal 130-day old human embryo. He was the first to recognize and fully elucidate on the real nature of the foramen of Magendie as an aperture, which develops within a saccular expansion of the embryonic fourth ventricular cavity. The persistence of this temporary fourth ventricular outpouching into the postnatal period and its significance either as separate entity or as an entity within the Dandy–Walker continuum has over the years been one of the most controversial topics in both neurosurgical and neuroradiological literature.

Methods: A search of the medical literature was conducted for publications addressing the historical, embryological, and neuororadiological features as well as the clinical presentation and management of persistent Blake's pouch.

Results: The literature on the various features of Blake's pouch cyst has limited areas of consensus between various authors.

Conclusion: Blake's pouch cyst is a rare entity that is thought to belong to the Dandy–Walker continuum. It has a variable clinical presentation and when symptomatic can be treated with an endoscopic third ventriculostomy or shunting.

Key Words: Blake's pouch cyst, Dandy–Walker continuum, endoscopic, ventriculostomy

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HISTORICAL BACKGROUND

In 1900, Joseph Blake [Figure 1] described a transient posterior evagination of the tela choroidea of the fourth ventricle in the normal 130-day old human embryo [Figure 2a].[7] He was the first to recognize and fully elucidate on the real nature of the foramen of Magendie as an aperture that develops within a saccular expansion of the embryonic fourth ventricular cavity.[7,35] In 1906, Wilson supported Blake's observations,[33,34] and later in 1937, added evidence of photographs [Figure 2b-d] of the condition in the human fetus, which constituted a convincing demonstration of the validity of the Blake's observations.[35] The persistence of the temporary fourth ventricular outpouching into the postnatal period and its significance either as separate entity or as an entity within the Dandy–Walker continuum has over the years been one of the most controversial topics in both neurosurgical and neuroradiological literature.[2,5,10,18,21,22,25,31]

In the original description of the Dandy–Walker malformation by Dandy and Blackfan in 1914, a huge cystic dilatation of the fourth ventricle with anterior displacement of the cerebellar vermis was described and attributed to primary atresia of the foramina of the fourth ventricle.[11] Over the years, many cases were successively reported expanding the limits of the malformation to include findings of one particular case or another and creating a great deal of confusion to the definition and pathoanatomical features of the syndrome.[21] Taggart and Walker in 1942 further defined the condition,[30] and Benda in 1954 introduced the name "Dandy–Walker syndrome" as well as the currently held opinion that atresia of the fourth ventricular exit foramina is not an essential feature of the malformation.[6]

In contrast, Harwood-Nash and Fitz introduced the term "Dandy–Walker variant" to describe conditions with posterior evagination of the anterior membranous area (AMA), partial vermian agenesis and a normal-sized posterior fossa,[20] while Raybaud used the same term to describe a malformation with variable degrees of agenesis of the vermis and expanded fourth ventricle communicating with the perimedullary subarachnoid space; he reserved the term "Dandy–Walker malformation" only to cases in which no communication between the dilated fourth ventricle and the subarachnoid space could be demonstrated.[24] Kollias *et al.* suggested using the term "vermian-cerebellar hypoplasia" to describe the group of congenital malformations characterized by normal-sized posterior fossa, varying degrees of vermian and cerebellar hypoplasia, and a prominent retrocerebellar space communicating freely with a normal or minimally dilated fourth ventricle through a prominent vallecula.[21]

Imprecise anatomical descriptions of mega cisterna magna and Blake's pouch cyst can be traced in the literature to parallel the overlapping and sometimes misleading terminology of the entities within the Dandy–Walker complex. In 1949, Robertson described three cases with very large cisterna magnae demonstrated by pneumoencephalograms and autopsy findings in one of them. It was speculated that the cyst had an ependymal origin based on its microscopic features, position within the posterior fossa, absence of arachnoidal abnormalities, and the relationship of the choroid plexus to the cyst wall; he nevertheless gave no name to the malformations.[26] In 1962, Gonsette and colleagues, coined the term "mega cisterna magna" to describe a series of adult patients with grossly enlarged cisterns, which they thought to be caused by cerebellar atrophy,[19] and the term was thereafter loosely applied to any large retrocerebellar cerebrospinal fluid (CSF) space with a normal vermis and cerebellar hemispheres.[22] In 1971, Gilles and Rockett demonstrated the clinical, ventriculographic, and pathologic findings associated with retrocerebellar cysts and suggested the origin in two of them to be a persistent Blake's pouch based on the presence of cuboidal epithelium with astroglia in the cyst walls; they termed the entity retrocerebellar "arachnoidal" cyst.[18] Raybaud in 1982 used the term "retrocerebellar arachnoid pouch" to describe evagination of the fourth ventricular tela choroidea either closed or open, with or without hydrocephalus, and with or without tentorial defect,[24] then later in 2010 described his term as both appropriate (being a pouch more than a cyst) and not appropriate as it is likely choroidal (Blake's pouch remnant rather than arachnoid).[25] The entity was, however, termed "Blake's pouch" by Harwood-Nash and Fitz.[20] Persistent Blake's cyst was also considered

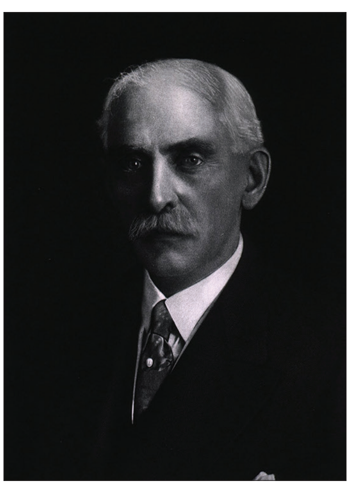

Figure 1: Joseph A. Blake (1864-1937). Images from the History of Medicine (IHM), National Library of Medicine, History of Medicine Division, Bethesda, Maryland, USA

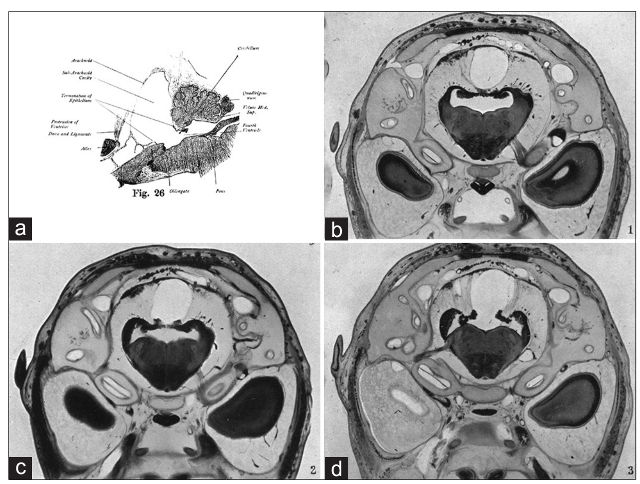

Figure 2: (a) Image No. 26 from Blake's original work[7] in 1900 demonstrating a sagittal section near the midline in 130-day human embryo. Note the posterior outpouching of the fourth ventricle. (b-d) Serial sections of the hindbrain of the human embryo at age of 5 months (129 mm crown rump length appearing in Wilson's paper of 1937[35] in support of Blake's observations. The sections are just in front of (b), at the rostral lip (c), and through the anterior part (d) of the foramen of Magendie. Reproduced with permission of John Wiley and Sons, Inc

synonymous with retrocerebellar arachnoid cyst[29] or mega cisterna magna[1] by some authors.

Barkovich *et al.* in 1989 pointed out that a clear separation of the Dandy–Walker malformation, Dandy–Walker variant, and mega cisterna magna into classical categories was not possible because of the new information obtained from magnetc resonance (MR) images. They considered these anomalies to represent a continuum of developmental anomalies of the posterior fossa and introduced the term "Dandy–Walker complex". They classified the cystic malforamations of the posterior fossa into two basic categories; Dandy–Walker complex and arachnoid cysts.[3] Strand *et al.* also held a similar opinion of unifying the nomenclature into Dandy–Walker complex.[29]

It was only in 1996 when Tortori-Donati *et al.* added persistent Blake's pouch cyst as an independent entity within the Dandy–Walker complex and proposed a classification based on embryopathogenesis. They held the opinion that anomalies of the AMA would result in either a Dandy–Walker malformation or a Dandy–Walker variant, while anomalies of the posterior membranous area (PMA) would result in a mega cisterna magna or persisting Blake's pouch. These authors stressed that a CSF collection in the posterior fossa should be termed mega cisterna magna only when there is neither hydrocephalus nor signs or symptoms secondary to compression of the nervous and ventricular structures of the posterior fossa.[31]

Blake's pouch cyst is currently considered one of the anomalies within the spectrum of Dandy–Walker complex by many authors.[8-10,32] Robinson and Goldstein suggested that the cisterna magna septa seen by ultrasonography represent the walls of Blake's pouch and supported the opinion that a persistent Blake's pouch and mega cisterna magna represent less severe abnormalities within the Dandy–Walker continuum.[27] Others, however, still hold the opinion that a Blake's pouch cyst does not qualify into the Dandy–Walker spectrum as the fourth ventricle does not communicate with the subarachnoid spaces in the midline,[28] while some consider it of no clinical significance unless causing mass effect due to hydrocephalus or associated cerebral or cerebellar dysgenesis.[5]

EMBRYOLOGY [Figure 3]

During embryogenesis, the plica choroidea divides the roof of the fourth ventricle into an AMA and a PMA. Both AMA and PMA are essentially the definitive tela choroidea that forms the roof of the posterior portion of the fourth ventricle. The embryonic roof plate, which is the primordium of the tela choroidea (or AMA and PMA), is invaginated by developing vascular structures to form the choroid plexus. The choroid plexus is not attached directly to the vermis at anytime during embryogenesis. Differentiation of the meninx primitiva around the neural tube results in the formation of the subarachnoid space of the cisterna magna.[17,21,31]

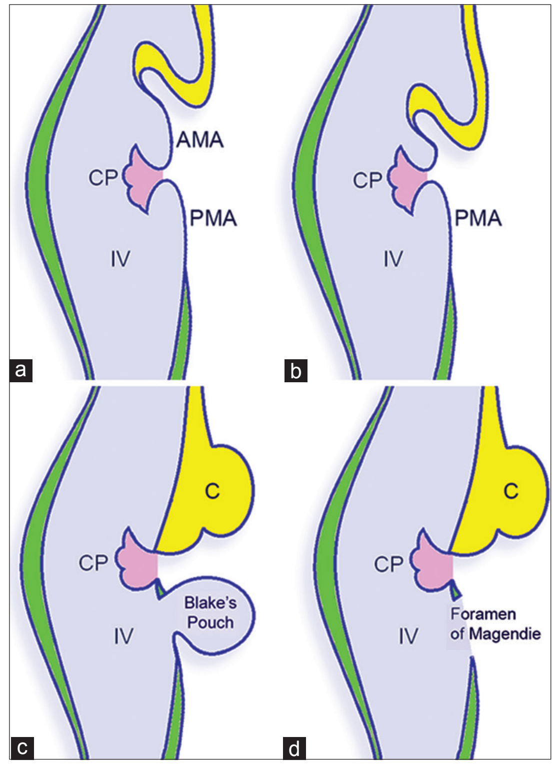

Figure 3: Embryonic sequence of events in the development of the roof of the fourth ventricle. The plica choroidea (choroid plexus) divides the roof of the fourth ventricle into an anterior membranous area and a posterior membranous area (a). The cerebellar vermis originates from the anterior membranous area (b), which eventually disappears. Blake's pouch appears as a protrusion of the posterior membranous area of the fourth ventricular roof (c), which later communicates with the subarachnoid space forming the foramen of Magendie (d). AMA: Anterior membranous area, C: Cerebellum, CP: Choroid plexus, IV: Fourth ventricle, PMA: Posterior membranous area. (Illustration by Waleed Azab, MD)

Blake's pouch is a transient finger-like protrusion of the PMA of the fourth ventricular roof, which extends posteriorly into the meninx primitiva caudal to the cerebellum. It is initially an ependymal-lined closed cavity, which later communicates with the subarachnoid space at a point from the 7th or 8th week up to the 4th month of gestation to form the foramen of Magendie. Persistence of the pouch with variable separation from the fourth ventricle and lack of communication with the subarachnoid space results in enlargement of the pouch to form the Blake's pouch cyst.[7,28,29]

In the absence of anomalies of the AMA – that is, when the vermis, cerebellar hemispheres, and fourth ventricle are normal – a defect of the PMA may produce two distinct malformations, the mega cistern magna and persisting Blake's pouch.[31] Failure of regression of

Blake's pouch (the rudimentary fourth ventricular tela choroidea) takes place secondary to nonperforation of the foramen of Magendie,[31] with a consequent enlargement of the fourth ventricle and the supratentorial ventricular system until the foramina of Luschka open and establish equilibrium of CSF outflow from the ventricles into the cisterns,[8] Notably, opening of the foramina of Luschka takes place late in the 4th month of gestation, that is, after the foramen of Magendie has been formed.[1,22,24,29,31] Notwithstanding, as the larger foramen of Magendie is permanently nonexistent, the ventricles will stay enlarged[10] with a compression rather than underdevelopment of the cerebellar hemispheres and vermis.[4]

It is important to note that the cerebellum, including the vermis, develops from the rhombic lip and the alar plate of the metencephalon. The rhombic lip also is the histogenic origin of the purkinje cells, external granular cell layer that becomes definitively the internal granular cell layer, the golgi cells, stellate cells, and basket cells as well as Bergmann and Fananas glial radial cells. This is germane to Dandy–Walker spectrum, because the vermis is hypoplastic or absent in that condition. This would indicate a migratory and/or proliferation arrest that would lead to genetical, epigenetical or disruption to growth factors and morphogens as etiological factors in the Dandy–Walker spectrum. The tela choroidea (or membranous area) rostrally attaches to the floccular peduncle of the flocculo-nodular lobe and does not give rise to the cerebellar lobes.

Genetic, growth, and morphogenic factors could be causative factors. The fenestration of the tela choroidea in the formation of the foramen of Luschka and foramen of Magendie is commonly accepted. Developmental apoptotic factors may be at play in such fenestrations, which take place at defined anatomical locations. Variations in the location of these foramina are rare or nonexistent. Therefore, if there is failure to obliterate a specific region of a membrane, then molecular factors may be downregulated or mutated. Alternatively, a failure of the choroid plexus to normally develop along with fenestration delays of the tela choroidea could also be taking place secondary to deviation of expression of growth factors or their receptors. Examples of such factors would include Vascular endothelial growth factor and its receptor (VEGF/VEGF-R), platelet derived growth factor and its receptor (PDGF/PDGF-R), transforming growth factor (TGF) isoforms, bone morphogenic protein, aquaporin/carbonic anhydrase (both noted in the choroids plexus) angiopoietin and its tyrosine kinase with immunoglobulin-like and EGF-like domains (TIE) receptors. Type II lissencephaly and Walker–Warburg syndrome/Fukuyumu muscle–eye–brain syndrome are associated with Dandy–Walker cyst in 50% of the cases. There is a 9q31-33 or 17 chromosomal defect in this complex. Even if there are no studies to demonstrate specifics, it is important to think in the direction of molecular etiological factors and not just be limited to evaginating mechanical origins of Blake's pouch cyst. Including the spectrum of molecular and genetical factors will be critical in future assessments as to a Blake's pouch cyst being a Dandy–Walker variant and/or continuum. The answer to these questions will come in the future not from a mechanical explanation, but a complex dysgenesis secondary to genomic/molecular errors with and without secondary mechanical consequences. The aqueductal stenosis, corpus callosal agenesis, cerebellar vermian hypoplasia, and encephaloceles of Dandy–Walker syndrome go beyond any mechanical origin to a singular effect of a delay in fenestration of the tela choroidea foraminal openings and is rather a more global central nervous system (CNS) developmental error.

RADIOLOGICAL FINDINGS [Figures 4 and 5]

The differential diagnosis of Blake's pouch cyst includes all other posterior fossa cysts in the Dandy–Walker complex [Figure 4], posterior fossa arachnoid cysts, and cyst-like malformations.[10] The Dandy–Walker malformation and related disorders have a variable combination of features that generated considerable confusion and controversy.[2,5,8,36] Moreover, conventional imaging modalities are insensitive in detection of

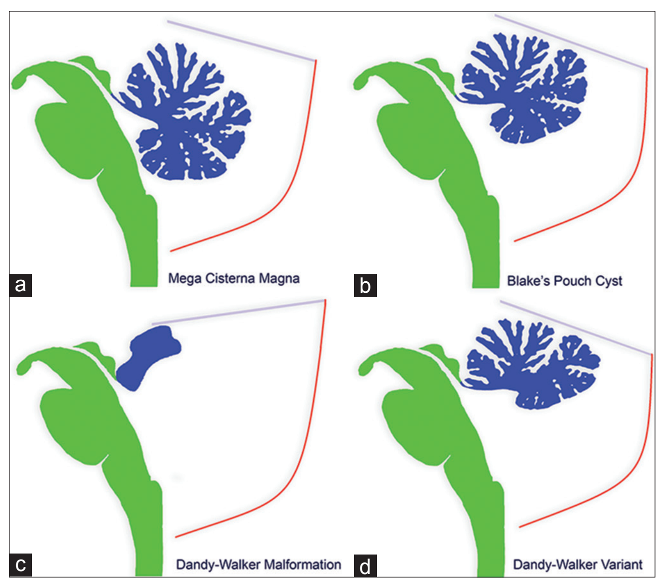

Figure 4: Diagrammatic representation of the cerebellar vermis in various entities within the Dandy–Walker complex. (a) Mega cisterna magna. Normal cerebellum and fourth ventricle. (b) Blake's pouch cyst. The vermis is relatively well-developed and nonrotated along with a cystic dilation of the fourth ventricle (c) Dandy-Walker malformation. Rotated small vermis with abnormal foliation and enlarged posterior fossa with elevation of the tentorium and torcula herophili. (d) Dandy-Walker variant. Partial vermian and cerebellar hypoplasia with a prominent retrocerebellar space. (Illustration by Waleed Azab, MD)

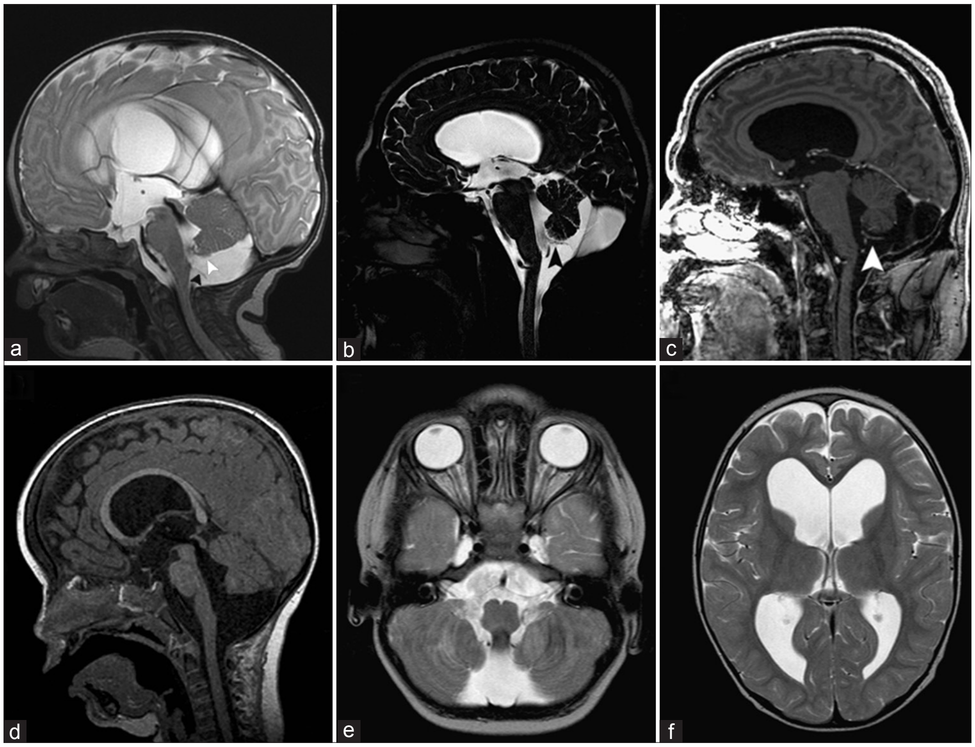

Figure 5: (a) Sagittal T2-weighted MRI image in a case with Blake's pouch cyst. Note the infracerebellar position of the cyst with compressed nonrotated relatively intact cerebellar vermis. The continuation of the fourth ventricular choroid plexus into the Blake's pouch cyst (white arrowhead) and the thin membrane demarcating the cyst from subarachnoid space (black arrowhead) are evident. (b, c) Images from one case reported by Cornips *et al.* 2011[10] demonstrating similar findings. (d-f) Another case from Cornips *et al.* 2011;[10] (d) Preoperative sagittal T1-weighted MR image demonstrating marked hydrocephalus with bulging third ventricular floor, downward bending mammillary bodies, and an open aqueduct. The cerebellum is not rotated. (e) Preoperative axial T2-weighted MR image demonstrating a bilateral indentation on the caudomedial cerebellar surface. (f) Preoperative axial T2-weighted MR image demonstrating enlarged lateral ventricles without periventricular hyperintensities

obstructive membranes in the CSF pathway.[12] It was even previously suggested that as long as the cyst wall histology usually is not known, it is best to describe posterior fossa cysts by location and effects, if any, on surrounding structures.[22]

Prenatal ultrasound usually reveals a large cisterna magna in many cases of Blake's pouch cyst,[15] however, spontaneous resolution of one-third to one-half of the cases is detected ultrasonographically before birth,[14,23] and the fenestration may not take place until 24–26 weeks of gestation.[23] Using 3D ultrasound, Paladini *et al.* suggested ultrasonographic criteria for prenatal diagnosis of Blake's pouch cysts including a normal anatomy and size of the vermis, mild-to-moderate anticlockwise rotation of the vermis and a normal size of the cisterna magna. Additionally, they were able to visualize the upper wall of the cyst in 11 out of 19 cases with choroid plexus on the superolateral margin of the cyst roof.[23]

Typically, the radiological features of a Blake's pouch cysts [BPC] are (1) tetraventricular hydrocephalus, (2) infra- or retrocerebellar localization of the cyst, (3) a relatively well-developed, nonrotated cerebellar vermis, (4) a cystic dilation of the fourth ventricle without cisternal communication, and (5) some degree of compression on the medial cerebellar hemispheres. Ideally, one may see the fourth ventricular choroid plexus continuing in the roof of the cyst on sagittal MR images.[10] Tortori-Donati *et al.* stressed on the presence of tetraventricular hydrocephalus to make a diagnosis of Blake's pouch cyst.[31] Nelson *et al.* noted that Blake's pouch cysts have a radiographic appearance similar to arachnoid cysts. If the choroid plexus is elongated or displaced under the inferior surface of the vermis to extend along the superior cyst wall, a persistent Blake's pouch should be considered. Care should be taken, however, to differentiate a choroid plexus from a prominent inferior vermian vein, which can be followed to the straight sinus. Choroid plexus is absent in Dandy–Walker malformation, and normal in arachnoid cyst.[22]

Other imaging features include a large posterior fossa and anterior displacement of the brainstem against the clivus.[28] In most of the cases, the falx cerebelli is present and the torcula is in normal position, however, an elevation of the torcula may be seen,[22] likely due to pushing of the developing tentorium into a relatively high position with an appearance of a nonspecific retrocerebellar cyst.[13] Other CNS malformations are rarely associated with Blake's pouch cysts.[22,29]

Table 1: Clinical presentations, treatment and outcome of the series by Cornips *et al.*, 2010[10]

Age, sex	Clinical presentation	Treatment	Outcome
1 Mo, M	Vomiting, Opisthotonus, Dist. conscious level , Tense fontanelle , Congested scalp veins	ETV	Died
28 Mo, M	Delayed gait development, Large head	ETV	Improved
33 Mo, M	Largehead, Open ant. fontanelle, Congested scalp Veins	ETV	Improved
22 Mo, F	Listeria meningitis, Abducent palsy, Dist. conscious level	Temporary EVD, No further treatment	Improved
69 Y, M	Gait disturbance, Memory disturbance, Occasional dysphasia, Sleepiness, Headaches, Nausea ,Blurred Vision, Nocturnal polyuria, Episodes of ophistotonus	ETV	NA
51 Y, M	Accidental , Post-head injury	None	Uneventful

Ant.: Anterior, Dist.: Disturbed, ETV: Endoscopic third ventriculostomy, EVD: External ventricular drain, F: Female, M: Male, Mo: Months, NA: Not available, Y: Year

Blake's pouch cysts usually communicate with the fourth ventricle.[8,22] In one study using cine phase contrast MRI, Yildiz *et al.* found that in patients with Blake's pouch cysts there was no flow between the cyst and the posterior cervical subarachnoid space and hyperdynamic pulsatile flow synchronized with the cardiac cycle was demonstrated within the third ventricle, aqueduct as well as the fourth ventricle in a pattern similar to that seen in communicating hydrocephalus. No CSF flow signal was detected within the cyst and no clear communication between the fourth ventricle and cyst or between the cisterna magna and cervical subarachnoid space. These authors explained the apparent lack of flow between the fourth ventricle and the Blake's pouch cyst by an incomplete coverage of the cyst by the scan volume or a small outlet of the cyst that is perpendicular to the plane of the slices.[36]

CLINICAL PRESENTATION AND TREATMENT

Clinically, Blake's pouch cysts may present with impaired neurological development, progressive hydrocephalus in young age, become symptomatic in adulthood or remain asymptomatic.[10] In over 90% of the surviving neonates with Blake's pouch cyst diagnosed on prenatal ultrasound, no associated anomalies were found and a normal developmental outcome was achieved at 1-5 years.[14] In very rare instances, Blake's pouch cyst has been reported in association with Beckwith–Wiedemann syndrome,[16] cardiac anomalies, and trisomy 21.[23]

The number of case series of Blake's pouch cyst and therefore data on its clinical spectrum and treatment options remains very limited. Gilles and Rockett in 1971 treated cysts thought to be originating from remnants of Blake's pouch with cyst wall excision and ventriculoureteral shunt insertion.[18] Arai and Sato reported a 17-month old girl who presented with mental retardation, hypotonia, and gait disturbance and was treated with a ventriculoperitoneal shunt, cyst wall excision, and a cystoperitoneal shunt.[2] Calabrò *et al.* reported two female patients aged 61 and 62 years, respectively. The first case was presented with syncopal attacks lasting several minutes and her examination revealed mild horizontal nystagmus. The second case was presented with recurrent headaches, vertigo, and frequent falls, her examination revealed mild papilledema, mild horizontal nystagmus, and imbalance on Romberg's and tandem gait testing. No information on treatment was offered.[8] Conti *et al.* reported a 37-year-old female with a BPC and a holocord syrinx whose clinical findings were all related to the cord pathology. Initial improvement after decompressive craniectomy and cyst fenestration was followed by a rapid relapse and a ventriculoperitoneal shunt was inserted.[9] Cornips and colleagues were the first to specifically report on the clinical presentation and treatment of Blake's pouch cyst [Table 1]. In their series of six patients, all patients had radiological evidence of tetraventricular hydrocephalus. Hemorrhage into the cyst occurred in one patient who had an associated biliary atresia of unknown cause. They demonstrated that endoscopic third ventriculostomy [ETV] is a valid option of treating this entity.[10] Warf *et al.* have also successfully used ETV to treat three cases of mega cisterna magna in which the authors thought the diagnosis might be Blake's pouch cyst.[32]

CONCLUSION

Blake's pouch cyst is a rare entity that is thought to belong to the Dandy–Walker continuum. It has a variable clinical presentation and when symptomatic can be treated with an endoscopic third ventriculostomy or shunting.

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