Syndrome de dandy walker pdf

Congenital Malformation of the Little Brain Dandy-Walker Malformation Co-NameDandy-Walker Syndrome (DWS),[1] Dandy-Walker Complex (DWC),[2] Dandy-Walker Continuum[3]Dandy-Walker Variant (DWV) T2 Weighted Sagtic MRI, with pony dysplasia and little brain vermis at 8-year-oldSpecialtyMedical genetics SymptomsHydrocephalus: increasing head size, vomiting, excessive drowsiness, irritability, vertical eye paralysis, convulsions[4]Related genetic conditions : congenital heart defects, eye abnormalities, intellectual disability, age of corpus callosum, skeletal abnormalities, etc. [5] Complications Main failure (displacation, overflow), subdural haematoma, infection[6]TypesDandy-Walker variant (DWV),[7][6] mega cisterna magna(?) [6] [8] causes ciliopathic or chromosome genetic diseases, often unrecognizable[5]Diagnostic methodMRI, prenatal ultrasound or CT[6]Differential diagnosisBlaken bag cyst (BPC),[3][8] mega-tank magna(?),[6][8] posterior fossa arachnoid cyst[6] [9]TreatmentCystoperitoneal shunt, ventriculoperitoneal shunt, endoscopic Third Ventriloquist (ETV)[6][10]Prediction15% risk of death, mostly hydrocephus or its treatment[6]Frequency1 from 25,000 to 1 in 50,000[5][11] Dandy-Walker deformity (DWM) , also known as Dandy-Walker Syndrome (DWS), is a rare congenital brain malformation in which the part connecting the cerebrospinal two hemispheres (cerebrospinal vermis) is not fully formed, and the fourth chamber and condition behind the cerebrospinal brain (posterior fossa) is enlarged by cerebrospinal fluid. Most sufferers develop hydrocephalus during the first year of life[6], which can occur as a reduction in head size, vomiting, excessive drowsiness, irritability, downward deviation of the eyes and seizures. [4] Other rare symptoms are usually associated with congenital genetic diseases and may include congenital heart defects, eye abnormalities, intellectual disability, congenital tumors, other brain damage such as corpus callosum age, skeletal abnormalities, skeletal encephalocele or under-developed genitals or kidneys. [5] It is sometimes found in adolescents or adults due to mental health problems. [5] [6] DWM is usually caused by a ciliopathic or chromosome genetic condition, although causality has only been identified by about half of those diagnosed before birth[6] and one third of those diagnosed after birth. [5] The mechanism is associated with a decrease in cell displacement and distribution, affecting a long period of development of the vermi of the measter. [6] The mechanism by which the hydrokephalus is present with DWM is not yet fully understood. [6] The condition is diagnosed with AN MRI or less frequent foetal ultrasound. [6] There are other malformations that can strongly resemble DWM, and there is disagreement about the criteria and ratings used in the deformity. [5] [12] Most people's treatment involves implanting a brain shunt in infancy. This is usually added to the posterior fossa, but shunt can be used in the side chambers instead or together. Endoscopic third ventriculostomy (ETV) is a less invasive option in patients over 1 year of age. Posterior fossa shunts are the most effective (80% of the time), but comes with the highest risk of complications, while ETV is the least effective, but has the lowest risk of complications. [6] Mortality is approximately 15 %, mainly due to complications caused by hydrokephus or its treatment, which may include subdural haematoma or infection. [6] After successful treatment with hydrokephalus, the prognosis is generally good, but depends on all associated burdens and its symptoms. [5] [6] Those without hydrokephalus are treated for associated symptoms or status. [13] The prevalence of DWM is estimated to be one in 25 000 to 1 in 50 000. [5] [11] DWM causes approximately 4,3 % of the cases of congenital hydrocephalus[14] and 2,5 % of all hydrocephalus cases. [6] At least 21 % of the DWM has a sibling with a deformed form and at least 16 % have a parent with a deformity. [5] The deformity was first described by the English surgeon John Bland-Sutton in 1887[6][15], although it was named by the German psychiatrist Clemens Ernst Benda [de] in 1954[1][6] after the American neurosurgeons Walter Dandy and Arthur Earl Walker, who described it in 1914 and according to Arthur Earl Walker, who described it in 1914 and Arthur Earl Walker. [6] [16] [17] Signs and symptoms The most common and visible symptoms of Hydrocephalus DWM are hydrocephalus-related symptoms during the postpartum period. Hydrokefalus occurs in an estimated 80% of patients with classical DWM. This usually occurs in the first year of life (85% of the time), most often in the first 3 months. [6] Hydrocephaly signs in babies include increasing head size, vomiting, excessive drowsiness, irritability, eye drop orientation (known as sunrise eyes) and convulsions. [4] Unlike classical DWM, only about 30% of those with the Dandy-Walker variant (DWV), where the back fossa is not enlarged, have a hydrocephalus. [6] Neurological Despite hypoplastic meacular haem, just over half of people with DWM (27-84%) there does not appear to be a significant intellectual disability or developmental delay. [5] [18] However, many genetic damage associated with DWM can cause developmental delays and other brain abnormalities. [5] [6] Corpus callosumin agenesis has been found in 5-17% of those receiving DWM. [10] [19] However, this alone does not appear to lead to intellectual disability. [18] Other brain abnormalities known to sometimes be associated with DWM include grey matter heterotopia, pachygyria (fewer ridges in the brain), lissencephaly (lower ridges), polymicrogyr, holoprosensphal and [6] [10] People with these characteristics usually have developmental delays or seizures. Those without other CNS abnormalities tend to have normal or almost normal intellectual intellectual In 2003, the review found that moderate to severe intellectual disability and non-DWM brain abnormalities occurred only in those with the most severe cere-brain vermis malformations (less than two cracks/three lobuls in the vermis) and made up 16 percent of the sample. All these patients were also affected by hydrocephalus. [12] In the Dandy-Walker variant (DWV), and in particular in mega-cisterna magna, which are milder malformations, the number of psychotic spectrum disorders such as schizophrenia, bipolar disorder, mania or catathonia appears to have increased. [2] [5] [20] Related abnormalities In 2017 2017, 27% of patients had congenital heart defects, the following links in patients with DWS (usually associated genetic condition or abnormality):[5] 27% of patients had congenital heart defects. These included patented duct artery disease, aorta coarchation, ventricular septic defect and eternity. Heart failure was reported in 2.7% of patients. [5] 24% of patients had at least one eye abnormality. These included cataracts, small eyes (microphthalmia), chorioretinal dysplasia/atrophy, optic nerve dysplasia/atrophy, small cornea (microcorn) or corneal opaqueness (jaw), short-sightedness (myopia) and colophobia (hole in the eye structure). [5] 16% of patients were diagnosed with a mental or behavioural disorder, and 6.4% also had learning difficulties. 5.3 percent had either bipolar disorder or psychotic spectrum disorder and 2.1 percent had ADHD. Slightly more of these were found in the Dandy-Walker variant (DWV) than in the classic DWM, although DWV was less common, in only about 20 percent of DWS diagnoses. [5] Approximately 12% of patients had cancers or tumours due to congenital genetic abnormalities. The most common were neurokutan melanosis (5.9%), hemangioma (4.8%, including those with PHACE syndrome) and Wilms tumour (4.4%). In these cases, melanovate tumors are thought to be associated with the same genetic defects in the development of the embryonic neural tube that lead to DWM, since the subsequent embryonic neural coat of arms causes, among other things, melanocytes. [5] 10% of patients had endocrine or metabolic disorders, and 2.7% had excessive hair growth (hypertrichosis). [5] Nine% of patients (almost all with classical DWM) had musculoskeletal abnormalities, including scoliosis or cyphosclosis and joint vaccine. [5] 5.9% of patients had under-developed reproductive organs such as hypoplastic genitals or unwanted testicles (cryptorchidism). [5] 5.3% of patients had an under-developed or polycystic kidney. [5] Takaraivo encephalocele may occur in DWM. [6] This has generally been established at an interest rate of between 6 % and 8 %. [21] [22] [19] It has been suggested that it compensates for the increased pressure of fossa in the foetal during fossa in the foetus during foetal life. [6] Syringomyel occurs with DWM, although it is not certain how often. [6] [23] According to one review, the sample represented 4,3 %. [7] [7] may be due to peaation of the cyst base via foramen magnum (a mechanism similar to chiari malformations). Alternatively, it may be caused by a hydrokephus, where it is formed as the fifth chamber due to the enlarged central canal. [6] Rarely has spina bifida been found on DWM. When it is present, it is usually spina bifida occulta. [24] The cause of the location of the fourth chamber (E) in red, between the brain and ponies (B) DWM is due to embryonic development disorders that affect the formation of the vermises of the little brain. This is usually a genetic mutation that leads to a decrease in cell displacement and distribution. A large number of genetic conditions can lead to an abnormality. In a large number of DWM cases, the status is identified by the person concerned, but in most cases the cause is not identified. At least 21 % of the DWM has a sibling with a deformed form and at least 16 % have a parent with deformed data. [5] Ciliopathic Genetic Conditions Main article: Ciliopathy The genetic condition is identified in approximately 33% of those diagnosed with DWM after birth. [5] In 2017, 4.3% were diagnosed with PHACE syndrome with brain, cardiovascular and eye aberries, while 2.3% had Joubert syndrome, which was associated with neurological and sometimes eye and kidney aberries. Between 21% and 81% of people with Phace syndrome have DWM. [25] [26] Other comorbidic genetic diseases found included oculokerebrokutan syndrome, oral digital syndrome, Coffin– Siris syndrome, Type 7 Meckel– Gruber syndrome and Kallmann syndrome. [5] DWM has also been associated with 3C syndrome, Rubinstein-Taybi syndrome, Marden-Walker syndrome, Sheldon-Hall syndrome, Shah-Waardenburg syndrome, Fryns syndrome,<[27] Walker-Warburg syndrome, Fukuyama's congenital muscular dystrophy, Ellis-van Creveld syndrome, Fraser syndrome, Aicardi syndrome, Cornelia de Lange syndrome,<[10] Klippel-Feil syndrome[28][29] and acrocallosaal syndrome,[30] mm. Many of these disorders are classified as ciliopathy, genetic disorders that affect primary cilia in cells, projections of thin cells made of microtubules that are believed to be crucial to signaling the distribution and migration of embryonic cells. [31] DWM is one of the largest predictors of ciliopathic genetic disease. [32] Other genes associated with DWM include ZIC1, ZIC4, FOXC1, FGF17, LAMC1 and NID1. [5] Chromosome abnormalities In those diagnosed with DWM prior to birth by ultrasound, up to half are diagnosed with chromosome abnormalities[6], the most common being Edwards syndrome (trisosomes 18), in approximately 26% of fetal DWM cases. [27] 6.5% of those diagnosed with DWM after birth also have Edwards syndrome. [5] Other chromosome ailations that may lead to DWM include: Patau syndrome (trisomia 13), trisomia 9 and partial removal or overlap of 3q. [5] [6] The 3q24 range includes ZIC1 and genes known to be related to DWM. [6] [27] External toxins During pregnancy the use of Warfarin has been known to lead to systemic defects in the foetus, such as ophthalmological dysgenesis, microcephaly, aging of corpus callosum, skeletal abnormalities and heart defects. In 1985, it also joined the DWM. [33] Patophysiology diagram of the meana, fourth chamber and pony. The white arrow shows the magendie (media opening) superiors connecting the fourth chamber to cisterna magna (3). This usually stays open on DWM. [6] The little brain begins to form in the fifth week of embryonic development. It stands out at the top of the metencephalon, while ponies (in the brain stem) stand out at the bottom, separated by a fourth chamber. The hemispheres of the little brain are formed by rhombic lips on the front surface of the fourth chamber, which expand and turn to the midline fuse, forming the vermi of the meaque 15th chamber. If this process is not completed, the vermis of the baby brain will not be completely formed. This long period of development of the vermis of the baby brain makes it especially susceptible to disorders. [6] With DWM, the fourth chamber opens and is continuous with almost the entire subaracnoid state of the back of the fossa. [6] Hydrokephalus pathophysiology The cause of hydrokephalus' in DWM is not yet fully understood. The earliest authors had put it in the clogging or narrowing of magends and Luschka's ancestors, which were the two openings in the fourth chamber that allow cerebrospinal fluid (CSF) to escape into the subaracnoid state of the back fossa. However, subsequent studies found that these foraminas tend to be open on DWM. [6] Hydrokefalus is also generally not (80% of the time) present at birth with DWM. [6] Csf flow loss may be outside the outlets of the fourth chamber. Theories of abnormal development or inflammation of the mother of arachnoid have been made in the fossa of the butt. [6] Mother Arachnoid contains granules necessary to restore brain and brain and brain and acolyees from subaracnoid states to dural use containers and blood circulation. Cyst removals of DWM have not been able to demonstrate whether it is impaired absorption of aracnoids, since subaracnoid status always takes days or weeks after removal. [6] Aqueductal htauma (narrowing the passage between the third and fourth chambers) does not appear to be a factor in DWM. It is usually open, and shunts placed on the backside from fossaky almost always empty all the chambers. When present, it can be caused by compression of herniated vermis or cyst or related developmental disorder. [6] It is known that once the hydrokephalus has started, the compression of the butt fossa cyst at the counts of the aracnoid mother participates in worsening pathology. [6] Diagnosis of Dandy-Walker deformity is diagnosed based on typical neuroimaging findings. It can be diagnosed ultrasound for 14 weeks of pregnancy, although it is usually diagnosed postnataly by MRI. It is diagnosed in the first year of life 41% of the time, usually due to signs of an increasing hydrocephus,[18] but 28% of the time it is found in adolescence or adulthood due to mental health problems such as psychosis or mood disorder. [5] [6] Criteria and classification of DWM's precise diagnostic criteria and classification systems have not been agreed and there is a significant dispute as to which conditions or criteria should be used. [5] [6] [12] The core criteria for DWM are the hypoplasia of the vermice of the small brain and the enlarged fourth chamber and the back fossa (the space behind the little brain), although no specific level of hypoplasia or cystic enlargement has been agreed for the diagnosis of DWM. [7] In addition, there are several similar conditions that some authors have grouped with DWM at different times into a continuum and separated separately, making the diagnosis even more difficult. [6] [8] In 1976, Harwood-Nash and Fitz proposed the term Dandy-Walker variant (DWV) for a deformity in which the rear fossa is not enlarged, but the vermin of the metica thumb brain is hypoplastic. [7] [6] In 1989 Barkovich et al. proposed the term Dandy-Walker complex (DWC), which included the classic DWM and DWV (under type A) and the third deformity (under type B), in which the vermis of the baby brain remains large enough to sit between the fourth chamber and the magnana of the container underneath it, and instead mainly an enlarged tank magnation. In this type, the vermic hypoplasia of the small brain does not get past the horizontal midline of the fourth chamber, and the back fossa is not equal either. The authors noted that this form would previously have been classified simply as mega-cisterna magna. [2] [6] In 1999, Calabró et al. first used the phrase Dandy-Walker continuum to refer to suggestions that the condition known as Blake's bag ply falls under the umbrella of the Dandy-Walker complex proposed by Barkovich. [3] Subsequent authors would look closely at these terms and systems and find that they have added significant confusion to the diagnosis of dwm. [5] [6] [12] However, they are still commonly used. [5] In 2011, Spennato and Others v Commission Posterior fossa (the space behind the cerebrospinal brain) has increased, and its flow of cerebrospinal fluid is constant with the fourth chamber. The rest of the vermis in the little brain is hypoplastic and pushed upwards and rotated forward due to the fossa of the enlarged back. The hemispheres of the little brain are pushed forward and to the side with the help of an enlarged butt fossa. The angle in the center of the vermis of the meager brain (representing the position of the fastigial core) is large, flattened appearance at the bottom of the vermis or fastigial core is completely missing. Due to the enlarged butt fossa, the joint confluence of the sinuses is elevated to the emptying system at the back of the rear of the end of the head section. (The adjacent tentorium of the little brain is also elevated.) Since the presence of hydroencephalus in DWM is inconsistent, Spennato and Klein suggested that it should not be considered a criterion for DWM. [6] [12] Klein's criteria differed mainly from the Spennto criteria in that it did not require obvious cerestrum hemisphere hypoplasia, but it may also have required vermis to touch tentorium or brain stem apostasies. [12] Methods DWM can be detected prenataally by ultrasound as early as 14 weeks of pregnancy[5], although MRI is the most useful diagnostic method. Mri scans can determine the shape and extent of the deformity and assess additional areas of deformities, such as cerequenge hemispheres, brain conduction or corpus callosum. Cardiac gate phase contrast MRI scans can monitor the flow of cerebrospinal fluid during cystolen and cardiac diastolen. In the real DWM, this finds a flow from the brain aqueduct to the back of the fossa and does not flow between the magna in the reservoir and the space behind the cervical spinal cord. [6] CT may also be used if MRI is not available but has fewer details. [6] Klein et al. (2003) suggested that a suspected CT or ultrasound diagnosis should not be confirmed before an MRI scan, since there are a large number of diseases that can cause a very similar and confusing diagnosis. [12] Differential diagnosis DWM has a large number of diseases that can occur very similarly in imaging and confusing diagnosis. [6] Blake's bag pen Blake's bag pen (BPC) or permanent Blake bag is the condition that arises when Blake's pouch, invagination in the fourth chamber, which tears during about 4 months of pregnancy into magendic (medial aperture) foramen, is not torn. This can lead to an enlarged fourth chamber of all four chambers and a subsequent hydrokephal vessel. [6] Blake's pouch pen, unlike DWM:[6] The meticle is not hypoplastic, although it can be squeezed (mass effect) by the enlarged rear fossa. Posterioric fossa is not enlarged. The tentorium/grouping of the little brain is not elevated. Hydrokefalus, when it happens, contains all four chambers. However, some authors consider Blake's bag pen as part of a continuum with DWM (Dandy-Walker continuum). [3] [8] Mega cisterna magna Mega cisterna magna is a condition in which the subaraknoid reservoir below the fourth chamber cisterna magna is enlarged. It has been suggested to be due to a delayed tear in Blake's bag and not a failed rupture. [9] Mega cisterna in magna, unlike DWM:[9] The little brain is usually not hypoplastic. The fourth chamber is relatively normal. Hydrokefalus is rare. There is. whether this deformities differ from the DWM or belong to the Dandy-Walker continuum. [6] [8] Posterior fossa arachnoid cyst Main article: Arachnoid cyst Araknoid cyst is a collection of cerebrospinal fluid (CSF) in a arachnoid mat. Of these, 10% occur at the back of the fossa. [9] In the rear fossa aracnoid cyst, unlike DWM:[6][9] The cyst is clearly localised to a specific location separate from the fourth chamber socket. The little brain is not hypoplastic, although it can be squeezed by a cyst (mass effect). Cyst brain and brain yy flow is not continuous with the fourth ventricle flow. Hydrocephalus, if it occurs, is caused by pressing the cyst into the cere brain and squeezing the extincesion of the brain's water channel or fourth ventricle. Treatment The main immediate goal of treatment is to control hydrokephus and enlarged posterior fossakysta, as they can lead to increased intracnal pressure and brain damage. A minority of sufferers do not develop hydrokefalus and are treated based on the associated symptoms or condition. [13] Hydrokefalus/cyst for Hydrokefalus or the back of the fossa cyst shunts are the centre of treatment. However, patients with DWM have more shunt-related complications than other hydrocephalus patients (mainly due to unconventional anatomy). [6] One explanation for the fact that the shunt has not reduced intractual pressure in the DWM has been that the cyst can hernize the forame to the magnum and form a scarred adhesion to the cervical crossing, preventing it from shrinking again. If this occurs, duraplasty can be used to attempt to reduce suboccipital pressure with duraplasty. [6] The DWM has not agreed whether the shunt should be placed in the fourth chamber (cystoperitoneal shunt or CP-shunt), side chambers (chamber hetoneal shunt or VP shunt) or both due to conflicting studies on whether deformities affect the cerebrovascular cycle. However, the CP shunt almost always clears both the fourth and lateral chambers of the DWM, and according to strict definitions of deformity, the water conductor must be assumed to be open[6], although imaging is important to confirm this. [10] Many authors therefore recommend CP-shunt as a logical alternative. However, it is associated with major complications, including displacing and crossing. Ylidrainage can lead to subdural hematomas, bound spinal cord, scars or downward herniation of the hemispheres. Spennato et al. therefore recommends a flow control or a cymphon valve. On the other hand, VP shunts have a lower number of complications than the CP shunt, and some recommend it at first. However, they are less effective in DWM, and the elevated status of the tentorium must be considered before installing the VP shunt. [6] In patients over 1 year of age, the endoscopic third chamber (ETV) can be considered as first-line therapy. This less invasive procedure creates an artificial hole chamber so the CSF can bypass all obstacles. It cannot be used for those with brain abnormalities, such as the age of corpus callosum, since the risk of csf escapes to other brain regions. However, a compressed brain stem is not a counter-topic. Etv's success rate is more modest than the shunt, as the hole often closes. It is more likely to fail in younger patients (less than 1 year), and its effects on the developing brain are not yet known. [6] Posterior fossa craniotomy and cystic membrane removal were previously used, which often failed to prevent cyst reformation and carried some mortality. This can still be reserved for patients with recurrent shunt defects/ infections. [6] [10] Other treatments for other symptoms tend to focus on the specific disease and may include supported training, physiotherapy or other services. Genetic advice can be offered to parents for future conceptions. [13] The prognosis Depends above all on the early and successful treatment of the hydrokephal, if any. Another significant factor influencing the prognosis is the presence of a comorbid gene condition or brain ailance. [5] [6] Dwm has a mortality rate of approximately 15 %. [6] A study of the Dandy-Walker variant (DWV) showed a mortality rate of 12,5 %. [7] The most common cause of death is complications caused by hydrokephus or its treatment. [6] [18] Untreated hydrokephalus may increase intracinal pressure and brain damage. The shunt used to treat DWM has a moderate to good success rate, but they have a higher than average failure rate, which can lead to a reduction in intracchial pressure or infection such as meningitis. Complications of crossing, such as subdural hematomas, are also possible and can lead to mortality. [6] [34] In the fourth chamber (systoperitoneal shunts or CP-shunts), the reduction in cyst and ventricular size is generally high, especially in cysts (at least 80 %). Studies of the side chambers (ventricular hetoneal shunt, or VP-shunt), have generally shown about 50% successfully reduced cyst size, and the successful reduction in the size of the chamber is about two-thirds of the time. [6] DWM often has other systemic or genetic diseases, each with its own significant impact on the prognosis. [6] Epidemiology The prevalence of DWM is estimated to be one in 25 000 to 1 in 50 000. [5] [11] DWM causes approximately 4,3 % of the cases of congenital hydrocephalus[14] and 2,5 % of all hydrocephalus cases. [6] In 2017, the review found that the majority of patients (65%) either Dandy-Walker malformation or Dandy-Walker syndrome was diagnosed, while 20% were diagnosed with the Dandy-Walker variant and 1.1% with mega cisterna magna. [5] History The deformity was first described in 1887 by the English surgeon John Bland-Sutton as a hypoplasia of the vermise of the small brain, butt fossax and and In 1914, American neurosurgeon Walter Dandy and American pediatrician Kenneth Blackfan described malformations as partial or complete absence of vermises of the small brain, enlarged fourth chamber

and hydrokephus. [6] [16] In 1942, John K. Taggart, an American physician, and Arthur Earl Walker, a Canadian-American neurosurgeon, reported extensively on the phenomenon, stating the possible cause of the underinformance of luschka and Magendi preinstaggies,[17] is no longer believed to be significant. [6] German psychiatrist Clemens Ernst Benda [de] introduced the term Dandy-Walker Syndrome (DWS) in 1954. He also once used the term Dandy–Walker deformed. [1] [6] In 1976, Harwood-Nash and Fitz proposed the term Dandy-Walker variant (DWV) for a deformity in which the back fossa is not enlarged, but the verse of the meticaque brain is hypoplastic. [7] [6] In 1989 Barkovich et al. suggested the term Dandy-Walker complex (DWC) to include classic DWM and DWV (under type A) and a third malformation (under type B) in which the vermis of the metica thumb remains large enough to sit between the fourth chamber and the tank magna, and instead mainly the cisterna magna has expanded (sometimes diagnosed as mega cisterna magna). In 1999, Calabró et al. first used the phrase Dandy-Walker continuum when referring to suggestions that the condition known as Blake's bag pen falls under the umbrella of the Dandy-Walker complex proposed by Barkovich. [3] Modern authors are mostly discouraged by these additional conditions due to the confusion and complexity caused by DWM diagnosis. [5] [6] [12] References ^ a b c Benda, Clemens E. (1954-01-01). Dandy-Walker syndrome or Foramen Magendie's so-called Atresia. Journal of Neuropathology and Experimental Neurology. 13 (1): 14–29. doi:10.1093/etc/13.1.14. ISSN 0022-3069. PMID 13118372. ^ a b c Barkovich, A. J.; Kjos, B.O.; Norman, D.; Edwards, M. S. (Dec. Malformations of rear fossa cysts and cyst-like cysts have been refined based on the results of multiplanar MR imaging. Ajr. American Journal of Roentgenology. 153 (6): 1289–1300. doi:10.2214/ajr.153.6.1289. ISSN 0361-803X. PMID 2816648. ^ a b c d e Calabrò, F.; Arcuri, T.; Jinkins, J. R. (2000-04-01). Blake's baggy pen: a creature in dandy-walker's continuum. Neuroradiology. 42 (4): 290–295. doi:10.1007/s00234005088. ISSN 1432-1920. PMID 10872175. ^ a b c Hydrocephalus newsletter | National Institute of Neurological Disorders and Stroke. www.ninds.nih.gov. Referenced 2019-12-31. ^ a b c d e f g h i j k l n o p q r s t u v x y z aa ab ac ae af ag ah ai aj ak Stambolliu, Emelina; Ioakeim-Ioannidou, Myrsini; Kontokostas, Kimonas; Dakoutrou, Mary; Kousoulis, Antonis A. (2017-09-01). The most common codeant values in Dandy-Walker syndrome patients: systematic review of case reports (PDF). Journal of Pediatric Neurology. 32 (10): 886–902. doi:10.1177/0883073817712589. ISSN 0883-0738. PMID 28635420. ^ a c d e f g h i j k l m m n o q r s t u v x y z aa ab ac ae af ag ah ai aj ak al am ao ap aq ar as at au av aw ax ay az ba bb bc bd be bf bg bh bi bj bk bl bn bo bp bq br bs bt bu bv bw bx by Spennato, Pietro; Mirone and Giuseppe. Nastro and Anna. Buonocore, Maria Consiglio; Ruggiero, Claudio. Trischitta and Vincenzo. Aliberti, Ferdinando; Cinalli, Giuseppe (Oct. Hydrokefalus Dandy-Walker deformity. A child's nervous system. 27 (10): 1665–1681. doi:10.1007/s00381-011-1544-4. ISSN 1433-0350. PMID 21928031. ^ a b c d e f Sasaki-Adams, Deanna; Elbabaa, Samer K.; Jewelry, Valerie. Carter and Lori. Campbell, Jeffrey W.; Ritter, Ann M. (Sept. Dandy-Walker variant: a series of cases of 24 paedal patients and an assessment of related abnormalities, hydrocephus inequacies and developmental outcomes. Journal of Neurosurgery. Pediatrics. 2 (3): 194–199. doi:10.3171/PED/2008/2/9/194. ISSN 1933-0707. PMID 18759601. S2CID 16364934. ^ a b c d e f Azab, Waleed A.; Shohoud, Sherien A.; Elmansoury, Tamer M.; Salaheddin, Waleed; Nasim, Khurram; Parwez, Aslam (2014-07-24). Blake's baggy pen... Surgical Neurology International. 5: 112. doi:10.4103/2152-7806.137533. ISSN 2229-5097. PMC 4123264. PMID 25101207. ^ a b c d e Bosemani, Thangamadhan; Orman and Gunes. Boltshauser, Eugen; Tekes, Aylin; Huisman, Thierry A. G. M.; Poretti, Andrea (2015-01-01). Posterior Fossa's congenital abnormalities. Radiography. 35 (1): 200–220. doi:10.1148/rg.351140038. ISSN 0271-5333. PMID 25590398. ^ a b c d e f Kollias, S. S.; Ball, W.S.; Prenger, E. C. (Nov. Cystic malformations of posterior fossa: differential diagnosis clarified by embryo analysis. Radiography. 13 (6): 1211–1231. doi:10.1148/radiography.13.6.8031352. ISSN 0271-5333. PMID 8031352. ^ a b c Orphanet: Isolated Dandy Walker deformity. www.orpha.net. Referenced 2019-12-30. ^ a b c d e f g h Klein, O.; Pierre-Kahn, A.; Boddaert, N.; Parisot, D.; Brunelle, F. (Aug. Dandy-Walker deformities: fetal diagnosis and prognosis. A child's nervous system. 19 (7–8): 484–489. doi:10.1007/s00381-003-0782-5. ISSN 0256-7040. PMID 12879343. ^a b c Dandy Walker's deformities. NORD (National Association for Rare Disorders). Retrieved 2020-01-06. ^ a b Lumenta, Christianto B.; Skotarczak, Ulrich (1995-03-01). Long-term follow-up in 233 patients with congenital hydrokephalus. A child's nervous system. 11 (3): 173–175. doi:10.1007/BF00570260. ISSN 1433-0350. PMID 7773979. ^ a b Sutton, J. Bland (1886-10-01). Lateral downsising of the fourth chamber; Their relationship to certain small-brain cysts and tumors, as well as the back of the head to Meningocele. Brain. 9 (3): 352–361. doi:10.1093/brain/9.3.352. ISSN 0006-8950. ^ a b Dandy, Walter E.; Blackfan, Kenneth D. (1914-12-01). EXPERIMENTAL, CLINICAL AND PATHOLOGICAL EXAMINATION: Part 1.—Experimental studies. American Journal of Diseases of Children. VIII (6): 406-482. doi:10.1001/archpedi.1914.02180010416002. ISSN 0096-8994. ^ a b Taggart, K.; Walker, A. Earl (1942-10-01). Luschka and Magendie's Foramen-born Atresia. Neurology and psychiatry archives. 48 (4): 583–612. doi:10.1001/archneurpsyc.1942.02290100083008. ISSN 0096-6754. ^ a b c d e Bindal, Ajay K.; Storrs, Bruce B.; McLone, David G. (1990). Dandy-Walker Syndrome Control. Pediatric neurosurgery. 16 (3): 163–169. doi:10.1159/000120518. ISSN 1016-2291. PMID 2134009. ^ a b Kumar, Raj; Jain and Manoj. Chhabra, Devendra (2001-05-01). Dandy-Walker syndrome: different treatment and outcome procedures in 42 cases. A child's nervous system. 17 (6): 348–352. doi:10.1007/s003810000425. ISSN 1433-0350. PMID 11417415. ^ Pandurangi, Swapna; Pandurangi, Aditya; Matkar, Abhay; Shetty and Nithin. Patil, Preetam (January 2014). Psychiatric manifestations associated with Mega Cisterna Magna. Journal of Neuropsychiatry and Clinical Neuroscience. 26 (2): 169–171. doi:10.1176/appi.neuropsych.13040097. ISSN 0895-0172. PMID 24763763. ^ Bindal, A. K.; Storrs, B.B.; McLone, D. G. (1990–1991). Dandy-Walker Syndrome Control. Pediatric neurosurgery. 16 (3): 163–169. doi:10.1159/000120518. ISSN 1016-2291. PMID 2134009.CS1 maint: date format (link) ^ Forzano, F.; Mansour, S.; Ierullo, A.; Homfray, T.; Thilaganathan, B. (2007). Posterior fossa deformed foetuses: report on 56 additional cases and literature review. Prenatal diagnosis. 27 (6): 495–501. doi:10.1002/sd.1722. ISSN 1097-0223. PMID 17367101. ^ Hammond, Christopher J.; Chitnavis, Bhupal; Penny, Christopher C.; Strong, Anthony J. (2002-01-01). Dandy-Walker Complex and Syringomyelia in adulthood: case report and discussion. Neurosurgery. 50 (1): 191–194. doi:10.1097/00006123-200201000-00028. ISSN 0148-396X. PMID 11844250. ^ Golden, Jeffrey A.; Rorke, Lucy B. Bruce, Derek A. (1987). Dandy-Walker syndrome and related abnormalities... Pediatric neurosurgery. 13 (1): 38–44. doi:10.1159/000120299. ISSN 1016-2291. PMID 3684814. ^ Metry DW, Dowd CF, Barkovich AJ, Frieden IJ (July 2001). Many faces of PHACE syndrome... J. Pediatr. 139 (1): 117–23. doi:10.1067/mpd.2001.114880. PMID 11445804. ^ Poetke, M.; Frommeld, T.; Berlien, H. P. (Dec. PHACE syndrome: new insights into diagnostic criteria. European Journal of Pediatric Surgery. 12 (6): 366–374. doi:10.1055/s-2002-36849. ISSN 0939-7248. PMID 12548487. ^ a b c Imataka, George; Yamanouchi and Hideo. Arisaka, Osamu (2007). Dandy-Walker syndrome and chromosome apostasies. Congenital abnormalities. 47 (4): 113–118. doi:10.1111/j.1741-4520.2007.00158.x. ISSN 1741-4520. PMID 17988252. ^ Pascual-Castroviejo, I.; Velez, A.; Pascual-Pascual, S.I.; Roche, M.C.; Villarejo, F. (1991-04-01). Dandy-Walker deformities: 38 cases. A child's nervous system. 7 (2): 88–97. doi:10.1007/BF00247863. ISSN 1433-0350. PMID 1863935. ^ Asai, Akio; Hoffman, Harold J.; Hendrick and Bruce. Humphreys, Robin P. (1989). Dandy-Walker Syndrome: Experience at a hospital for sick children in Toronto. Neurosurgery. 15 (2): 66–73. doi:10.1159/000120445. ISSN 1423-0305. PMID 2635298. ^ OMIM Entry - #200990 – ACROCALLOSAAL SYNDROME; ACLS. www.omim.org. Retrieved 2020-01-06. ^ Baker, Kate; Beales, Philip L. (2009). Making Cilia think in disease: human ciliopathies. American Journal of Medical Genetics Part C: Seminars on Medical Genetics. 151C (4): 281-295. doi:10.1002/ajmg.c.30231. ISSN 1552-4876. PMID 19876933. ^ Badano JL, Mitsuma N, Beales PL, Katsanis N (2006). Ciliopathies: an emerging class of human genetic disorders. Annu Rev Genom Hum Genet. 7: 125–48. doi:10.1146/annurev.genom.7.080505.115610. PMID 16722803.12.2003. S2CID 40223129. ^ Kaplan, Lawrence C. (1985). Innate Dandy Walker deformity associated with the first trimester warfarin: case report and literary review. Teratology. 32 (3): 333–337. doi:10.1002/tera.1420320302. ISSN 1096-9926. PMID 4082063. ^ Yüceer N, Mertol T, Arda N (2007). Surgical treatment for 13 paediat child patients with Dandy-Walker syndrome. Paediatrin neurosurgeon. 43 (5): 358–63. doi:10.1159/000106383. PMID 17785999. This article contains public material from the Dandy-Walker Syndrome Information Page of the U.S. Government. Read more of Metry Phaces' article in the Journal of Pediatrics, July 2001 E-Medical Website Definition Dandy–Walker Deformity Article Pan Arab Journal of Neurosurgery External Links ClassificationDICD-10: Q03.1ICD-9-CM: 742.3OMIM: 220200MeSH: D003616DiseasesDB: 3449Oly Resourcesmedicine: radio/206 Retrieved from