REVIEW ARTICLE

Incidental findings on brain magnetic resonance imaging (MRI) in adults: a review of imaging spectrum, clinical significance, and management

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ABSTRACT:

Utilization of brain MRI has dramatically increased in recent decades due to rapid advancement in imaging technology and improving accessibility. As a result, radiologists increasingly encounter findings incidentally discovered on brain MRIs which are performed for unrelated indications. Some of these findings are clinically significant, necessitating further investigation or treatment and resulting in increased costs to healthcare systems as well as increased patient anxiety. Moreover, management of these incidental findings poses a significant challenge for referring physicians. Therefore, it is important for interpreting radiologists to know the prevalence, clinical consequences, and appropriate management of these findings. There is a wide spectrum of incidental findings on brain MRI such as asymptomatic brain infarct, age-related white matter changes, microhemorrhages, intracranial tumors, intracranial cystic lesions, and anatomic variants. This article provides a narrative review of important incidental findings encountered on brain MRI in adults with a focus on prevalence, clinical implications, and recommendations on management of these findings based on current available data.

INTRODUCTION

Utilization of brain MRI has dramatically increased in past decades due to rapid advancement in imaging technology and improving accessibility. As a result, radiologists increasingly encounter findings incidentally discovered on brain MRIs which are performed for unrelated indications.[1–6] Some of these findings are clinically significant, necessitating further investigation or treatment and resulting in increasing healthcare costs and patient anxiety.[7,8] Moreover, management of these incidental findings poses a significant challenge for referring physicians because there are still no standard guidelines for management of many asymptomatic incidental findings partly due to lack of randomized controlled trials.[2,9] Some researchers have classified incidental findings based on the level of clinical urgency of referral for additional work-up. However, this urgency varies depending on individual practice and perceptions and patient characteristics (*e.g.* age, health condition, comorbidities), further complicating management.[2] Therefore, it is important for interpreting radiologists to know the prevalence, clinical consequences, and appropriate management of incidental findings.

The prevalence of incidental findings on brain MRI in the healthy adult population ranges from 9 to 54%.[1–6,10,11] Variability of the prevalence of incidental findings likely depends on several factors, including target population clinical and demographic characteristics (*i.e.* age, health status), inclusion criteria, definition of incidental findings, and imaging techniques.[2,3,5,10] Some incidental findings such as asymptomatic brain infarcts, white matter volume loss, and neoplasms are age-related and seen more frequently in older populations.[2,3] The prevalence of incidental findings is also higher when utilizing high-resolution MRI sequences, compared to conventional MRI techniques.[2] Previously reported incidental brain MRI findings are summarized in Table 1.

This article reviews important incidental adult brain MRI findings, with a focus on prevalence, clinical implication,

Table 1. Incidental findings on brain MRI

Incidental findings on brain MRI
<u>Non-neoplastic brain parenchymal lesions</u>
• Changes in relation to cerebrovascular disease, *e.g.* white matter hyperintensities, volume loss, asymptomatic brain infarcts, hemorrhages
• Other acquired parenchymal lesions, *e.g.* demyelination, post-traumatic injury, neurodegenerative diseases
<u>Intracranial tumors</u>
• - Intra-axial tumors, *e.g.* primary brain tumors (glioma, MVNT, ganglioglioma, DNET), brain metastases
• - Extra-axial tumors, *e.g.* meningioma, schwannoma
• - Intraventricular tumors, *e.g.* subependymoma, central neurocytoma, meningioma
• - Pituitary incidentalomas
• - Pineal tumors, *e.g.* pineocytoma
<u>Intracranial cystic lesions</u>
• - Rathke's cleft cyst
• - Pineal cyst
• - Arachnoid cyst
• - Colloid cyst
• - Epidermoid cyst
• - Neuroenteric cyst
<u>Anatomical variants/Developmental structural abnormalities</u>
• - Cavum septum pellucidum, cavum et vergae, velum interpositum
• - Ventricular asymmetry
• - Chiari I malformation
• - Gray matter heterotopia
• - Mega cisterna magna
<u>Other abnormalities</u>
• - Intracranial lipoma
• - Hydrocephalus
• - Extra-axial fluid collection/hematoma
• - Empty sella

DNET, dysembryoplastic neuroepithelial tumor; MVNT, multinodular and vacuolating neuronal tumor.

and appropriate management. Incidental intracranial vascular findings such as aneurysm or arteriovenous malformation are covered in a separate article in this issue.

## CEREBROVASCULAR ISCHEMIC CHANGES AND MICROHEMORRHAGES

Brain parenchymal changes secondary to cerebrovascular disease [such as asymptomatic or silent brain infarct (SBI), age-related white matter changes, and microhemorrhages] are common incidental findings on brain MRI, and frequently seen in the elderly.[3,5] A recent systematic review shows that the overall prevalence of SBI in individuals between 62 and 76 years old is approximately 18%[12] . The vast majority of SBIs are lacunar infarcts (3–20 mm diameter). Although patients with SBI do not have overt stroke symptoms, the presence of SBI should be considered as a marker of cerebrovascular disease rather than an incidental finding. SBI is associated with twofold higher risk of subsequent stroke and is an independent factor associated with future cognitive dysfunction.[12–14]

Age-related white matter changes (WMCs) are very common incidental findings on brain MRI and can be seen in more than half of elderly individuals.[15,16] The lesions can be classified into two types based on the location, including (1) periventricular and (2) deep/subcortical white matter.[17] Periventricular WMCs are non-vascular in origin and commonly caused by disruption of the ependymal lining and subependymal gliosis. Deep and subcortical WMCs are further divided into three subtypes based on lesion extent, including punctate, early confluent, and confluent WMCs (Figure 1).[17,18] Punctate WMCs are commonly non-ischemic in origin, caused by widening of periarteriolar spaces and focal loss of myelination. Early confluent and confluent WMCs represent a continuum of progressive ischemic lesions. Punctate WMCs typically show no or minimal progression while early confluent and confluent WMCs demonstrate a more rapid increase in lesion volume on follow-up imaging.[16,17] There is also a strong correlation between confluent WMCs and impaired cognition and functional decline.[17,19]

Incidental brain microhemorrhages are seen in 4–6% of otherwise healthy adults.[20,21] The prevalence of brain microhemorrhages ranges up to 31–36% in ischemic stroke patients and 54–64% in those with prior intracerebral hemorrhage.[20,22] Microhemorrhages are associated with cardiovascular risk factors such as hypertension and diabetes, and increased risk of both ischemic and hemorrhagic strokes in the general population.[20,23] As such, brain microhemorrhages are an important marker of underlying cerebrovascular disease and should be interpreted along with other neuroimaging markers and clinical factors. Patients with

Figure 1. Age-related white matter changes. Axial FLAIR images of three different patients who underwent brain MRI for unrelated indication. There are three patterns of deep/subcortical white matter changes based on extent of the lesions, including (A) punctate, (B) early confluent, and (C) confluent white matter changes. Punctate changes are typically of non-ischemic origin, whereas early confluent and confluent changes represent progressive ischemic lesions. FLAIR, fluid attenuated inversion recovery.
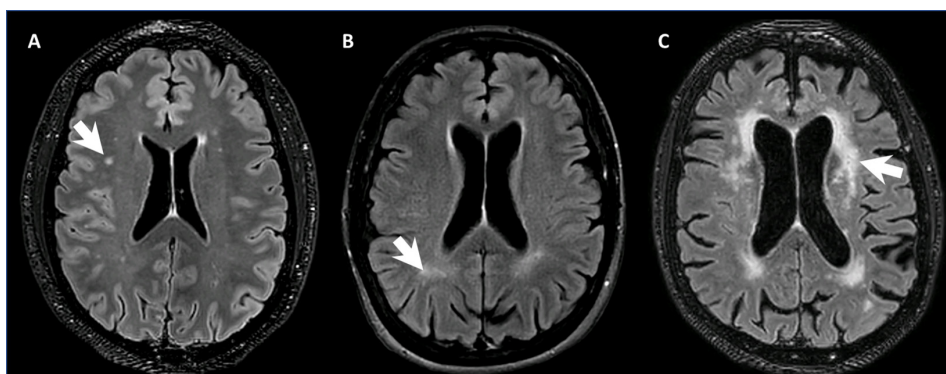
brain parenchymal changes related to cerebrovascular disease should receive clinical assessment for the potential risks of stroke which may warrant medical preventive measures.[22,24]

INTRACRANIAL TUMORS

The prevalence of incidental intracranial tumors detected on brain MRI is approximately 0.5–2.5%, with meningioma being the most common neoplasm.[1–3,5,10,25] Previous large meta-analyses found an increasing prevalence with age for incidental intracranial tumors, potentially driven by the age-related increased prevalence of meningiomas.[2,3] In this section, we discuss incidental intracranial tumors categorized into intra-axial, extra-axial, intraventricular, pineal and pituitary tumors.

Intra-axial tumors
Incidental glioma
Incidental gliomas are relatively rare, with an incidence of 0.05–0.2% in the general population.[25,26] Most incidental gliomas are low-grade and isocitrate dehydrogenase (IDH) mutated[26]. Patients become symptomatic at a median time of 48 months after initial discovery, typically presenting with new onset seizure or other neurologic deficits such as motor weakness. The

average tumor growth rate of incidental gliomas is approximately 3.5 mm per year[27]. Malignant transformation of low-grade gliomas is reported to occur at a median interval of between 2 and 10 years[26]. On MRI, low-grade gliomas manifest as ill-defined T1 hypointense and T2 hyperintense lesions, with little to no enhancement (Figure 2).[26] New areas of enhancement on follow-up imaging generally reflect anaplastic changes.[28] Advanced MRI techniques are important complimentary imaging tools for initial tumor assessment, treatment planning, and post-treatment evaluation.[29] MR perfusion and MR spectroscopy can help with tumor grading, target selection for biopsy, and differentiation between tumor progression and pseudoprogression.[29] Diffusion tensor imaging can be used to assess integrity of white matter tracts and provides better tumor delineation compared with conventional MRI.[26,29] The current management of low-grade gliomas points toward an early maximal tumor resection.[26,30] In general, incidental gliomas tend to be significantly smaller than symptomatic low-grade gliomas and are usually located in non-eloquent areas which makes them more amenable to gross total resection.[26,31]

Figure 2. Incidental low-grade glioma A 39-year-old female undergoing brain MRI for intermittent headaches. Patient denied any focal neurological deficits. Axial FLAIR (A) and axial contrast-enhanced T_1WI (B) demonstrate a small ill-defined non-enhancing lesion in the left frontal lobe (arrows). Arterial spin labelling (C) and dynamic susceptibility contrast-enhanced MR perfusion (D) show no evidence of increased perfusion within the lesion (arrows). Multivoxel MR spectroscopy (TE = 144 ms) (E) shows decreased NAA peak, indicative of neuronal loss. There is no abnormal elevation of choline peak to indicate significantly increased cellular proliferation. Findings are concerning for low-grade neoplasm. Patient subsequently received surgical resection, with final pathology showing IDH-mutant, WHO Grade II glioma. FLAIR, fluid attenuated inversion recovery; IDH, isocitrate dehydrogenase; NAA, N-acetylaspartate; T_1WI, T_1 weighted imaging; TE, echo time.
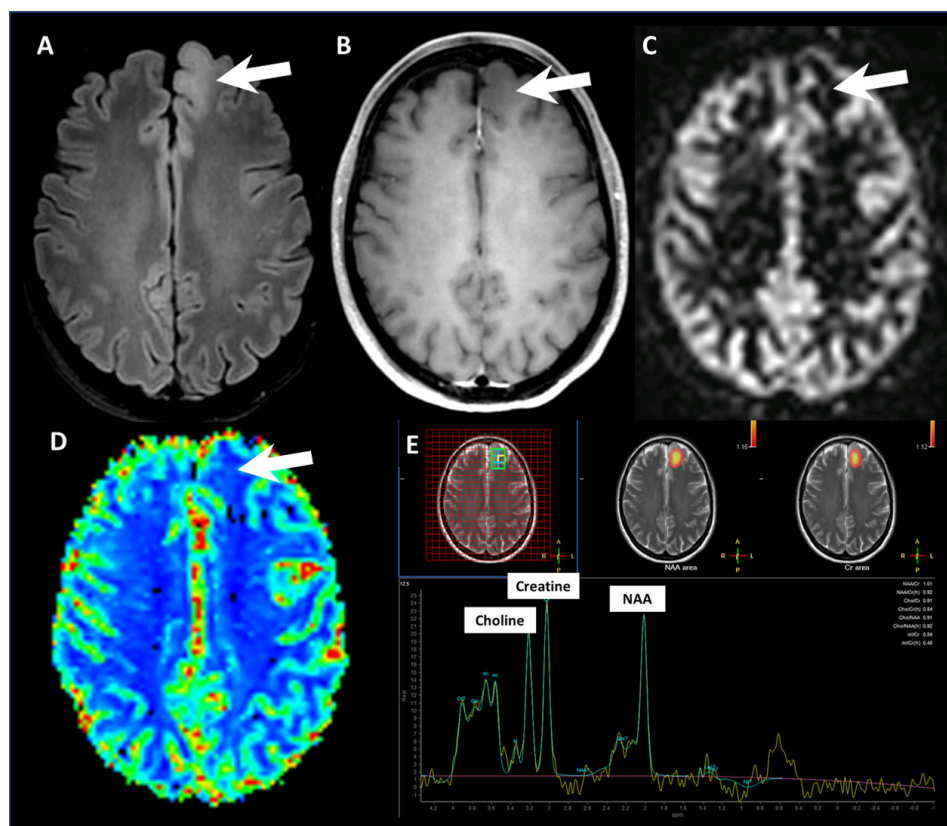
Figure 3. MVNT. A 60-year-old female undergoing brain MRI for ear pressure and retro-orbital pressure sensation. Axial (A) and coronal (B) FLAIR images demonstrate a cluster of multiple small FLAIR hyperintense foci in the left parietal lobe primarily involving superficial subcortical white matter and adjacent deep cortical gray matter (arrows)—characteristic imaging features of MVNT. Lesions show no restricted diffusion or contrast enhancement (not shown). No mass effect or abnormal gyral expansion noted. FLAIR, fluid attenuated inversion recovery; MVNT, multinodular and vacuolating neuronal tumor.
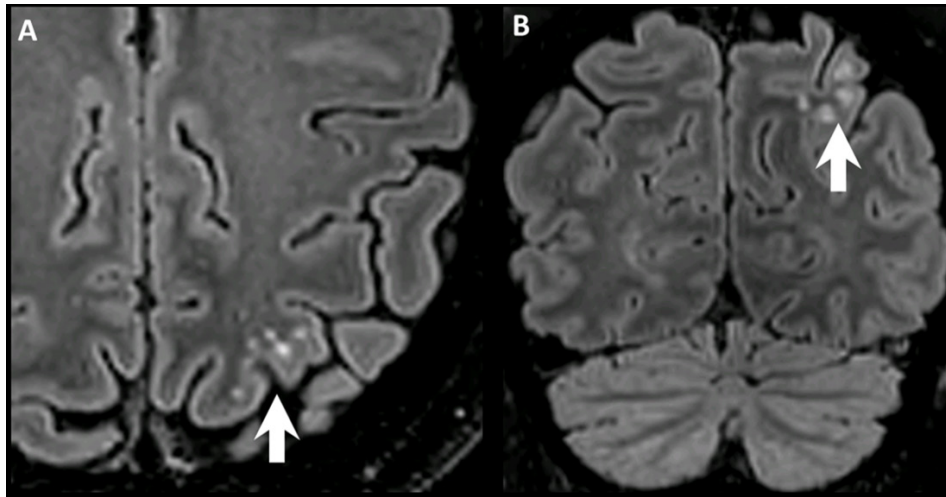
MULTINODULAR AND VACUOLATING NEURONAL TUMOR

Multinodular and vacuolating neuronal tumor (MVNT) is a newly defined benign glial and neural tumor of the central nervous system (CNS) first described by Huse et al. in 2013.[32] It was included in the WHO Classification of CNS Tumors for the first time in 2016. Patients with MVNT may be symptomatic, or MVNT may be incidentally discovered on MRI. The most common clinical manifestations (when symptomatic) are seizures and headaches. The prevalence of incidental MVNT remains unknown. The most common location is in the supratentorial brain, with predilection for the subcortical white matter and overlying cortex. On MRI, MVNT classically presents as a cluster of small, "bubbly" T1 hypointense and T2 hyperintense, non-enhancing foci centered in the subcortical white matter and sometimes involving the overlying cortical gray matter (Figure 3). The affected gyri and cortices are often otherwise normal in appearance. There is no associated vasogenic edema or mass effect.[33] The tumor may have a focal cystic component or subtle enhancement on post-contrast sequences, but these features are uncommon.[32–34] MVNT has a benign course, with stability on clinical and imaging follow-up. As such, it usually requires no surgical intervention unless it is felt to be responsible for patient symptoms.[33,35]

Extra-axial tumors
Incidental meningioma
Meningiomas are common incidental brain tumors in adults, with an incidence of 0.9–2.5%.[2,3,5,10] Most incidental meningiomas are small (size 1.0–3.0 cm) and commonly occur at the cerebral convexity, the falx cerebri, and in the posterior fossa (Figure 4).[10] The majority of incidental meningiomas show interval growth within 3 years after diagnosis although it is usually slow.[36] Management of asymptomatic meningiomas is controversial.[37] Treatment options include conservative

management, surgical resection, or radiation therapy. In general, a conservative approach is recommended for small tumors that do not show interval growth on serial imaging. Surgical resection is typically reserved for patients who manifest neurologic symptoms or show substantial tumor growth on imaging surveillance.[38] If invasive treatment is chosen, radiation may be considered for meningiomas located in surgically challenging anatomic locations, and in patients who are elderly (>65 years old) or with high-risk comorbidities precluding surgery.[37]

VESTIBULAR SCHWANNOMA

Vestibular schwannoma is another commonly encountered incidental extra-axial brain tumor, seen in up to 0.07% of patients.[39] Incidental vestibular schwannomas tend to be small and demonstrate slow interval growth.[40] In addition, the majority of patients have no symptoms or minimal symptoms related to the lesion, allowing for conservative management at the time of diagnosis. However, the rate of tumor growth varies significantly among patients who undergo conservative management, with approximately 15–85% of lesions showing continuous growth.[39] Thus, management of these tumors should be optimized to individual patients. Patients with vestibular schwannomas who are treated conservatively should be followed annually with MRI for 5 years and gradually less frequently thereafter, if the lesion remains stable in size.[41,42] Cystic tumors tend to grow faster than solid tumors and as such they should be followed more closely, particularly in cases with initial solid tumors that express interval cystic changes.[39,41]

Intraventricular tumors
There is a wide variety of tumors that may arise in the ventricles, including ependymoma, subependymoma, central neurocytoma, choroid plexus tumors, meningioma, and metastases.[43,44] Intraventricular tumors can be subclinical and discovered incidentally on MRI, although the true prevalence is unknown.

Figure 4. Incidental meningioma. A 69-year-old female undergoing brain MRI for right tinnitus and intermittent vertigo. Axial (A) and coronal (B) contrast-enhanced T_1WI demonstrate a dural-based enhancing extra-axial mass at the right posterior parietal convexity with focal invasion into the superior sagittal sinus, findings most consistent with meningioma. Patient was managed conservatively, and the tumor has been stable on serial follow-up MRI for 9 years.
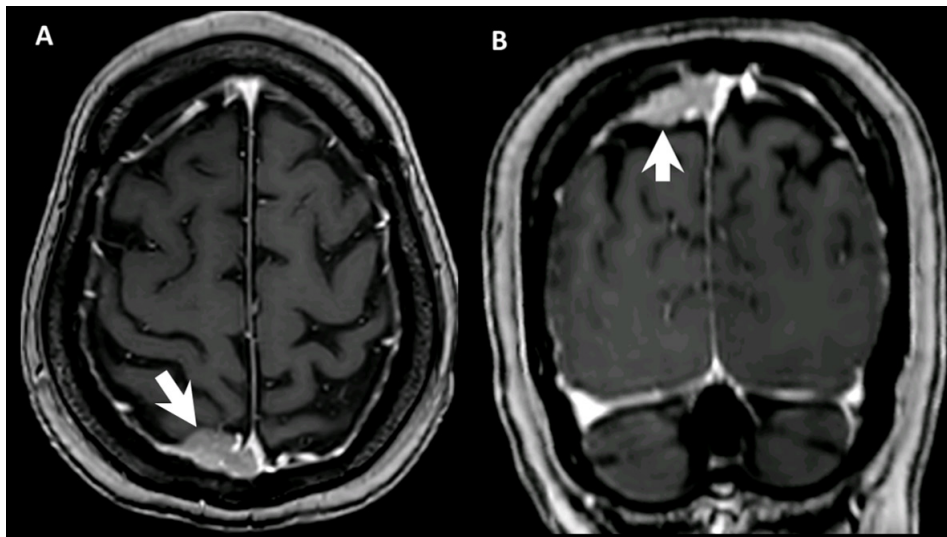
When symptomatic, patients may present with signs and symptoms related to obstructive hydrocephalus and increased intracranial pressure. Tumors in the supratentorial compartment may cause focal neurologic deficits or seizure.[43,44] Differentiating intraventricular tumors on imaging can be challenging because there is considerable overlap of imaging features. However, differential diagnosis can be narrowed by using tumor location, patient age, and underlying medical conditions.[43,44] Ependymomas and choroid plexus tumors commonly occur in the fourth and lateral ventricles and are more prevalent in children. Subependymomas occur in the same location but typically affect middle-aged patients. In contrast to ependymomas and choroid plexus tumors, most subependymomas show no or minimal enhancement (Figure 5). Central neurocytomas frequently occur in the lateral ventricles and may originate from the septum pellucidum or ventricular walls. Intraventricular meningiomas most commonly occur in the atrium of the lateral ventricles. Advanced MRI techniques such as MR spectroscopy can help

with characterizing intraventricular tumors.[45,46] However, more research is needed to evaluate the value of these techniques. The primary treatment of intraventricular tumors is surgery.[43,44]

Pineal tumors

The pineal gland is a small neuroendocrine organ that is composed of two cell types, pineocytes and astrocytes.[47] Tumors in the pineal region may originate from the pineal gland or nearby structures. Germ cell tumors are by far the most common pineal tumors (50–60% of all pineal tumors) followed by pineal parenchymal tumors (30%).[47] Other less common pineal tumors include glioneuronal or neuronal tumors, embryonal tumors (*e.g.* atypical teratoid rhabdoid tumor, medulloblastoma), meningiomas and metastases. Pineal tumors are occasionally incidentally found on cross-sectional imaging; however, prevalence of incidental pineal tumors remains unknown (Figure 6).[10] Imaging plays a limited role in differentiating pineal gland tumors due to

Figure 5. Incidental subependymoma. A 48-year-old female undergoing brain MRI for imaging surveillance of multiple sclerosis. Axial T_2WI (A), contrast-enhanced T_1WI (B), FLAIR (C), and coronal FLAIR (D) demonstrate a circumscribed non-enhancing mass in the left lateral ventricle. There were no symptoms attributed to the mass. Patient underwent surgical resection, with final pathology showing subependymoma (WHO Grade I). Note multiple T2 hyperintense white matter lesions in relation to underlying demyelinating disease (arrowheads on C and D). FLAIR, fluid attenuated inversion recovery.
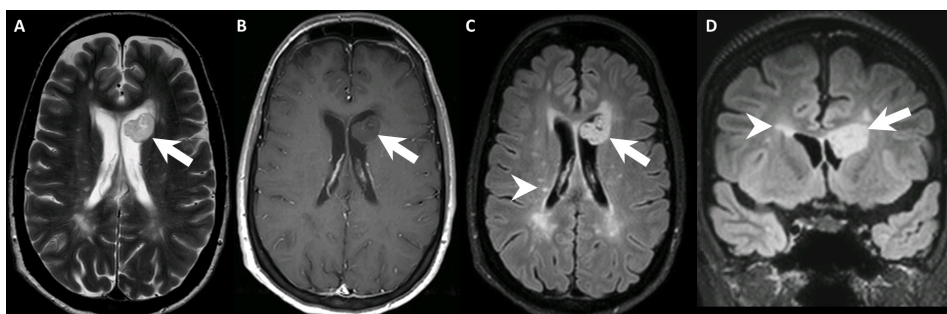
Figure 6. Incidental pineal tumor. A 59-year-old female undergoing brain MRI for headache after a mechanical fall. Sagittal high-resolution T_2WI (A), sagittal (B) and axial (C) contrast-enhanced T_1WI demonstrate a small extra-axial enhancing mass at the pineal region. There is no significant local mass effect or obstructive hydrocephalus. Patient was managed conservatively, and the mass has been stable on serial follow-up MRI for 2 years. It is favored to be benign tumor such as meningioma or pineocytoma.
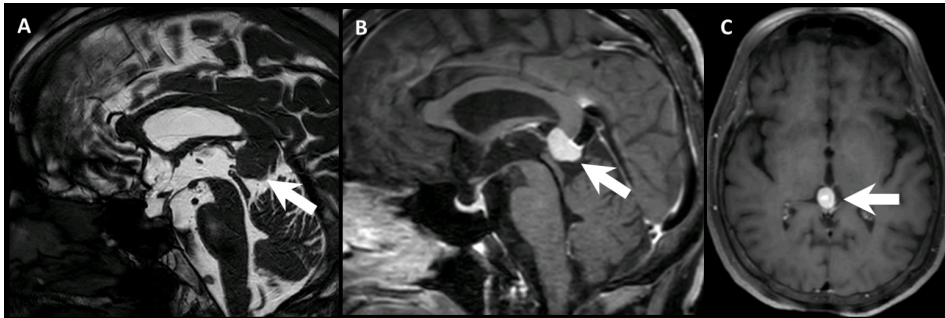
an overlap in imaging appearance.[47] The distribution of intralesional calcifications on CT may narrow the differential diagnosis. Pineal parenchymal tumors characteristically contain peripheral calcifications which are described as "exploded calcifications", whereas calcifications within germ cell tumors are more centrally located due to the engulfed pineal gland.[47] Treatment of benign pineal tumors is surgical resection. Multimodality therapy including surgery and adjuvant chemoradiation is required for treatment of malignant tumors such as pineoblastoma. Germinomas are highly radiosensitive and can be treated with radiation therapy alone.[47,48]

Pituitary tumors/incidentalomas

Incidental pituitary lesions discovered on brain MRI (so-called pituitary incidentalomas) are relatively common, with prevalence ranging from 0.3 to 12.3%.[1–3,10] The most common incidental pituitary lesions are pituitary cysts (Rathke's cleft and pars intermedia cysts) (prevalence = 0.7–11.8%) (Figure 7) and pituitary adenoma (prevalence = 0.15–0.5%) (Figure 8).[1–3,5,10,49,50] Other sellar lesions affecting the pituitary such as metastasis are rarer.[51] Some pituitary incidentalomas may not represent true

pathology, and may be secondary to technical artifact, physiologic pituitary hyperplasia, or normal anatomical variation.[50,52]

A clinical practice guideline for pituitary incidentalomas published by The Endocrine Society in 2011[53] recommends that patients with pituitary incidentalomas undergo complete clinical and laboratory evaluation for potential hormonal dysfunction, regardless of clinical symptoms. In addition, patients should undergo a formal visual field examination if the lesion abuts the optic nerves or optic chiasm. Surgery is indicated in patients with visual disturbance, imaging findings of optic nerve or chiasm abutment, other neurologic deficits due to compressive effects, or if the tumor is hormone-secreting (except for prolactinoma which may be medically managed). Incidentaloma patients who do not meet surgical criteria should receive clinical and imaging follow-up with MRI to monitor for growth and symptom development. Frequency of follow-up MRI depends on several factors including lesion size (*i.e.* micro- *vs* macroadenoma), detection of growth or change in features on serial imaging, and whether or not the patient develops signs or symptoms potentially related to the lesion.[53]

Figure 7. Rathke's cleft cyst. A 38-year-old female undergoing brain MRI for postpartum headache. Sagittal T_1WI (A) and coronal T_2WI (B) demonstrate a small well-circumscribed cystic lesion centered in the pars intermedia of the pituitary gland (arrows). The lesion contains T1 hyperintense and T2 hypointense content likely secondary to high proteinaceous fluid content. Findings are most consistent with Rathke's cleft cyst. The lesion has been stable on serial follow-up MRI.
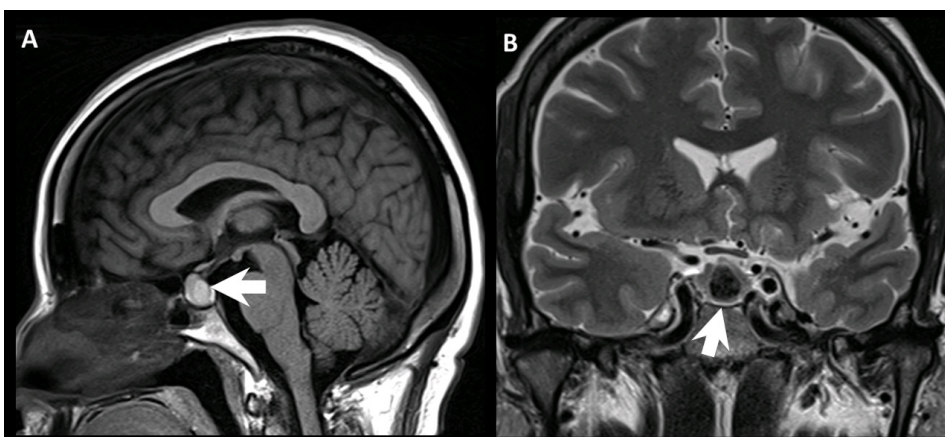
Figure 8. Incidental pituitary adenoma. A 51-year-old male undergoing MRI for surveillance for the left vestibular schwannoma previously treated with radiation therapy. Coronal contrast-enhanced T_1WI with fat suppression (A) demonstrates a small hypoenhancing lesion centered in the right lateral aspect of pituitary gland (arrow), most consistent with pituitary adenoma. There was no evidence for endocrinological dysfunction on physical exam or laboratory work-ups. The lesion was presumed to be a nonfunctioning pituitary microadenoma. Note a small enhancing vestibular schwannoma in the left internal auditory canal and cerebellopontine angle cistern (arrowhead on Figure 8B).
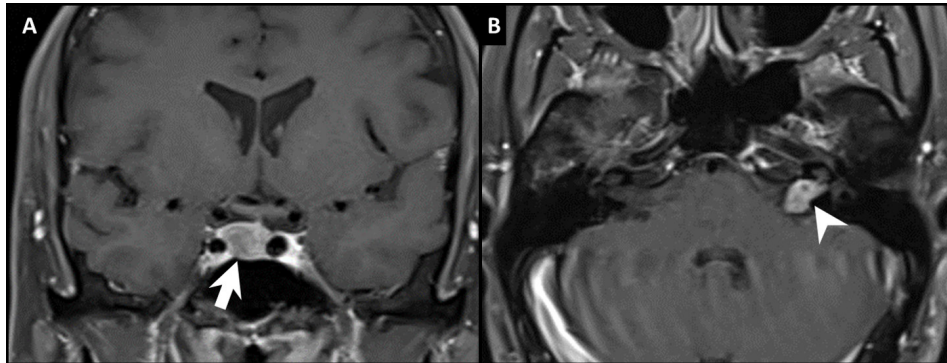
In 2018, the American College of Radiology (ACR) published recommendations for management of incidental pituitary lesions in adults detected on CT, MRI, or positron emission tomography/CT.[49] There are three important imaging features of pituitary lesions considered in the algorithm, including: (1) composition (solid, cystic, or mixed solid-cystic), (2) size, and (3) mass effect or invasion of nearby structures (optic nerves, optic chiasm, and cavernous sinuses). According to the ACR recommendations, and in contradistinction to the Endocrine Society Guidelines, a simple cystic pituitary lesion is likely a Rathke's cleft cyst, and requires no further work-up unless causing mass effect or invading surrounding structures. Small solid or mixed solid-cystic incidental, asymptomatic lesions with size <5 mm are most often clinically insignificant and do not require further follow-up imaging. Patients with solid or mixed solid-cystic lesions between 5 and 10 mm in size should receive clinical evaluation to determine if there is associated endocrinological dysfunction. If a lesion is deemed to be a non-functioning adenoma, follow-up imaging may not be required. Patients with

solid or mixed solid-cystic lesions measuring >10 mm should receive both clinical evaluation and follow-up imaging with MRI in 6–12 months. Any lesions that demonstrate mass effect or invasion of surrounding structures on imaging should receive neurosurgical and/or endocrine consultation.[49] The ACR recommendations for management of incidental pituitary lesions detected on CT or MRI in adults are summarized in Figure 9. As these represent generalized recommendations (and do not cover all imaging features of incidental pituitary lesions or all clinical scenarios), follow-up protocols should be optimized to each individual patient.

INTRACRANIAL CYSTIC LESIONS
Pineal cyst
Pineal cysts are a relatively common incidental finding on brain MRI, with a prevalence of approximately 1.0–2.0%[54]. The prevalence rises to a peak in late childhood and decreases with advancing age during adulthood.[54,55] Simple pineal cysts

Figure 9. The American College of Radiology recommendations for management of incidental pituitary findings on CT or MRI. Reference: Hoang JK, Hoffman AR, Gonzalez RG, Wintermark M, Glenn BJ, Pandharipande PV, et al. Management of Incidental Pituitary Findings on CT, MRI, and (18)F-Fluorodeoxyglucose PET: A White Paper of the ACR Incidental Findings Committee. *J Am Coll Radiol.* 2018;15(7):966–72. The flow chart is reproduced with permission from the *Journal of the American College of Radiology*.
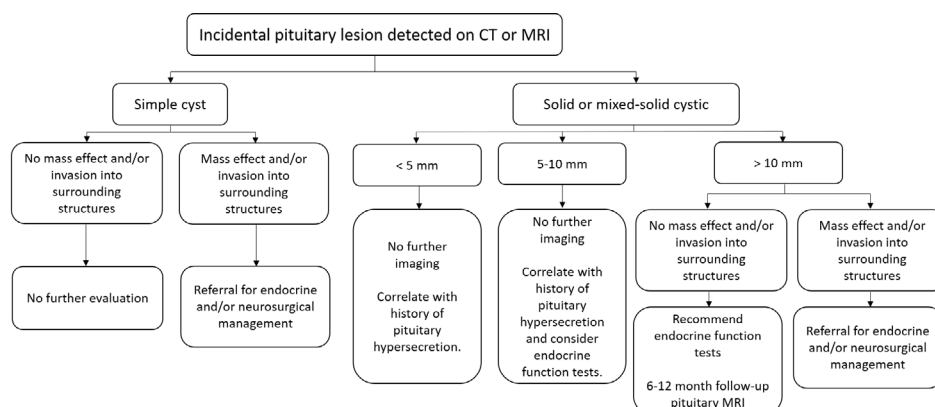
Figure 10. Incidental pineal cyst. A 31-year-old female undergoing brain MRI for dizziness. Axial T_2WI (A), FLAIR (B), contrast-enhanced T_1WI (C) and sagittal contrast-enhanced T_1WI (D) demonstrate a 2.0 cm cystic lesion centered in the pineal region (arrows). No abnormal solid component or solid enhancement is present. The cyst has been stable on serial follow-up MRIs for 4 years. FLAIR, fluid attenuated inversion recovery
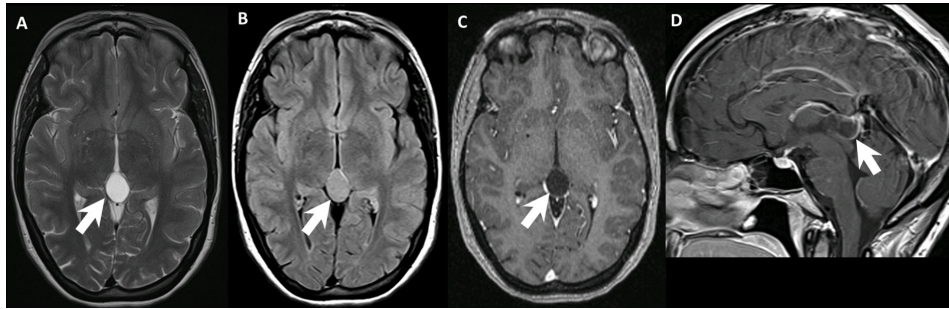
are unilocular, smooth, and thin-walled, with size ranging from a few millimeters to >25 millimeters (Figure 10).[56] Atypical or complex pineal cysts are defined as having internal septations, solidly enhancing components, wall thickness >2 mm, calcifications, and/or hemorrhage. Any enhancement is usually located peripherally, at the posterior aspect near the internal cerebral veins.[56]

Most patients with pineal cysts are asymptomatic. In a large retrospective study, 80% of pineal cysts were stable on serial follow-up MRI and clinical evaluation (mean interval = 3.4 years from initial exam).[54] Less than 3% of lesions showed a slight increase in size, with a mean change in maximum diameter of 3.5 mm. None of the patients in this study had or developed symptoms related to pineal cysts at baseline or during the follow-up period.[54] This benign course is supported by several additional studies.[57–60] In rare cases, pineal cysts may become symptomatic due to obstructive hydrocephalus, or cranial nerve IV compression with resultant gaze palsy or Parinaud syndrome (a brainstem syndrome caused by damage to the vertical gaze center in the posterior commissure of the dorsal midbrain).[54,61] Although extremely rare, sudden death due to acute hydrocephalus from pineal hemorrhage has been reported.[62]

Management of incidental pineal cysts remains controversial. In children, serial follow-up imaging and clinical evaluation are typically recommended, to exclude interval growth or development of symptoms.[56] Children with pineal cysts ≥15 mm are more at risk of developing visual impairment and hydrocephalus.[63] It should be noted that the majority of atypical pineal cysts are not primary pineal tumors; however, imaging cannot always reliably distinguish the two, and thus serial follow-up imaging is still recommended for atypical pineal cysts at many institutions to ensure their stability.[56,64] Some clinicians may elect to obtain annual MRI to follow up atypical pineal cysts for 5 years and no long-term follow-up is required if lesions are stable.[56]

ARACHNOID CYST
Arachnoid cyst is a common incidental intracranial cystic lesion, with a prevalence of 0.3–3.1% in the adult population.[2,9] A systematic review of incidental findings on brain MRI found that, excluding changes of cerebrovascular disease, arachnoid cyst is the single most common non-neoplastic incidental finding.[2] The etiology of arachnoid cyst remains uncertain but it is thought to be due to a splitting of the arachnoid membrane during development.[65,66] Occasionally, arachnoid cysts may be caused by preceding trauma or inflammation affecting the subarachnoid space. The most common locations of arachnoid cyst are in the middle cranial fossa and retrocerebellar cistern. The vast majority of arachnoid cysts are incidental and asymptomatic, and do not require follow-up imaging.[67] However, they may cause symptoms from regional mass effect or less commonly from cyst rupture resulting in subdural hygroma or intracystic hemorrhage due to rupture of blood vessels around the cyst wall.[68,69] In such cases, surgical intervention (*e.g.* cyst excision, fenestration, or shunting) is indicated.[70]
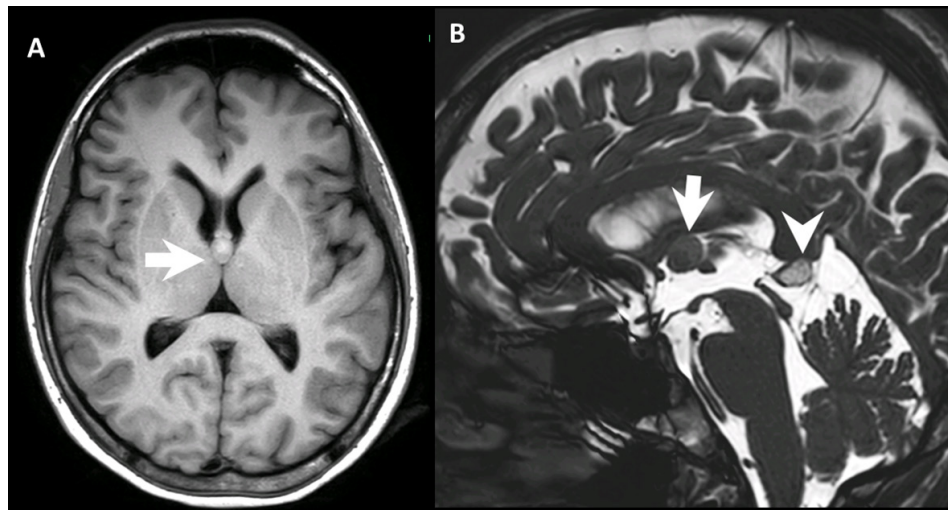
COLLOID CYST
Colloid cysts are benign intracranial cystic lesions that arise from ectopic endodermal tissue that migrates into the velum interpositum during development. Most colloid cysts are discovered incidentally on MRI, with a prevalence of 0.01–0.07%.[2,71] They are most commonly located within the anterior third ventricle in the vicinity of the foramina of Monro.[71] They typically present incidentally as hyperdense cystic lesions on CT and cysts with variable signal contents on MRI (depending on fluid protein concentration) (Figure 11). The lesions may demonstrate discontinuous thin rim enhancement, due to adjacent enhancing septal veins.[56] The most common clinical presentation is headache secondary to increased intracranial pressure from obstruction at the foramina of Monro. Symptomatic colloid cysts require surgical intervention to restore CSF flow.[56,71]

The natural history of incidental colloid cysts is less well-studied. A recent systematic review demonstrated that 6–19% of asymptomatic colloid cysts showed some progression over 3–5 years of radiological follow-up, with a 5–15% risk of progression requiring surgical treatment in the 5 years following diagnosis[72]. Beaumont et al developed the Colloid Cyst Risk Score (CCRS)–a simple 5-point score to stratify risk of developing symptoms including obstructive hydrocephalus in patients with colloid cysts.[71] The five major risk factors implemented in CCRS include: (1) age at presentation, (2) presence of headache, (3) axial cyst diameter >7 mm, (4) FLAIR hyperintensity on MRI, and (5) cyst location in an anatomical risk zone (*i.e.* within the third ventricle). A CCRS score ≤2 is considered to be low-risk, whereas

Figure 11. Incidental colloid cyst. A 37-year-old female undergoing brain MRI for concussion. Axial T_1WI (A) and sagittal high-resolution T_2WI (B) demonstrate a 1.0 cm cystic lesion centered in the foramen of Monro (arrows), most consistent with colloid cyst. No imaging evidence of obstructive hydrocephalus. The cyst has been stable on serial follow-up MRIs for 5 years. Incidentally noted small pineal cyst (arrowhead on image B).

a CCRS score ≥4 represents a high-risk lesion.[71] The CCRS is found to have good interrater reliability and high predictive value on independent validations.[73–75] It is recommended that all patients with incidental colloid cysts be evaluated by neurosurgery to ascertain symptoms and discuss conservative *vs* surgical treatment options.[56]

EPIDERMOID CYST

Epidermoid cysts are benign slow-growing congenital inclusion cysts arising from ectodermal remnants during neural tube closure.[76] Epidermoid cysts slowly grow due to desquamation of keratinizing squamous epithelium along the cyst wall. Many epidermoid cysts are clinically silent but may occasionally cause symptoms due to local mass effect.[76] The prevalence of incidental epidermoid cysts on brain MRI is 0.01–0.06%.[2] They are commonly found in the basal cisterns, with cerebellopontine angle cistern being the most common location.[76,77] Rarely, they may occur in the brain parenchyma or inside the bones.[76,78] Although rare, malignant transformation of epidermoid cysts and aseptic meningitis from cyst rupture have been reported.[79,80] On MRI, epidermoid cysts classically present as non-enhancing cystic lesions with signal resembling CSF on T_1WI and T_2WI. They are hyperintense on FLAIR sequence due to incomplete signal suppression and demonstrate a variable degree of diffusion restriction on DWI which is a key distinguishing imaging feature from arachnoid cysts[76] . Occasionally, epidermoid cysts may exhibit atypical radiological features, with intrinsic hyperintensity on T_1WI (so-called white epidermoid cyst). This may be due to the presence of cholesterol crystals or internal hemorrhage.[77,81] Treatment is surgical. Recurrence may occur if the cyst wall is not completely removed.[82]

NEUROENTERIC CYST

Neuroenteric cysts are rare congenital cystic lesions derived from the retained endodermal cells within the neuroenteric canal connecting between the foregut and the notochord.[83,84] In the neuroaxis, neuroenteric cysts are most commonly found in the cervical and upper thoracic spine. Intracranial neuroenteric cysts are rare and account for only 10–18% of all neuroenteric cysts.[83,84] Most intracranial neuroenteric cysts (70–90%) occur in the posterior fossa. The true prevalence of incidental neuroenteric cysts is unknown. On histopathology, the cyst wall is lined by simple or pseudostratified cuboidal or columnar cells similar to gastrointestinal or respiratory epithelium. On MRI, most neuroenteric cysts are iso- or hyperintense to CSF on T_1WI (due to high proteinaceous content), hyperintense on T_2WI and T_2-FLAIR, and usually have mild restricted diffusion on DWI. Most neuroenteric cysts show no enhancement on post-contrast sequences (Figure 12).[83,84] Imaging features of neuroenteric cysts are non-specific and largely overlap with other cystic lesions such as arachnoid cysts or epidermoid cysts, making radiological diagnosis difficult. Treatment of symptomatic neuroenteric cysts is surgical excision.[83,85]

ANATOMICAL VARIANTS AND DEVELOPMENTAL STRUCTURAL FINDINGS

There are a wide variety of anatomical variants and structural developmental anomalies that are incidentally discovered on brain MRIs, such as Chiari I malformation, gray matter heterotopia, and CSF space variations.[5] Anatomical variants of the ventricular system such as asymmetric size and morphology, coarctation, cavum septum pellucidum, cavum vergae, and cavum velum interpositum are very common in healthy individuals. These variations are generally considered to be within the normal spectrum and have no clinical significance in the appropriate clinical context.[86] However, some studies have suggested that the presence of cavum septum pellucidum and cavum vergae is associated with cognitive impairment in patients with repetitive head trauma.[87]

One of the more common anatomical variants in the posterior fossa is cerebellar tonsillar ectopia or Chiari I malformation. The

Figure 12. Neuroenteric cyst. A 37-year-old female undergoing brain MRI for headaches. Axial T_1WI (A), axial T_2WI (B), axial contrast-enhanced T_1WI (C), axial DWI (D) and ADC maps (E) show a small extra-axial cystic lesion centered in the right cerebellomedullary cistern (arrows). The lesion is slightly hyperintense to CSF on T1 and isointense on T2, with no definite enhancement. There is no diffusion restriction. The lesion exerts minimal mass effect on the lower brainstem. The patient underwent surgical excision, with final pathology showing neuroenteric cyst. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging.
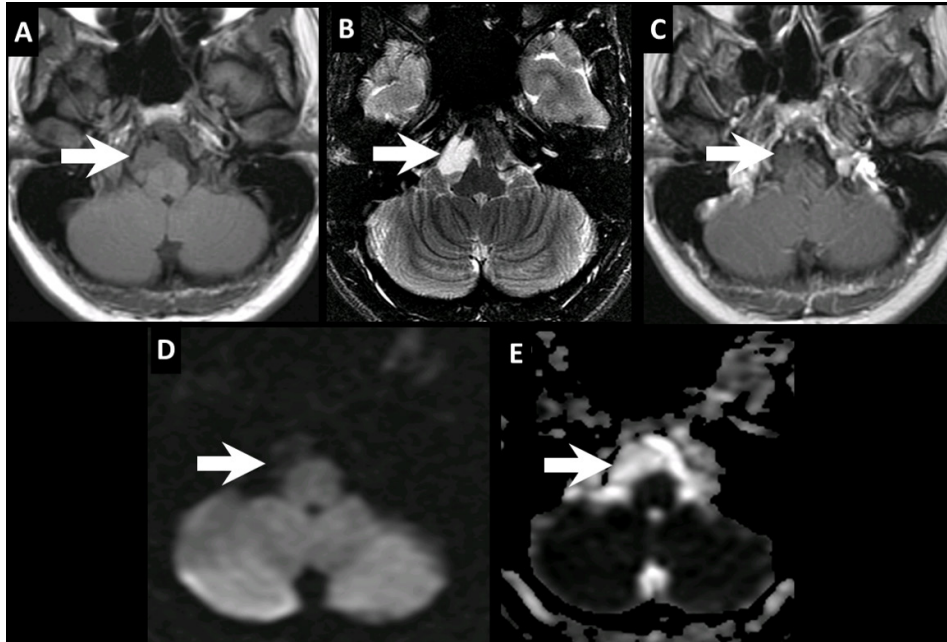
definition differentiating between cerebellar tonsillar ectopia and Chiari I malformation is somewhat complicated because the use of these terms is not uniform in literature. In general, cerebellar tonsillar ectopia is a broader term used to describe patients whose cerebellar tonsils are below the foramen magnum encompassing both congenital and acquired etiologies such as intracranial hypertension or craniospinal hypotension.[88] Chiari I malformation is defined as >5 mm inferior protrusion of the cerebellar tonsils below the opisthion-basion line. Patients with Chiari I malformation are often symptomatic and may have other structural abnormalities such as spinal syrinx.[89,90] Cerebellar tonsillar ectopia can be seen in up to 0.9% of the general adult population.[3] Acquired cerebellar tonsillar ectopia from intracranial hypertension may have a peg-like appearance, mimicking Chiari I malformation. Thus, the findings of cerebellar tonsillar ectopia or Chiari I malformation should be interpreted along with other radiographic signs and within the clinical context.[90] The natural history of patients with asymptomatic or mildly symptomatic Chiari I malformation is relatively benign, with the vast majority of patients remaining clinically stable or improving with conservative management.[91,92] Surgical intervention (suboccipital craniectomy and C1 posterior arch resection) is typically reserved for patients with symptoms attributable to either the Chiari I malformation or syrinx, such as Valsalva-induced headache or cranial nerve dysfunction.[89]

CONCLUSION

Incidental findings on brain MRI amongst patients undergoing imaging are common. Brain parenchymal changes secondary to cerebrovascular disease and intracranial tumors, particularly meningiomas, are age-related, with increasing prevalence in elderly population. Management of these incidental findings is primarily based on the natural history of the lesions and clinical background of an individual patient.

AUTHOR DISCLOSURES

All authors have no conflicts of interest to disclose and there were no financial incentives that would alter the contents of this manuscript.

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