



Incidental Findings on Brain and Spine Imaging in Children

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abstract

In recent years, the utilization of diagnostic imaging of the brain and spine in children has increased dramatically, leading to a corresponding increase in the detection of incidental findings of the central nervous system. Patients with unexpected findings on imaging are often referred for subspecialty evaluation. Even with rational use of diagnostic imaging and subspecialty consultation, the diagnostic process will always generate unexpected findings that must be explained and managed. Familiarity with the most common findings that are discovered incidentally on diagnostic imaging of the brain and spine will assist the pediatrician in providing counseling to families and in making recommendations in conjunction with a neurosurgeon, when needed, regarding additional treatments and prognosis.

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www.pediatrics.org/cgi/doi/10.1542/peds.2015-0071

DOI: 10.1542/peds.2015-0071

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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INTRODUCTION

The importance of brain imaging in contemporary medical practice can hardly be exaggerated, and technological advances have been matched by expansion of access to this technology. When the senior author entered medical school, there was precisely 1 new first-generation computed tomography (CT) unit at his prestigious referral teaching hospital. Now, much more sophisticated devices are found even in small facilities. In recent years, the use of diagnostic imaging of the brain has expanded dramatically, and requests for consultation to assess the meaning of unexpected findings have multiplied. Although insurance carriers scrutinize them, few population-based data have been published describing actual numbers of studies performed, particularly among children. In every year from 2008 through 2012 in the state of Delaware, slightly more than 2% of all children enrolled in Medicaid underwent CT scanning or MRI of the brain (P. White, Delaware Division of Medicaid and Medical Assistance, personal communication, 2013). A recent review of 6 large integrated health systems in the United States found that MRI use quadrupled between 1996 and 2010, reflecting a 10% annual growth rate.¹ MRI use is influenced by many factors, including increasing availability, patient-generated demand, and “defensive” practice.² Although rates of MRI use vary substantially by geographic region within

the United States,^{3,4} overall MRI use is substantially higher in the United States than in most other industrialized countries. In the most recent data from the Organization for Economic Cooperation and Development, MRI use in the United States per 1000 population was 97.7, the highest rate in the survey and more than double the Organization for Economic Cooperation and Development average.⁵ As a result, the increase in incidental brain and spine findings is of particular importance in the United States. An unintended consequence of such widespread diagnostic imaging is the discovery of many incidental findings unrelated to the original reasons for the studies.

BENIGN ENLARGEMENT OF THE SUBARACHNOID SPACES

Benign enlargement of the subarachnoid spaces (BESS), also known as “benign external hydrocephalus,” is a transient developmental phenomenon that is really a variant of normal and is commonly seen in neurosurgery practice.^{6–8} Because the indication for the brain imaging that discloses BESS is usually macrocephaly, it may not be an incidental finding in a strict sense, but it is incidental inasmuch as treatment is seldom necessary. In the era of CT scanning, it was the focus of some confusion, the term “benign subdural effusions of infancy” suggests, but contemporary ultrasonography with Doppler or MRI can make reliable distinctions between subdural fluid collections and prominence of the subarachnoid spaces. Likewise, the term “external hydrocephalus” suggests a disturbance of cerebrospinal fluid (CSF) physiology, which has never been substantiated experimentally.

The clinical picture is fairly consistent. Typically, the affected patient exhibits accelerated head growth in midinfancy but otherwise thrives. Often, 1 parent has a head circumference at or beyond the 95th

percentile. Despite the presence of macrocephaly, the fontanel is slack and there is no suture separation. Brain imaging may show a minor degree of ventriculomegaly that is not in proportion to the expansion of the subarachnoid spaces. Although seldom documented because of its benign clinical course, the natural history of BESS is resolution later in childhood; and from a clinical standpoint, the child’s head circumference can be expected to drift gradually back toward the top of the normal range over a period of years.⁹ The developmental prognosis of BESS is the topic of some discussion in the literature and remains imprecisely defined, even after imaging mimickers, such as achondroplasia, Sotos syndrome, and mucopolysaccharidoses, have been excluded.^{8,10,11} Generally, after initial gross motor delays attributable to the excessive size of the head, development is normal, but careful observation is warranted.

Neurosurgical referral is not necessary in the absence of ventricular enlargement or subdural fluid collections unless parents require the reassurance of the subspecialist. Although a degree of ventricular enlargement may accompany BESS, a report of ventricular enlargement raises a question of hydrocephalus that must be addressed by a neurosurgeon. Likewise, a description of subdural hygroma or chronic subdural hematoma indicates referral.^{6,12–14} In addition, if the interpretation of the initial imaging study fails to distinguish between BESS and subdural hematoma or hygroma, neurosurgical referral is indicated necessarily as well. The disproportion between the volume of the cranium and the volume of the brain that characterizes BESS is widely believed to create susceptibility for development of subdural fluid collections, but the finding of a subdural collection cannot be dismissed. A recent cohort

study identified subdural collections in 4 of 177 young children with BESS (2.3%), 1 of whom was determined to be a victim of abuse.¹⁵ Even in the setting of BESS, chronic subdural hematoma still constitutes an indication for investigation of the possibility of abuse, including dilated funduscopic examination and radiographic skeletal survey.¹⁵

CHOROID PLEXUS CYST

Choroid plexus cysts are common findings on antenatal ultrasonography in the second trimester. Prevalence rates between 0.6% and 2.3% have been reported.^{16–19} Choroid plexus cysts are of some significance in perinatal medicine because of their association with fetal aneuploidy in the setting of other risk factors. Although Shuangshoti and Netsky²⁰ reported small choroid plexus cysts in the majority of unselected autopsy specimens, they are infrequent incidental findings on brain ultrasonography in infancy and are seldom noted on brain imaging studies in older children and adults. Thus, choroid plexus cysts in the fetus are generally believed to regress before or shortly after birth. The neurosurgical literature contains many case reports of choroid plexus cysts that were symptomatic at presentation from obstructive hydrocephalus, but we are aware of only a single example in print of a cyst detected in the second trimester that persisted to term and progressed postnatally to become a clinical problem.²¹ Thus, there seems to be little cause for concern about postnatal neurosurgical complications of antenatally detected choroid plexus cysts. Indeed, the authors are unable to reference any published guidelines for follow-up imaging in infancy. Incidentally detected choroid plexus cysts associated with mass effect or hydrocephalus certainly require neurosurgical referral. There is no

evidentiary basis for recommendations about smaller cysts, but neurosurgical consultation is likely to prove more conclusive and less expensive than sequential imaging studies.

CHOROIDAL FISSURE CYST

The choroidal fissure can be found on the mesial surface of the temporal lobe between the hippocampus and the diencephalon, and a choroidal fissure cyst is a loculated cavity filled with CSF lying in the fissure. The relationship of the cyst to the fissure is best visualized on coronal brain imaging; on axial imaging (as in most CT scans), the location of this structure can be misconstrued.²² Almost without exception, choroidal fissure cysts are incidental findings.²² The natural history of choroidal fissure cysts has not been documented in detail, but in general, cysts without mass effect in school-aged children are static and require no follow-up.^{23,24} In infancy, choroidal fissure cysts can exhibit an unstable, progressive course.²⁵ Because the imaging diagnosis can be subtle and because other cystic lesions of the mesial temporal structures can have very different clinical implications, neurosurgical referral is indicated.

LIPOMA OF THE FILUM TERMINALE

Filum terminale lipomas are a type of lumbosacral lipoma in which fat is entirely within the filum terminale and separate from the conus medullaris. Filum lipoma is sometimes detected as a genuinely incidental finding but more often is a finding of uncertain significance that comes to light in the course of an investigation of intractable urinary incontinence, chronic constipation, pes cavus, gait abnormalities, or anomalies of the intergluteal crease. The prevalence of filum lipoma at autopsy has been reported to be 6%.²⁶ Its prevalence among children and adults undergoing MRI for

unrelated reasons is 1.5% to 4%.²⁷⁻³¹ The concern is that filum lipoma may be a cause of or marker for spinal cord tethering.³² Symptoms of tethered cord syndrome include back and lower extremity pain, urologic abnormalities, lower extremity weakness, gait disturbance, and foot and ankle deformities such as pes cavus.³³⁻³⁵ With the increasing use of MRI, filum lipomas have been identified more frequently in asymptomatic individuals.^{28,36,37} The clinical significance of a filum lipoma in an asymptomatic child is a subject of debate.^{27,36,37} Division of a lipomatous filum for relief of tethering (a relatively simple neurosurgical procedure) is undertaken commonly in selected patients with pain, progressive neurologic deficits, scoliosis, or disturbances of bowel and bladder function.^{34,38,39} Unfortunately, symptoms such as back pain and urologic complaints are common in children without neurologic abnormalities, and many of these symptoms resolve with time, medical management, and behavioral therapy.^{40,41} Therefore, the management of children with subjective symptoms of tethered cord syndrome is controversial, especially for those patients without lower than normal position of the spinal cord on imaging. The untreated natural history of asymptomatic filum lipoma is probably benign for most patients.²⁹ For this reason, prophylactic untethering is not indicated for most patients, although some authorities in the past have promoted aggressive prophylactic treatment.³⁸ Neurosurgical evaluation is appropriate, but surgical intervention will seldom prove necessary for incidentally discovered lesions.

PERINEURAL (TARLOV) CYSTS

Perineural cysts arise from the spinal nerve root or dorsal root ganglion. They have a meningeal lining and

contain CSF that is in variable communication with CSF of the thecal sac, and nerve rootlets and ganglion cells lie in the cyst or within the cyst wall. The eponym "Tarlov" is applied to cysts arising in the sacral region, which is by far the most common site.⁴² Among adults, the imaging prevalence of Tarlov cysts has been reported to be between 1.5% and 4.6%.^{31,43-45} The prevalence among children is much lower, consistent with gradual acquisition and expansion of the lesions based on upright posture and hydrostatic CSF pressure. Incidental cysts greatly outnumber symptomatic ones. Clinical assessment must focus on correlating anatomy with symptoms and with the segmental neurologic examination. Perineural cysts are a doubtful explanation for nonspecific low back pain, but they can cause radicular pain and neurologic signs. In the sacral region, perineal pain, bladder symptoms, and sexual dysfunction are relevant. Characteristic features are exacerbation by Valsalva-type events and relief with recumbency. Anatomically consistent radiculopathy indicates neurosurgical referral, and when the nature of the presenting symptoms is obscure, neurosurgical consultation is necessary as well. No imaging surveillance is required in most cases.

INCIDENTAL BRAIN TUMORS

There is no alternative to neurosurgical referral and management of a child with an imaging study interpreted to show an incidental brain tumor. Nevertheless, the primary care physician can address family anxiety and adjust expectations more effectively with some understanding of management issues.

The great majority of incidentally discovered brain tumors in childhood are benign. This fact is readily explained by the overall slight predominance of benign over

malignant tumors in childhood and the much longer time interval between the threshold of imaging detection and production of symptoms for benign tumors.⁴⁶ Examples of benign childhood brain tumors include astrocytoma (World Health Organization grades 1 and 2), ganglioglioma, and dysembryoplastic neuroepithelial tumor.

Reliable statistics describing the prevalence of asymptomatic brain tumors in childhood do not exist. Imaging case series of children with headache are uninformative for a variety of reasons. Such series feature only highly selected patients. Brain tumors reported in these series are seldom explicitly stated to be incidental, and no accounting is made for possible indolent tumors among what are called “white matter lesions” or “parenchymal tissue abnormalities.”⁴⁷⁻⁵¹ The recruitment of normal adults for MRI research is generating a growing volume of literature on the prevalence of incidental findings among genuinely asymptomatic individuals, but there is very little information on normal pediatric research subjects. Kim et al⁵² found 1 likely cerebellar tumor among 225 children (0.4%) enrolled in MRI research. Jordan et al⁵³ found 4 asymptomatic brain tumors among 953 children (0.4%) with sickle cell disease recruited to an imaging study of silent cerebral infarction. Seki et al⁵⁴ noted no brain tumors among the MRI studies in 150 normal Japanese children between 5 and 8 years of age. No brain tumors were recognized among 96 normal children from Malawi studied via MRI by Potchen et al.⁵⁵ Because the neoplastic nature of an isolated incidental finding can often be confirmed only by growth revealed on sequential studies, cross-sectional surveys of normal subjects necessarily produces low estimates. Whatever their true prevalence may be, incidental brain tumors are a regular feature of pediatric

neurosurgery and neurooncology practice.⁵⁶⁻⁵⁸ As expected, benign lesions predominate. In 1 relatively large series, only 3 of 47 tumors were malignant.⁵⁸ Management is individualized on the basis of the appearance and location of the lesion. Lesions recognized to be malignant, such as medulloblastoma, are investigated and treated aggressively. Lesions for which even a small degree of expansion may magnify the difficulty of surgical treatment, such as craniopharyngioma, are also investigated and treated expediently. On the other hand, the neurosurgeon may choose to observe lesions that appear to be benign glial tumors.⁵⁶⁻⁵⁸ Benign glial tumors in childhood have a favorable natural history compared with histologically similar lesions among adults, for whom eventual and lethal malignant degeneration is the rule. Benign childhood gliomas grow slowly, and incidental lesions can be treated surgically for cure before they cause symptoms. Malignant degeneration is rare among children, so withholding of treatment until imaging surveillance reveals growth is often a safe and attractive strategy. The emotional stress of periodic imaging is burdensome for some families, so patient and family preferences must be weighed.

INCIDENTAL VASCULAR LESIONS

Four types of vascular malformations are recognized in the central nervous system: arteriovenous malformation (AVM), cavernous hemangioma, developmental venous malformation (DVM), and telangiectasia. The last of these lesions, telangiectasia, is almost invisible on CT scan. It requires selected MRI sequences, contrast administration, and ideally, high magnet strengths for clear identification.⁵⁹ It is believed to pose no risk of hemorrhage and is not a common incidental finding in childhood.⁶⁰

AVMs vary greatly in size and complexity, but the defining feature is

the direct connection between the arterial and venous systems without an intervening capillary bed. AVMs pose a lifelong risk of hemorrhage at arterial pressures, leading to high rates of neurologic disability and possible mortality. Incidental discovery of a cerebral AVM is an indication for neurosurgical referral, and families may be counseled that a recommendation for proactive treatment may be forthcoming.

Cavernous hemangioma, or “cavernoma,” is a less-threatening lesion composed of thin-walled vessels of varying luminal size without any intercalated normal brain tissue. Constituent vessels that are not thrombotic convey blood at capillary or venous pressures and at very low flow rates. Cavernomas are invisible on catheter angiography, but they have a vivid and distinctive MRI appearance. Multiple lesions are not uncommon, and follow-up may reveal enlargement of existing lesions and de novo appearance of new ones. The classic autopsy prevalence is 0.4%.⁶¹ The imaging prevalence of incidental cavernomas in childhood has been estimated at 0.3%, and prevalence seems to increase with age.⁶² Cavernomas become symptomatic either with seizures or from hemorrhage. They are distinct from AVMs in that hemorrhages are smaller, are less likely to cause disability depending on anatomic location, and are infrequently fatal. The annual risk of hemorrhage is lower as well; a recent report of a pediatric series estimated a 0.2% per lesion per year rate for incidental cavernomas.⁶² Finally, in comparison with AVMs, the surgical management of cavernomas is relatively straightforward. Neurosurgeons are generally reluctant to operate on asymptomatic cavernomas, but exceptions may be made on the basis of such factors as interval enlargement, anatomic location, or family preference. The referring physician may reassure the family that an incidental cavernoma poses

no immediate threat to the life and well-being of the child, but allowance must be made for exercise of the consultant's judgment in treatment recommendations.

What were formerly called "venous angiomas" are now denominated "developmental venous malformations" (DVMs), because they have come to be recognized as nothing more than anomalous patterns of venous drainage of normal brain parenchyma. DVMs are common incidental imaging findings with an autopsy prevalence of 2.5%.⁶¹ They can be seen at any level of the neuraxis, but in the cerebral hemispheres they have a typical morphology: a dominant, radially oriented vein drains superficially or deeply and converging on the origin of this vein are a number of smaller tributaries. This typical morphology, the "caput medusae," recalls the appearance of snakes emanating from the head of Medusa in Greek mythology. DVMs can be seen in association with other vascular malformations, most commonly cavernomas, in which case clinical management is dictated by the character of the associated lesion.⁶³⁻⁶⁵ In isolation, DVMs have no recognized causal relationship with hemorrhage, seizures, or any other clinical phenomena.⁶⁴ Children with incidentally discovered DVMs deserve neurosurgical review, but in the absence of any associated vascular pathology, isolated DVMs do not require treatment or follow-up.^{60,65}

PITUITARY ABNORMALITIES

Unanticipated imaging findings involving the pituitary gland are so common that the term "pituitary incidentaloma" has established itself in the literature.⁶⁶ The autopsy prevalence of pituitary tumors has been estimated at 14%,⁶⁷ but the mix of incidental findings also includes cysts of various kinds, physiologic and pathophysiologic hypertrophy of

the gland, the so-called empty sella syndrome, and morphologic anomalies of the sella itself that can distort the appearance of the gland.⁶⁸⁻⁷¹

As is true of incidentally discovered brain tumors, there is no alternative to neurosurgical referral for incidentally discovered pituitary lesions, but the general pediatrician can set expectations and initiate the investigation. A sensible first step is to obtain a menstrual history, if appropriate, and screening endocrine data: cortisol, thyroxine (T₄), triiodothyronine (T₃), thyroid-stimulating hormone, insulin-like growth factor 1 or somatomedin, prolactin, and, in postmenarchial girls, β -human chorionic gonadotropin. In this context, note must be made of the natural hypertrophy of the pituitary gland associated with pregnancy⁷⁰ and the unnatural but secondary hypertrophy of the pituitary gland associated with primary hypothyroidism.⁷¹ Unlike in adults, the most common secretory tumor of the pituitary gland in childhood is the corticotroph adenoma, followed by prolactinoma and somatotroph adenoma, but management proceeds according to the same principles as in adulthood.⁷² Visual field testing is indicated for expansive lesions large enough to distort the optic chiasm, and as for adults, surgical intervention is usually indicated.

Normal developmental expansion of the pituitary gland in peripubertal girls continues to be an occasion for confusion.⁷³ Normative values for the dimensions of the gland have been analyzed.⁷⁴ The gland may take on a spherical shape, the diaphragm of the sella may assume a convex contour, and the gland may abut the optic chiasm. Uniform signal intensity, prompt homogeneous contrast enhancement, and normal endocrine laboratory data confirm the physiologic nature of these changes. Similar but less pronounced features

may be seen in boys.⁷⁴ Neurosurgical referral may be considered in doubtful cases, but experienced neurosurgeons are very reluctant to call an enlarged pituitary abnormal in this clinical setting.⁷³

Patients with other incidental findings and normal endocrine laboratory values will be referred for neurosurgical consultation as well, but families may be reassured that imaging surveillance will likely be the recommendation. The frequency and duration of imaging surveillance remain a matter for neurosurgical judgment.⁷⁵

ARACHNOID CYSTS

Arachnoid cysts are very common. Most recent large studies have estimated arachnoid cyst prevalence on imaging at ~2%.⁷⁶⁻⁷⁹ The prevalence of arachnoid cysts does not change significantly with advancing age. Boys are nearly twice as likely to harbor arachnoid cysts as girls.^{77,80-83} Most arachnoid cysts are found in the anterior middle fossa and retrocerebellar locations.^{76,77,80-83} Middle fossa arachnoid cysts have a left-sided predominance.^{76,77,79,80,83} Arachnoid cysts may be very large or very small, but size does not correlate precisely with symptoms or the need for treatment.

Arachnoid cysts occasionally present with neurologic signs or symptoms; however, in most cases they are asymptomatic and found incidentally.^{77,84,85} Because arachnoid cysts are common incidental findings, individuals frequently present with both an arachnoid cyst and an unrelated condition or symptom. Clinicians should exercise caution when ascribing any nonspecific symptom, such as headache, behavior disturbance, or epilepsy, to the presence of an arachnoid cyst.^{85,86} Furthermore, such symptoms often persist after surgical treatment of an

arachnoid cyst.^{81,86} Although arachnoid cysts may occasionally enlarge or decrease in size, most do not change substantially over time.^{76,77}

Although most arachnoid cysts should not be treated, there are certain clear exceptions that will benefit from treatment. Cysts should be treated in most cases if they are causing clear and specific neurologic symptoms.⁸⁷⁻⁹⁰ Even small cysts may require treatment if they block normal CSF pathways and cause symptomatic hydrocephalus.

Arachnoid cysts in the suprasellar location are especially likely to cause symptoms and require treatment. As with any surgical procedure, these operations are associated with potential morbidity,^{81,91-93} and the decision to treat surgically should be made very carefully and only after taking the prevalence and natural history of these cysts into account. Most middle fossa and retrocerebellar arachnoid cysts should not be treated. A minority of neurosurgeons continue to make an argument that arachnoid cysts may alter cognition and have used this argument as a justification for surgery in some cases.^{94,95} Evidence of mass effect on imaging is not, by itself, a sufficient indication for surgical treatment of an arachnoid cyst. Any large intracranial cyst can have the appearance of mass effect on imaging. This criterion, therefore, is too inclusive to be used as a reliable indicator for selecting patients for surgical treatment. Because arachnoid cysts are common and the untreated natural history of most arachnoid cysts is benign, most believe that surgical treatment should be avoided except in the unusual instance in which a cyst is clearly responsible for specific symptoms.

Arachnoid cysts may occasionally develop associated subdural hygromas resulting from a spontaneous or traumatic tear in the outer cyst lining.^{86,96,97} These hygromas are rare, and although they

are often symptomatic, they do not always require surgical treatment. Furthermore, surgical treatment of arachnoid cysts can cause iatrogenic hygromas.^{86,87,93} For these reasons, prophylaxis against future hygroma risk should not be regarded as an adequate indication for surgical treatment. Hemorrhage may occasionally occur into an arachnoid cyst after trauma.⁹⁷⁻¹⁰⁰ Because this is also a very rare event, prophylaxis against future hemorrhage risk should not be used to justify surgical treatment in an asymptomatic child. Furthermore, hemorrhages associated with arachnoid cysts are associated with generally good outcomes, even when surgical evacuation of the hemorrhage is deemed necessary.⁹⁷

PINEAL CYSTS

Pineal cysts are also frequently discovered on brain MRIs in children.¹⁰¹⁻¹⁰⁶ Pineal cyst prevalence changes with age, but these cysts are found more frequently in girls than in boys in all age groups.^{101-103,105-109} They are relatively unusual in infants, becoming more common in childhood and adolescence, and peak in young adulthood when they are seen in as many as 2% to 4% of brain MRIs. They become increasingly uncommon with advancing age in adulthood.^{101,103,105,110} Because most pineal cysts are too small to be detected on MRI, the prevalence of microcysts on autopsy studies is even higher.^{111,112} Despite the frequent occurrence of this finding on imaging, the discovery of a pineal cyst often results in a neurosurgical consultation.^{105,107,113}

Pineal cysts have a typical appearance on MRI. Cyst contents may be either isointense or slightly hyperintense to CSF on T1-weighted and hyperintense on T2-weighted imaging.^{104,108,114-116} The cyst rim appears smooth and thin in most cases, but multiple septations within

the cyst itself is a common finding.^{111,112,117} Many benign pineal cysts show evidence of irregular nodular enhancement or ring enhancement on MRI^{118,119} because of surrounding venous structures or the displaced pineal tissue.^{101,103,120,121}

Several groups have reported on the natural history of untreated pineal cysts over time.^{101-103,105,107,120} Although some cysts enlarge and others involute, most cysts remain the same size over several years of follow-up. Importantly, the vast majority of asymptomatic cysts remain asymptomatic. Some growth over time is more often seen in children, and involution is more often seen in adults. Taken together with the cyst prevalence data, the natural history data suggest that pineal cysts frequently arise and change during childhood and then involute during adulthood. For this reason, cyst growth alone in an asymptomatic child should be regarded as the natural course of these cysts and should almost never, by itself, be used as a justification for surgery.¹⁰¹⁻¹⁰³

Pineal cysts are almost always asymptomatic. Rarely, hydrocephalus may result from cerebral aqueductal obstruction due to a large pineal cyst.¹⁰⁶ Some reports have suggested that cysts larger than 1 cm in maximal dimension are more likely to be symptomatic.^{106,112,119,122} Most cysts that cause hydrocephalus are greater than 2 cm in maximal dimension.¹²³ Rarely, large pineal cysts have also been associated with gaze palsy and Parinaud syndrome.^{106,108,118,122,124,125} Although large cysts may be symptomatic, this occurrence is rare. The vast majority of even large cysts may be expected to present incidentally and remain asymptomatic.^{101-103,107,115,120} Given their common incidence on imaging, pineal cysts have a frequent coincidental association with common symptoms such as headache.

A substantial number of children present to their pediatrician with headaches and a pineal cyst that is coincidentally rather than causally associated. Although there are reports of pineal cyst surgery performed for the treatment of chronic headaches,^{119,122,126,127} most surgeons do not regard a history of chronic headache in the absence of hydrocephalus as an indication for surgical treatment of a pineal cyst. Not surprisingly, headaches can be expected to have reliable postoperative symptomatic relief only if the headaches were caused by hydrocephalus.¹²³

Pineal cysts are usually incidentally identified and, once identified, do not result in clinical symptoms in most cases. Specialty consultation and follow-up imaging should be considered merely optional for most asymptomatic children with a conclusive imaging diagnosis of a pineal cyst. In almost every case, cysts smaller than 1 cm will not require neurosurgical evaluation. Larger cysts may also be managed without surgery in many cases but may benefit from an evaluation by a neurosurgeon.

CHIARI AND SYRINGOMYELIA

Chiari malformation type I (CM) is a condition found frequently in children as well as in younger and middle-aged adults. CM is occasionally associated with neurologic symptoms. The classic presentation of CM is headache precipitated by Valsalva-type events, such as sneezing, coughing, or straining. Typically, these headaches are short-lived and located posteriorly. Other symptoms are legion but often nonspecific. These include visual disturbances, dysphonia, dysphagia, sleep apnea, clumsiness and incoordination, and sensory disturbances.^{128,129} CM may also lead to the development of syringomyelia, which, in turn, may lead to other symptoms including

motor and sensory difficulties, pain, and scoliosis.¹³⁰⁻¹³⁴ Surgical case series tend to overestimate the frequency of syringomyelia in patients with CM.¹³⁵⁻¹³⁷ The prevalence of syringomyelia in the setting of CM estimated from imaging databases has been reported to be between 12% and 23%.^{138,139} Syringes are less likely to be found associated with CM in children younger than 5 years but may develop later in childhood.¹³⁹ Lower position of the cerebellar tonsils in CM is associated with a greater likelihood of syringomyelia.¹³⁹⁻¹⁴¹

It is probable that Chiari symptoms and the formation of spinal syringes are the result of crowding at the foramen magnum that leads to abnormal movement of CSF at the craniocervical junction.¹³⁴ Children with CM often have a smaller than normal posterior fossa volume, resulting in crowding of the posterior fossa and foramen magnum contents.¹⁴²⁻¹⁴⁴ This crowding at the foramen magnum may be appreciated on sagittal MRI by the typical “peg-shaped” appearance of the cerebellar tonsils. Because “crowding” is difficult to quantify or objectively determine, the diagnosis of CM on imaging is usually made by a determination of cerebellar tonsil position on MRI. Most often, children are assigned a diagnosis of CM if the cerebellar tonsils are found to be 5 mm or more below the foramen magnum, usually defined as a line between the basion and opisthion in the midsagittal plane.^{78,139,145-148} This definition of CM resulted from the publication of several clinical studies involving small numbers of patients at the dawn of the MRI era.^{145,146} Although cerebellar tonsil measurements that define CM on MRI may be a convenient marker for crowding at the foramen magnum, the correlation is not exact. Patients with less than 5 mm of descent can occasionally present with typical CM symptoms or even syringomyelia attributable to crowding at the

foramen magnum.¹⁴⁹ Conversely, many patients with cerebellar tonsils that are more than 5 mm below the foramen magnum are completely asymptomatic.^{139,150,151} Although CM does present with symptoms in many cases, it is also a very common incidental finding in asymptomatic individuals. Unfortunately, the current clinical tendency appears to be unreservedly admitting such patients to a patho-anatomic group we call CM on the basis of tonsil position alone.

Like most morphometric measurements, cerebellar tonsil position with respect to the foramen magnum follows an essentially normal distribution in all age groups.¹⁵² Those on the lower end of this population distribution fall within the range that may be diagnosed as CM on MRI. The average position of the cerebellar tonsils trends inferiorly during childhood and young adulthood, but the trend reverses with advancing age in adulthood.^{147,152} Females have, on average, a lower tonsil position than males, and CM is more frequently diagnosed in females.^{139,140,151}

If the current imaging definition of tonsils 5 mm below the foramen magnum is used, CM is not rare.^{78,138-140,153,154} CM is found in approximately 0.8% to 1% of all patients undergoing MRI when age is not considered.^{78,138,153} When prevalence is stratified by age, it is clear that the prevalence in children and young adults is greater.¹⁵² As many as 3.6% of children undergoing MRI of the brain or cervical spine have CM by imaging criteria; most children who meet the definition of CM by imaging criteria are asymptomatic.^{139,154}

There are no universally accepted surgical indications for CM. Asymptomatic individuals without a spinal syrinx are only exceptionally considered for surgical treatment. Those with clear symptoms who also have syringomyelia are generally

considered excellent candidates for Chiari decompression surgery.¹⁵⁵ Treatment of those with nonspecific symptoms such as headache (without the usual characteristic features of Chiari-associated headache) is controversial. Because headaches are a common symptom in the general population and CM is a common incidental finding on imaging, surgeons must exercise restraint when selecting these patients for treatment. The natural history of an incidentally discovered Chiari may be expected to follow a benign course in most cases.^{150,151,156} Symptoms and MRI findings are stable over time for most patients, with no symptoms or minimal symptoms that are followed without surgical treatment, although spontaneous improvement or worsening of both the CM as well as syringomyelia does occasionally occur.^{150,151,156}

Although most cases of clinically relevant syringomyelia are associated with CM, it may also be seen in patients with spinal cord tumor, tethered cord, or arachnoiditis.^{157,158} In general, the management of syringomyelia should be directed at the primary disorder. When no primary disorder is identified, the syrinx is considered idiopathic. The untreated natural history of idiopathic syringomyelia is excellent,^{158,159} and the majority of such cases should not be considered for surgical treatment. Small spinal syringes may be difficult to distinguish from minimal dilations of the central canal of the spinal cord. Central spinal cord fluid collections of less than 2 or 3 mm in maximal diameter on axial imaging generally represent only a dilated central canal and are not associated with symptoms.

Children with imaging diagnoses of CM or syringomyelia generally require neurosurgical consultation, but the pediatrician can reassure parents that incidental, asymptomatic

findings are unlikely to require surgical treatment.

CONCLUSIONS

Unexpected findings on imaging studies now account for a large fraction of new patients referred to pediatric neurosurgical practices. They are the cause of a great deal of parental distress, and they put patients at risk of unnecessary additional testing and unnecessary surgery. Rational use of diagnostic technology and subspecialty consultation can minimize these confusing family experiences, but the diagnostic process will always generate an irreducible minimum of unexpected findings that must be accepted and managed for the patient's welfare. Familiarity with the most common entities allows the pediatrician to allay parental anxieties with informed preliminary counseling and to set appropriate priorities for subsequent referrals and investigations. By improving their knowledge base about incidental findings on neuroimaging, pediatricians can provide guidance to families, with neurosurgical consultation when needed regarding clinical relevance, need for additional testing, and need for follow-up.

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ACKNOWLEDGMENT

We thank Ms Holly Wagner for providing editorial assistance.

REFERENCES

1. Smith-Bindman R, Miglioretti DL, Johnson E, et al. Use of diagnostic imaging studies and associated radiation exposure for patients enrolled in large integrated health care systems, 1996–2010. *JAMA*. 2012; 307(22):2400–2409
2. Studdert DM, Mello MM, Sage WM, et al. Defensive medicine among high-risk specialist physicians in a volatile malpractice environment. *JAMA*. 2005; 293(21):2609–2617
3. Bhargavan M, Sunshine JH. Utilization of radiology services in the United States: levels and trends in modalities, regions, and populations. *Radiology*. 2005;234(3):824–832
4. Parker L, Levin DC, Frangos A, Rao VM. Geographic variation in the utilization of noninvasive diagnostic imaging: national medicare data, 1998–2007. *AJR Am J Roentgenol*. 2010;194(4): 1034–1039
5. Organization for Economic Cooperation and Development (OECD). StatExtracts. Published 2013. Available at: http://stats.oecd.org/Index.aspx?DataSetCode=HEALTH_PROC. Accessed August 8, 2014
6. Hellbusch LC. Benign extracerebral fluid collections in infancy: clinical presentation and long-term follow-up. *J Neurosurg*. 2007;107(2 suppl):119–125
7. Kleinman PK, Zito JL, Davidson RI, Raptopoulos V. The subarachnoid spaces in children: normal variations in size. *Radiology*. 1983;147(2):455–457
8. Yew AY, Maher CO, Muraszko KM, Garton HJ. Long-term health status in benign external hydrocephalus. *Pediatr Neurosurg*. 2011;47(1):1–6
9. Muenchberger H, Assaad N, Joy P, Brunsdon R, Shores EA. Idiopathic macrocephaly in the infant: long-term neurological and neuropsychological outcome. *Childs Nerv Syst*. 2006;22(10): 1242–1248
10. Paciorkowski AR, Greenstein RM. When is enlargement of the subarachnoid spaces not benign? A genetic

- perspective. *Pediatr Neurol*. 2007;37(1):1–7
11. Zahl SM, Egge A, Helseth E, Wester K. Benign external hydrocephalus: a review, with emphasis on management. *Neurosurg Rev*. 2011;34(4):417–432
 12. Ravid S, Maytal J. External hydrocephalus: a probable cause for subdural hematoma in infancy. *Pediatr Neurol*. 2003;28(2):139–141
 13. McNeely PD, Atkinson JD, Saigal G, O'Gorman AM, Farmer JP. Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse. *AJNR Am J Neuroradiol*. 2006;27(8):1725–1728
 14. Ghosh PS, Ghosh D. Subdural hematoma in infants without accidental or nonaccidental injury: benign external hydrocephalus, a risk factor. *Clin Pediatr (Phila)*. 2011;50(10):897–903
 15. McKeag H, Christian CW, Rubin D, Daymont C, Pollock AN, Wood J. Subdural hemorrhage in pediatric patients with enlargement of the subarachnoid spaces. *J Neurosurg Pediatr*. 2013;11(4):438–444
 16. Geary M, Patel S, Lamont R. Isolated choroid plexus cysts and association with fetal aneuploidy in an unselected population. *Ultrasound Obstet Gynecol*. 1997;10(3):171–173
 17. Digiovanni LM, Quinlan MP, Verp MS. Choroid plexus cysts: infant and early childhood developmental outcome. *Obstet Gynecol*. 1997;90(2):191–194
 18. Reinsch RC. Choroid plexus cysts—association with trisomy: prospective review of 16,059 patients. *Am J Obstet Gynecol*. 1997;176(6):1381–1383
 19. Perpignano MC, Cohen HL, Klein VR, et al. Fetal choroid plexus cysts: beware the smaller cyst. *Radiology*. 1992;182(3):715–717
 20. Shuangshoti S, Netsky MG. Neuroepithelial (colloid) cysts of the nervous system: further observations on pathogenesis, location, incidence, and histochemistry. *Neurology*. 1966;16(9):887–903
 21. Becker S, Niemann G, Schöning M, Wallwiener D, Mielke G. Clinically significant persistence and enlargement of an antenatally diagnosed isolated choroid plexus cyst. *Ultrasound Obstet Gynecol*. 2002;20(6):620–622
 22. Sherman JL, Camponovo E, Citrin CM. MR imaging of CSF-like choroidal fissure and parenchymal cysts of the brain. *AJR Am J Roentgenol*. 1990;155(5):1069–1075
 23. de Jong L, Thewissen L, van Loon J, Van Calenberg F. Choroidal fissure cerebrospinal fluid-containing cysts: case series, anatomical consideration, and review of the literature. *World Neurosurg*. 2011;75(5–6):704–708
 24. Morioka T, Nishio S, Suzuki S, Fukui M, Nishiyama T. Choroidal fissure cyst in the temporal horn associated with complex partial seizure. *Clin Neurol Neurosurg*. 1994;96(2):164–167
 25. Tubbs RS, Muhleman M, McCluggage SG, et al. Progressive symptomatic increase in the size of choroidal fissure cysts. *J Neurosurg Pediatr*. 2012;10(4):306–309
 26. McLendon RE, Oakes WJ, Heinz ER, Yeates AE, Burger PC. Adipose tissue in the filum terminale: a computed tomographic finding that may indicate tethering of the spinal cord. *Neurosurgery*. 1988;22(5):873–876
 27. Al-Omari MH, Eloqayli HM, Qudseih HM, Al-Shinag MK. Isolated lipoma of filum terminale in adults: MRI findings and clinical correlation. *J Med Imaging Radiat Oncol*. 2011;55(3):286–290
 28. Brown E, Matthes JC, Bazan C III, Jinkins JR. Prevalence of incidental intraspinal lipoma of the lumbosacral spine as determined by MRI. *Spine*. 1994;19(7):833–836
 29. Cools MJ, Al-Holou WN, Stetler WR Jr, et al. Filum terminale lipomas: imaging prevalence, natural history, and conus position. *J Neurosurg Pediatr*. 2014;13(5):559–567
 30. Okumura R, Minami S, Asato R, Konishi J. Fatty filum terminale: assessment with MR imaging. *J Comput Assist Tomogr*. 1990;14(4):571–573
 31. Park HJ, Jeon YH, Rho MH, et al. Incidental findings of the lumbar spine at MRI during herniated intervertebral disk disease evaluation. *AJR Am J Roentgenol*. 2011;196(5):1151–1155
 32. Tani S, Yamada S, Knighton RS. Extensibility of the lumbar and sacral cord. Pathophysiology of the tethered spinal cord in cats. *J Neurosurg*. 1987;66(1):116–123
 33. Metcalfe PD, Luerissen TG, King SJ, et al. Treatment of the occult tethered spinal cord for neuropathic bladder: results of sectioning the filum terminale. *J Urol*. 2006;176(4 pt 2):1826–1829; discussion 1830
 34. Pierre-Kahn A, Zerah M, Renier D, et al. Congenital lumbosacral lipomas. *Childs Nerv Syst*. 1997;13(6):298–334; discussion 335
 35. Yamada S, Won DJ, Pezeshkpour G, et al. Pathophysiology of tethered cord syndrome and similar complex disorders. *Neurosurg Focus*. 2007;23(2):E6
 36. Bulsara KR, Zomorodi AR, Enterline DS, George TM. The value of magnetic resonance imaging in the evaluation of fatty filum terminale. *Neurosurgery*. 2004;54(2):375–379; discussion 379–380
 37. Uchino A, Mori T, Ohno M. Thickened fatty filum terminale: MR imaging. *Neuroradiology*. 1991;33(4):331–333
 38. La Marca F, Grant JA, Tomita T, McLone DG. Spinal lipomas in children: outcome of 270 procedures. *Pediatr Neurosurg*. 1997;26(1):8–16
 39. Xenos C, Sgouros S, Walsh R, Hockley A. Spinal lipomas in children. *Pediatr Neurosurg*. 2000;32(6):295–307
 40. Drake JM. Occult tethered cord syndrome: not an indication for surgery. *J Neurosurg*. 2006;104(5 suppl):305–308
 41. Drake JM. Surgical management of the tethered spinal cord—walking the fine line. *Neurosurg Focus*. 2007;23(2):E4
 42. Tarlov IM. Cysts, perineurial, of the sacral roots; another cause, removable, of sciatic pain. *J Am Med Assoc*. 1948;138(10):740–744
 43. Joo J, Kim J, Lee J. The prevalence of anatomical variations that can cause inadvertent dural puncture when performing caudal block in Koreans: a study using magnetic resonance imaging. *Anaesthesia*. 2010;65(1):23–26
 44. Langdown AJ, Grundy JR, Birch NC. The clinical relevance of Tarlov cysts. *J Spinal Disord Tech*. 2005;18(1):29–33
 45. Paulsen RD, Call GA, Murtagh FR. Prevalence and percutaneous drainage of cysts of the sacral nerve root sheath

- (Tarlov cysts). *AJNR Am J Neuroradiol*. 1994;15(2):293–297; discussion 298–299
46. Kohler BA, Ward E, McCarthy BJ, et al. Annual report to the nation on the status of cancer, 1975–2007, featuring tumors of the brain and other nervous system. *J Natl Cancer Inst*. 2011;103(9):714–736
 47. Schwedt TJ, Guo Y, Rothner AD. “Benign” imaging abnormalities in children and adolescents with headache. *Headache*. 2006;46(3):387–398
 48. Becker LA, Green LA, Beaufait D, Kirk J, Froom J, Freeman WL. Use of CT scans for the investigation of headache: a report from ASPN, part 1. *J Fam Pract*. 1993;37(2):129–134
 49. Graf WD, Kayyali HR, Abdelmoity AT, Womelduff GL, Williams AR, Morriss MC. Incidental neuroimaging findings in nonacute headache. *J Child Neurol*. 2010;25(10):1182–1187
 50. Gupta S, Kanamalla U, Gupta V. Are incidental findings on brain magnetic resonance images in children merely incidental? *J Child Neurol*. 2010;25(12):1511–1516
 51. Streibert PF, Piroth W, Mansour M, Haage P, Langer T, Borusiak P. Magnetic resonance imaging of the brain in children with headache: the clinical relevance with modern acquisition techniques. *Clin Pediatr (Phila)*. 2011; 50(12):1134–1139
 52. Kim BS, Illes J, Kaplan RT, Reiss A, Atlas SW. Incidental findings on pediatric MR images of the brain. *AJNR Am J Neuroradiol*. 2002;23(10):1674–1677
 53. Jordan LC, McKinstry RC III, Kraut MA, et al; Silent Infarct Transfusion Trial Investigators. Incidental findings on brain magnetic resonance imaging of children with sickle cell disease. *Pediatrics*. 2010;126(1):53–61
 54. Seki A, Uchiyama H, Fukushi T, Sakura O, Tatsuya K; Japan Children’s Study Group. Incidental findings of brain magnetic resonance imaging study in a pediatric cohort in Japan and recommendation for a model management protocol. *J Epidemiol*. 2010;20(suppl 2):S498–S504
 55. Potchen MJ, Kampondeni SD, Mallewa M, Taylor TE, Birbeck GL. Brain imaging in normal kids: a community-based MRI study in Malawian children. *Trop Med Int Health*. 2013;18(4):398–402
 56. Bredlau AL, Constine LS, Silberstein HJ, Milano MT, Korones DN. Incidental brain lesions in children: to treat or not to treat? *J Neurooncol*. 2012;106(3): 589–594
 57. Perret C, Boltshauser E, Scheer I, Kellenberger CJ, Grotzer MA. Incidental findings of mass lesions on neuroimages in children. *Neurosurg Focus*. 2011;31(6):E20
 58. Roth J, Keating RF, Myseros JS, Yaun AL, Magge SN, Constantini S. Pediatric incidental brain tumors: a growing treatment dilemma. *J Neurosurg Pediatr*. 2012;10(3):168–174
 59. El-Koussy M, Schroth G, Gralla J, et al. Susceptibility-weighted MR imaging for diagnosis of capillary telangiectasia of the brain. *AJNR Am J Neuroradiol*. 2012; 33(4):715–720
 60. Chalouhi N, Dumont AS, Randazzo C, et al. Management of incidentally discovered intracranial vascular abnormalities. *Neurosurg Focus*. 2011; 31(6):E1
 61. Sarwar M, McCormick WF. Intracerebral venous angioma: case report and review. *Arch Neurol*. 1978;35(5):323–325
 62. Al-Holou WN, O’Lynnner TM, Pandey AS, et al. Natural history and imaging prevalence of cavernous malformations in children and young adults. *J Neurosurg Pediatr*. 2012;9(2):198–205
 63. Ostertun B, Solymosi L. Magnetic resonance angiography of cerebral developmental venous anomalies: its role in differential diagnosis. *Neuroradiology*. 1993;35(2):97–104
 64. Töpper R, Jürgens E, Reul J, Thron A. Clinical significance of intracranial developmental venous anomalies. *J Neurol Neurosurg Psychiatry*. 1999; 67(2):234–238
 65. Ruiz DS, Yilmaz H, Gailloud P. Cerebral developmental venous anomalies: current concepts. *Ann Neurol*. 2009; 66(3):271–283
 66. Reincke M, Allolio B, Saeger W, Menzel J, Winkelmann W. The ‘incidentaloma’ of the pituitary gland: is neurosurgery required? *JAMA*. 1990;263(20): 2772–2776
 67. Ezzat S, Asa SL, Couldwell WT, et al. The prevalence of pituitary adenomas: a systematic review. *Cancer*. 2004; 101(3):613–619
 68. Bruneton JN, Drouillard JP, Sabatier JC, Elie GP, Tavernier JF. Normal variants of the sella turcica. *Radiology*. 1979;131(1): 99–104
 69. Chanson P, Daujat F, Young J, et al. Normal pituitary hypertrophy as a frequent cause of pituitary incidentaloma: a follow-up study. *J Clin Endocrinol Metab*. 2001;86(7): 3009–3015
 70. Scheithauer BW, Sano T, Kovacs KT, Young WF Jr, Ryan N, Randall RV. The pituitary gland in pregnancy: a clinicopathologic and immunohistochemical study of 69 cases. *Mayo Clin Proc*. 1990;65(4): 461–474
 71. Yamada T, Tsukui T, Ikejiri K, Yukimura Y, Kotani M. Volume of sella turcica in normal subjects and in patients with primary hypothyroidism and hyperthyroidism. *J Clin Endocrinol Metab*. 1976;42(5):817–822
 72. Partington MD, Davis DH, Laws ER Jr, Scheithauer BW. Pituitary adenomas in childhood and adolescence. Results of transsphenoidal surgery. *J Neurosurg*. 1994;80(2):209–216
 73. Aquilina K, Boop FA. Nonneoplastic enlargement of the pituitary gland in children. *J Neurosurg Pediatr*. 2011; 7(5):510–515
 74. Elster AD, Chen MY, Williams DW III, Key LL. Pituitary gland: MR imaging of physiologic hypertrophy in adolescence. *Radiology*. 1990; 174(3 pt 1):681–685
 75. Freda PU, Beckers AM, Katznelson L, et al; Endocrine Society. Pituitary incidentaloma: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;96(4):894–904
 76. Al-Holou WN, Terman S, Kilburg C, Garton HJ, Muraszko KM, Maher CO. Prevalence and natural history of arachnoid cysts in adults. *J Neurosurg*. 2013;118(2):222–231
 77. Al-Holou WN, Yew AY, Boomsaad ZE, Garton HJ, Muraszko KM, Maher CO. Prevalence and natural history of arachnoid cysts in children. *J Neurosurg Pediatr*. 2010;5(6):578–585
 78. Vernooij MW, Ikram MA, Tanghe HL, et al. Incidental findings on brain MRI in the general population. *N Engl J Med*. 2007;357(18):1821–1828

79. Weber F, Knopf H. Incidental findings in magnetic resonance imaging of the brains of healthy young men. *J Neurol Sci*. 2006;240(1-2):81-84
80. Helland CA, Lund-Johansen M, Wester K. Location, sidedness, and sex distribution of intracranial arachnoid cysts in a population-based sample. *J Neurosurg*. 2010;113(5):934-939
81. Levy ML, Wang M, Aryan HE, Yoo K, Meltzer H. Microsurgical keyhole approach for middle fossa arachnoid cyst fenestration. *Neurosurgery*. 2003;53(5):1138-1144; discussion 1144-1145
82. Oberbauer RW, Haase J, Pucher R. Arachnoid cysts in children: a European co-operative study. *Childs Nerv Syst*. 1992;8(5):281-286
83. Wester K. Peculiarities of intracranial arachnoid cysts: location, sidedness, and sex distribution in 126 consecutive patients. *Neurosurgery*. 1999;45(4):775-779
84. Pradilla G, Jallo G. Arachnoid cysts: case series and review of the literature. *Neurosurg Focus*. 2007;22(2):E7
85. Tamburrini G, Dal Fabbro M, Di Rocco C. Sylvian fissure arachnoid cysts: a survey on their diagnostic workup and practical management. *Childs Nerv Syst*. 2008;24(5):593-604
86. Di Rocco C. Sylvian fissure arachnoid cysts: we do operate on them but should it be done? *Childs Nerv Syst*. 2010;26(2):173-175
87. Fewel ME, Levy ML, McComb JG. Surgical treatment of 95 children with 102 intracranial arachnoid cysts. *Pediatr Neurosurg*. 1996;25(4):165-173
88. Gangemi M, Seneca V, Colella G, Cioffi V, Imperato A, Maiuri F. Endoscopy versus microsurgical cyst excision and shunting for treating intracranial arachnoid cysts. *J Neurosurg Pediatr*. 2011;8(2):158-164
89. Kang JK, Lee KS, Lee IW, et al. Shunt-independent surgical treatment of middle cranial fossa arachnoid cysts in children. *Childs Nerv Syst*. 2000;16(2):111-116
90. Maher CO, Goumnerova L. The effectiveness of ventriculocystocisternostomy for suprasellar arachnoid cysts. *J Neurosurg Pediatr*. 2011;7(1):64-72
91. Shim KW, Lee YH, Park EK, Park YS, Choi JU, Kim DS. Treatment option for arachnoid cysts. *Childs Nerv Syst*. 2009;25(11):1459-1466
92. Spacca B, Kandasamy J, Mallucci CL, Genitori L. Endoscopic treatment of middle fossa arachnoid cysts: a series of 40 patients treated endoscopically in two centres. *Childs Nerv Syst*. 2010;26(2):163-172
93. Tamburrini G, Caldarelli M, Massimi L, Santini P, Di Rocco C. Subdural hygroma: an unwanted result of Sylvian arachnoid cyst marsupialization. *Childs Nerv Syst*. 2003;19(3):159-165
94. Torgersen J, Helland C, Flaatten H, Wester K. Reversible dyscognition in patients with a unilateral, middle fossa arachnoid cyst revealed by using a laptop based neuropsychological test battery (CANTAB). *J Neurol*. 2010;257(11):1909-1916
95. Wester K. Intracranial arachnoid cysts—do they impair mental functions? *J Neurol*. 2008;255(8):1113-1120
96. Albuquerque FC, Giannotta SL. Arachnoid cyst rupture producing subdural hygroma and intracranial hypertension: case reports. *Neurosurgery*. 1997;41(4):951-955; discussion 955-956
97. Parsch CS, Krauss J, Hofmann E, Meixensberger J, Roosen K. Arachnoid cysts associated with subdural hematomas and hygromas: analysis of 16 cases, long-term follow-up, and review of the literature. *Neurosurgery*. 1997;40(3):483-490
98. Bilginer B, Onal MB, Oğuz KK, Akalan N. Arachnoid cyst associated with subdural hematoma: report of three cases and review of the literature. *Childs Nerv Syst*. 2009;25(1):119-124
99. Domenicucci M, Russo N, Giugni E, Pierallini A. Relationship between supratentorial arachnoid cyst and chronic subdural hematoma: neuroradiological evidence and surgical treatment. *J Neurosurg*. 2009;110(6):1250-1255
100. Mori K, Yamamoto T, Horinaka N, Maeda M. Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles: twelve cases of chronic subdural hematoma associated with arachnoid cyst. *J Neurotrauma*. 2002;19(9):1017-1027
101. Al-Holou WN, Garton HJ, Muraszko KM, Ibrahim M, Maher CO. Prevalence of pineal cysts in children and young adults. *J Neurosurg Pediatr*. 2009;4(3):230-236
102. Al-Holou WN, Maher CO, Muraszko KM, Garton HJ. The natural history of pineal cysts in children and young adults. *J Neurosurg Pediatr*. 2010;5(2):162-166
103. Al-Holou WN, Terman SW, Kilburg C, et al. Prevalence and natural history of pineal cysts in adults. *J Neurosurg*. 2011;115(6):1106-1114
104. Di Costanzo A, Tedeschi G, Di Salle F, Golia F, Morrone R, Bonavita V. Pineal cysts: an incidental MRI finding? *J Neurol Neurosurg Psychiatry*. 1993;56(2):207-208
105. Sawamura Y, Ikeda J, Ozawa M, Minoshima Y, Saito H, Abe H. Magnetic resonance images reveal a high incidence of asymptomatic pineal cysts in young women. *Neurosurgery*. 1995;37(1):11-15; discussion 15-16
106. Wisoff JH, Epstein F. Surgical management of symptomatic pineal cysts. *J Neurosurg*. 1992;77(6):896-900
107. Barboriak DP, Lee L, Provenzale JM. Serial MR imaging of pineal cysts: implications for natural history and follow-up. *AJR Am J Roentgenol*. 2001;176(3):737-743
108. Manderla M, Marcol W, Bierzyńska-Macyszyn G, Kluczevska E. Pineal cysts in childhood. *Childs Nerv Syst*. 2003;19(10-11):750-755
109. Pu Y, Mahankali S, Hou J, et al. High prevalence of pineal cysts in healthy adults demonstrated by high-resolution, noncontrast brain MR imaging. *AJNR Am J Neuroradiol*. 2007;28(9):1706-1709
110. Sener RN. The pineal gland: a comparative MR imaging study in children and adults with respect to normal anatomical variations and pineal cysts. *Pediatr Radiol*. 1995;25(4):245-248
111. Hasegawa A, Ohtsubo K, Mori W. Pineal gland in old age; quantitative and qualitative morphological study of 168 human autopsy cases. *Brain Res*. 1987;409(2):343-349
112. Tapp E, Huxley M. The histological appearance of the human pineal gland from puberty to old age. *J Pathol*. 1972;108(2):137-144

113. Piatt JH Jr. Unexpected findings on brain and spine imaging in children. *Pediatr Clin North Am*. 2004;51(2):507–527
114. Fakhran S, Escott EJ. Pineocytoma mimicking a pineal cyst on imaging: true diagnostic dilemma or a case of incomplete imaging? *AJNR Am J Neuroradiol*. 2008;29(1):159–163
115. Mamourian AC, Towfighi J. Pineal cysts: MR imaging. *AJNR Am J Neuroradiol*. 1986;7(6):1081–1086
116. Welton PL, Reicher MA, Kellerhouse LE, Ott KH. MR of benign pineal cyst. *AJNR Am J Neuroradiol*. 1988;9(3):612
117. Carr JL. Cystic hydrops of the pineal gland with a report of six cases. *J Nerv Ment Dis*. 1944;99(5):552–572
118. Fain JS, Tomlinson FH, Scheithauer BW, et al. Symptomatic glial cysts of the pineal gland. *J Neurosurg*. 1994;80(3):454–460
119. Fleege MA, Miller GM, Fletcher GP, Fain JS, Scheithauer BW. Benign glial cysts of the pineal gland: unusual imaging characteristics with histologic correlation. *AJNR Am J Neuroradiol*. 1994;15(1):161–166
120. Golzarian J, Balériaux D, Bank WO, Matos C, Flament-Durand J. Pineal cyst: normal or pathological? *Neuroradiology*. 1993;35(4):251–253
121. Korogi Y, Takahashi M, Ushio Y. MRI of pineal region tumors. *J Neurooncol*. 2001;54(3):251–261
122. Klein P, Rubinstein LJ. Benign symptomatic glial cysts of the pineal gland: a report of seven cases and review of the literature. *J Neurol Neurosurg Psychiatry*. 1989;52(8):991–995
123. Fetell MR, Bruce JN, Burke AM, et al. Non-neoplastic pineal cysts. *Neurology*. 1991;41(7):1034–1040
124. Mena H, Armonda RA, Ribas JL, Ondra SL, Rushing EJ. Nonneoplastic pineal cysts: a clinicopathologic study of twenty-one cases. *Ann Diagn Pathol*. 1997;1(1):11–18
125. Michielsen G, Benoit Y, Baert E, Meire F, Caemaert J. Symptomatic pineal cysts: clinical manifestations and management. *Acta Neurochir (Wien)*. 2002;144(3):233–242; discussion 242
126. Gore PA, Gonzalez LF, Rekatte HL, Nakaji P. Endoscopic supracerebellar infratentorial approach for pineal cyst resection: technical case report. *Neurosurgery*. 2008;62(3 suppl 1):108–109; discussion 109
127. Stevens QE, Colen CB, Ham SD, Kattner KA, Sood S. Delayed lateral rectus palsy following resection of a pineal cyst in sitting position: direct or indirect compressive phenomenon? *J Child Neurol*. 2007;22(12):1411–1414
128. George TM, Higginbotham NH. Defining the signs and symptoms of Chiari malformation type I with and without syringomyelia. *Neurol Res*. 2011;33(3):240–246
129. Tubbs RS, Lyster MJ, Loukas M, Shoja MM, Oakes WJ. The pediatric Chiari I malformation: a review. *Childs Nerv Syst*. 2007;23(11):1239–1250
130. Bogdanov EI, Mendelevich EG. Syrinx size and duration of symptoms predict the pace of progressive myelopathy: retrospective analysis of 103 unoperated cases with craniocervical junction malformations and syringomyelia. *Clin Neurol Neurosurg*. 2002;104(2):90–97
131. Eule JM, Erickson MA, O'Brien MF, Handler M. Chiari I malformation associated with syringomyelia and scoliosis: a twenty-year review of surgical and nonsurgical treatment in a pediatric population. *Spine*. 2002;27(13):1451–1455
132. Lipson AC, Ellenbogen RG, Avellino AM. Radiographic formation and progression of cervical syringomyelia in a child with untreated Chiari I malformation. *Pediatr Neurosurg*. 2008;44(3):221–223
133. Nishizawa S, Yokoyama T, Yokota N, Tokuyama T, Ohta S. Incidentally identified syringomyelia associated with Chiari I malformations: is early interventional surgery necessary? *Neurosurgery*. 2001;49(3):637–640; discussion 640–641
134. Oldfield EH, Muraszko K, Shawker TH, Patronas NJ. Pathophysiology of syringomyelia associated with Chiari I malformation of the cerebellar tonsils: implications for diagnosis and treatment. *J Neurosurg*. 1994;80(1):3–15
135. Haines SJ, Berger M. Current treatment of Chiari malformations types I and II: a survey of the Pediatric Section of the American Association of Neurological Surgeons. *Neurosurgery*. 1991;28(3):353–357
136. Rocque BG, George TM, Kestle J, Iskandar BJ. Treatment practices for Chiari malformation type I with syringomyelia: results of a survey of the American Society of Pediatric Neurosurgeons. *J Neurosurg Pediatr*. 2011;8(5):430–437
137. Schijman E, Steinbok P. International survey on the management of Chiari I malformation and syringomyelia. *Childs Nerv Syst*. 2004;20(5):341–348
138. Aitken LA, Lindan CE, Sidney S, et al. Chiari type I malformation in a pediatric population. *Pediatr Neurol*. 2009;40(6):449–454
139. Strahle J, Muraszko KM, Kapurch J, Bapuraj JR, Garton HJ, Maher CO. Chiari malformation type I and syrinx in children undergoing magnetic resonance imaging. *J Neurosurg Pediatr*. 2011;8(2):205–213
140. Elster AD, Chen MY. Chiari I malformations: clinical and radiologic reappraisal. *Radiology*. 1992;183(2):347–353
141. Pillay PK, Awad IA, Little JR, Hahn JF. Symptomatic Chiari malformation in adults: a new classification based on magnetic resonance imaging with clinical and prognostic significance. *Neurosurgery*. 1991;28(5):639–645
142. Badie B, Mendoza D, Batzdorf U. Posterior fossa volume and response to suboccipital decompression in patients with Chiari I malformation. *Neurosurgery*. 1995;37(2):214–218
143. Noudel R, Jovenin N, Eap C, Scherpereel B, Pierot L, Rousseaux P. Incidence of basioccipital hypoplasia in Chiari malformation type I: comparative morphometric study of the posterior cranial fossa. *J Neurosurg*. 2009;111(5):1046–1052
144. Sgouros S, Kountouri M, Natarajan K. Skull base growth in children with Chiari malformation Type I. *J Neurosurg*. 2007;107(3 suppl):188–192
145. Abouelezz AO, Sartor K, Geyer CA, Gado MH. Position of cerebellar tonsils in the normal population and in patients with Chiari malformation: a quantitative approach with MR imaging. *J Comput Assist Tomogr*. 1985;9(6):1033–1036
146. Barkovich AJ, Wippold FJ, Sherman JL, Citrin CM. Significance of cerebellar tonsillar position on MR. *AJNR Am J Neuroradiol*. 1986;7(5):795–799

147. Mikulis DJ, Diaz O, Egglin TK, Sanchez R. Variance of the position of the cerebellar tonsils with age: preliminary report. *Radiology*. 1992;183(3):725–728
148. Rekate HL. Natural history of the Chiari type I anomaly. *J Neurosurg Pediatr*. 2008;2(3):177–178; discussion 178
149. Markunas CA, Tubbs RS, Moftakhar R, et al. Clinical, radiological, and genetic similarities between patients with Chiari type I and type 0 malformations. *J Neurosurg Pediatr*. 2012;9(4):372–378
150. Benglis D Jr, Covington D, Bhatia R, et al. Outcomes in pediatric patients with Chiari malformation type I followed up without surgery. *J Neurosurg Pediatr*. 2011;7(4):375–379
151. Novegno F, Caldarelli M, Massa A, et al. The natural history of the Chiari type I anomaly. *J Neurosurg Pediatr*. 2008; 2(3):179–187
152. Smith BW, Strahle J, Bapuraj JR, Muraszko KM, Garton HJ, Maher CO. Distribution of cerebellar tonsil position: implications for understanding Chiari malformation. *J Neurosurg*. 2013;119(3):812–819
153. Meadows J, Kraut M, Guarnieri M, Haroun RI, Carson BS. Asymptomatic Chiari type I malformations identified on magnetic resonance imaging. *J Neurosurg*. 2000;92(6):920–926
154. Wu YW, Chin CT, Chan KM, Barkovich AJ, Ferriero DM. Pediatric Chiari I malformations: do clinical and radiologic features correlate? *Neurology*. 1999;53(6):1271–1276
155. Tubbs RS, Beckman J, Naftel RP, et al. Institutional experience with 500 cases of surgically treated pediatric Chiari malformation type I. *J Neurosurg Pediatr*. 2011;7(3):248–256
156. Strahle J, Muraszko KM, Kapurch J, Bapuraj JR, Garton HJ, Maher CO. Natural history of Chiari malformation type I following decision for conservative treatment. *J Neurosurg Pediatr*. 2011;8(2):214–221
157. Roy AK, Slimack NP, Ganju A. Idiopathic syringomyelia: retrospective case series, comprehensive review, and update on management. *Neurosurg Focus*. 2011;31(6):E15
158. Singhal A, Bowen-Roberts T, Steinbok P, Cochrane D, Byrne AT, Kerr JM. Natural history of untreated syringomyelia in pediatric patients. *Neurosurg Focus*. 2011;31(6):E13
159. Magge SN, Smyth MD, Governale LS, et al. Idiopathic syrinx in the pediatric population: a combined center experience. *J Neurosurg Pediatr*. 2011; 7(1):30–36

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