PICTORIAL REVIEW

Brain stones revisited—between a rock and a hard place

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Abstract

Objectives and methods Large intracranial calcifications are occasionally encountered in routine computed tomography (CT) scans of the brain. These calcifications, also known as "brain stones", can be classified according to location and aetiology. Combining imaging findings with relevant clinical history and physical examination can help narrow down the differential diagnosis and may allow confident diagnosis in certain situations.

Results This article provides a pictorial review illustrating various clinical entities resulting in brain stones.

Discussion Based on location, brain stones can be classified as extra- or intra-axial. Extra-axial brain stones comprise tumours and exaggerated physiological calcifications. Intra-axial brain stones can further be classified according to aetiology, namely neoplastic, vascular, infectious, congenital and endocrine/metabolic. Imaging findings combined with essential clinical information can help in narrowing the differential diagnosis, determining disease state and evaluating effect of therapy.

Teaching Points

• *Based on location, brain stones can be either extra- or intra-axial.*

• *Extra-axial brain stones comprise tumours and exaggerated physiological calcifications.*

• *Intra-axial aetiologies include neoplastic, vascular, infectious, congenital and endocrine/metabolic.*

• *CT scan is the mainstay in identifying and characterising brain stones.*

• *Certain MRI sequences (gradient echo T2* and susceptibility-weighted imaging) are considered adjunctive.*

Introduction

Brain stones or cerebral calculi are defined as large solid intracranial calcifications [1, 2]. These lesions were once thought to be rare and were mostly seen in patients with seizures [1, 2]. Intracerebral haematomas, tuberculomas and brain abscesses were postulated to be the most common underlying pathologies leading to the development of these intracranial calcifications [2, 3]. The introduction of cross-sectional imaging modalities, particularly computed tomography (CT), has allowed easier identification of intracerebral calcifications and specifically the lesser common brain stones in a wide range of clinical entities and disease states [4]. This article provides a pictorial review illustrating various clinical entities resulting in brain stones (Table 1).

Definition

Tiberin [1] defined "brain stones" or "cerebral calculi" as general terms referring to "large, solitary or multiple, well-circumscribed bony hard areas of pathological intracerebral calcification", the aetiopathologies of which were thought to be the end result of chronic, insidious "non-neoplastic space-occupying lesions". The lack of specific data regarding the amount and configuration of calcium salts that would allow detection by plain x-ray is compounded by the fact that no consensus exists on how histological characteristics of intracranial calcifications explain the appearance of intracranial

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**Table 1** Classification of brain stones according to location and aetiopathology

Location/aetiology	Examples
Extra-axial	Meningiomas, dural osteomas, calcifying tumours (e.g., craniopharyngiomas), calcifying pseudoneoplasms of the neuraxis (CAPNON), exaggerated physiological calcifications
Intra-axial	
Neoplastic	Oligodendrogliomas, medulloblastoma, germ cell tumours, primitive neuroectodermal tumours (PNET), dysembryonic neuroectodermal tumours (DNET), gangliomas, pilocytic astrocytomas
Vascular	Cavernous malformations, arteriovenous malformations, dystrophic calcification in chronic infarction, chronic vasculitis, aneurysms
Infectious	TORCH (toxoplasmosis, rubella, cytomegalovirus, herpes simplex), tuberculosis, parasitic infections (e.g. neurocyticercosis, cerebral hydatid cyst disease)
Congenital	Sturge-Weber syndrome, tuberous sclerosis, lipomas, neurofibromatosis
Endocrine/metabolic	Fahr's syndrome, hypoparathyroidism, pseudohypoparathyroidism, hyperparathyroidism

calcifications on plain x-rays [3, 5, 6]. Similarly, there is no clear cutoff value as to what "large" pertains to in the definition of brain stones. The superiority of CT in detecting macroscopic intracranial calcifications as compared to plain x-rays has resulted in routine visualisation of various forms and sizes of intracranial calcification [7]. Despite the ability of CT to quantify calcification, the subjective designation of intracranial calcifications as brain stones has been considered of less importance lately, although the challenge of reaching a radiological diagnosis on the basis of their presence has remained an intellectual curiosity [2].

Imaging modalities

In a historical context, conventional skull x-rays have been most instrumental in identifying brain stones. The use of various projections has helped localise brain stones and, in some instances, narrow the differential diagnosis [6, 8, 9]. The introduction of the Potter-Bucky grid in the 1920s further increased the detection rate of skull x-rays [5]. CT (and especially the multidetector CTs), because of their multiplanar reconstruction (MPR) and 3D reconstruction capabilities, have supplanted the use of skull x-rays in the diagnosis of intracranial pathology. They are also considered to be better than magnetic resonance imaging (MRI) in identifying and characterising intracranial

Fig. 1 A 59-year-old female with back pain and an abnormal bone scan that showed a focal lesion in the skull. The faint calcification seen at the lateral skull x-ray (a) was confirmed on non-contrast-enhanced CT (NCECT) to be a calcified meningioma overlying the right frontal cortex (b), better visualised on the 3D reconstruction (c). Note the displacement of adjacent brain parenchyma ("cortical buckling"), which strongly suggests the extra-axial location of this lesion
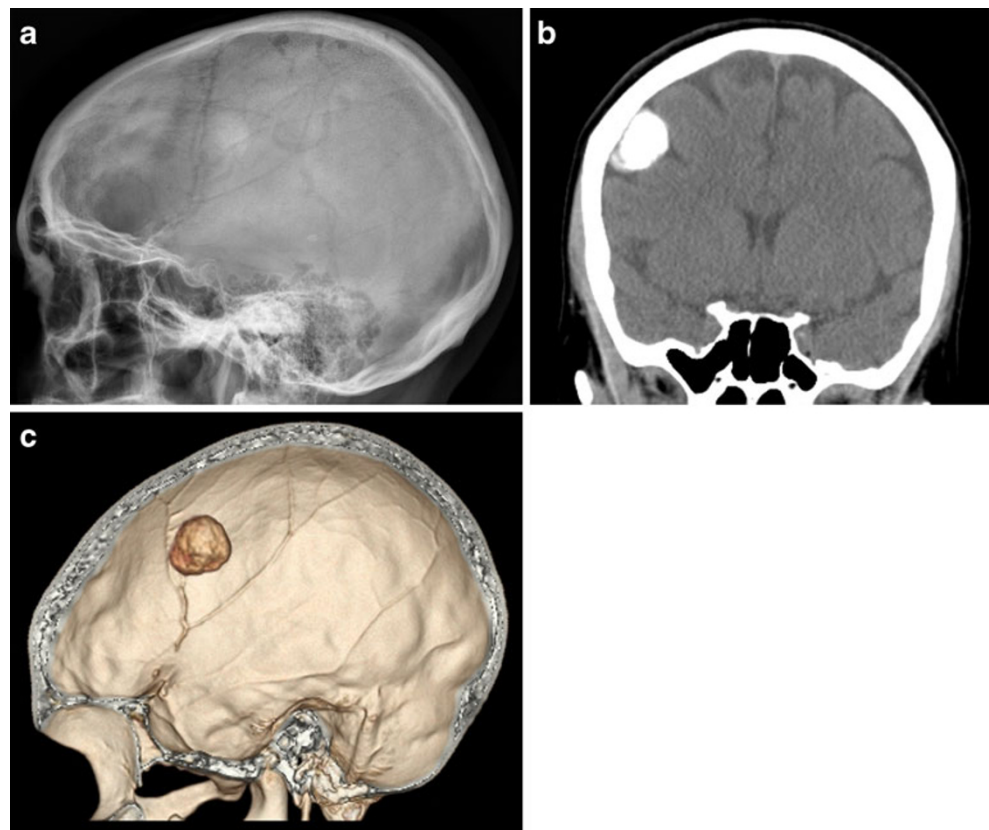
calcification [4, 10], although newer MRI sequences such as gradient echo T2* and susceptibility-weighted imaging have been shown to be promising in detecting intracranial calcification [11–13]. In certain clinical situations, neurological symptoms have even been shown to correlate better with MRI findings than with the corresponding CT-identified calcifications [14–16].

Classification of brain stones

As with many other intracranial pathologies, brain stones can be localised and classified as either extra- or intra-axial. Various imaging findings have been found to suggest an extra-axial localisation of brain lesions such as buckling of adjacent white matter, expansion of the ipsilateral subarachnoid space, presence of bony reactions and the "dural tail" sign [17].

Extra-axial

The most common extra-axial aetiologies of brain stones include meningiomas, dural osteomas, calcifying tumours (e.g. craniopharyngiomas) as well as exaggerated physiological calcifications [5, 8, 18]. Macroscopic calcifications are present in meningiomas in up to 60 % of cases, the pattern of which may range from diffuse, rim or sand-like to focal or even globular (Fig. 1) [8, 18, 19]. Calcified meningiomas can be associated with focal thickening of the overlying skull bones or with dilatation of the adjacent air-containing sinus

Fig. 2 Dural osteoma. A 53-year-old female who presented with a headache and distant history of head trauma. The FLAIR **(a)** and MIP-SWI **(b)** MRI sequences showed an area of signal loss (*white arrows*) in the right temporal lobe initially thought to represent an enlarged post-traumatic air cell. Subsequent NCECT **(c)** and 3D reconstruction **(d)** revealed a solid calcified lesion (*black arrow*) adherent to the squamous part of the temporal bone. Note the cortical buckling **(a)**, which suggests an extra-axial location. The homogeneously solid calcification **(c)** suggests a dural osteoma instead of a dural ossification
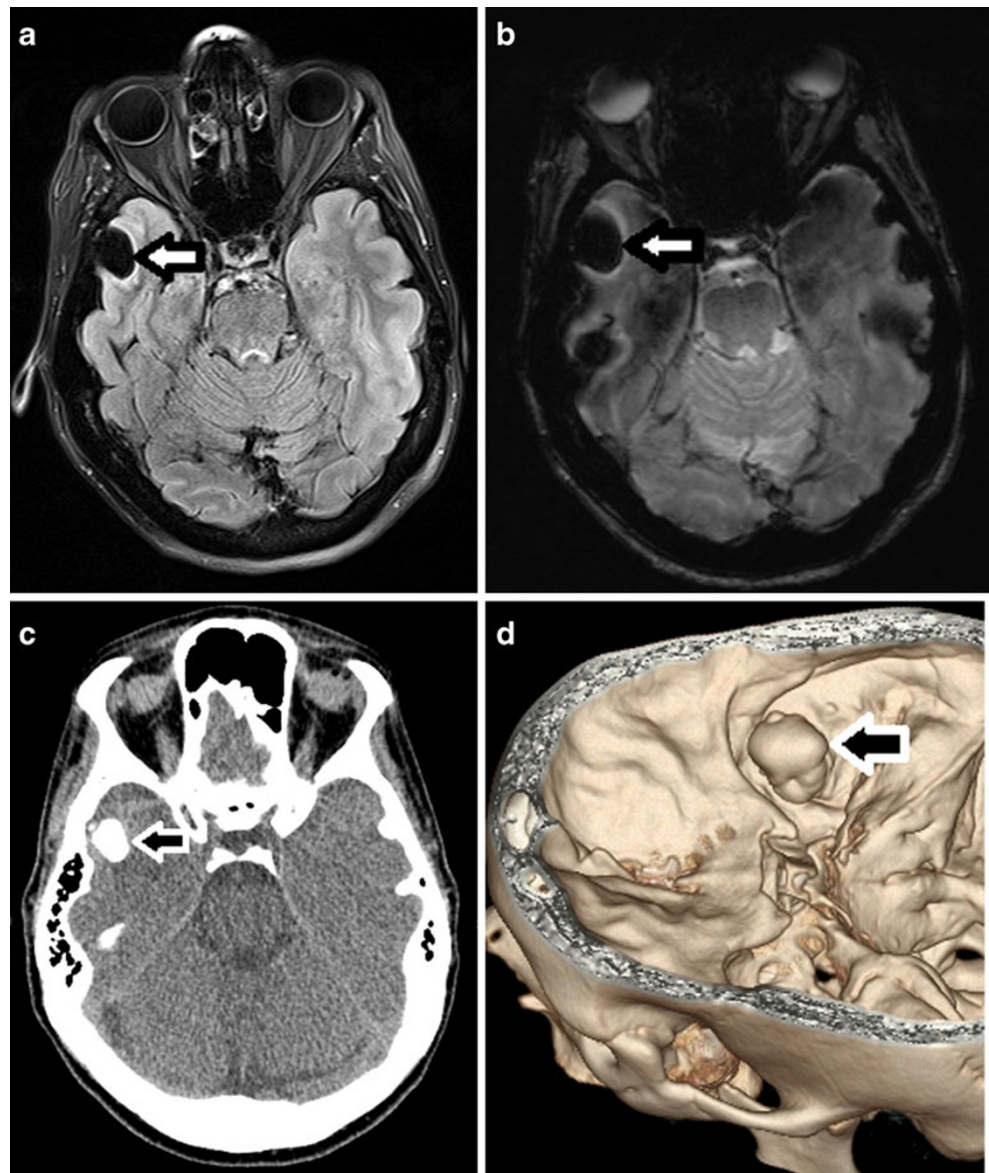
of the skull base [20]. Dural osteomas are believed to be a result of new bone formation arising from the dura and falx; conversely, intra-axial intracerebral osteomas are postulated to originate from primitive mesenchymal cells that have migrated from connective tissue (Fig. 2). Unique to these rare ossified tumours is the fact that they do not possess a laminated bony architecture, but present as a solid mass of calcification [21, 22]. Craniopharyngiomas are frequently located in the suprasellar region and usually present with haemorrhage and calcification mainly in an amorphous and sometimes lobulated pattern [8, 23]. Calcifying pseudoneoplasms of the neuraxis (CAPNON) are extremely rare fibro-osseous lesions that can be extra- or intra-axial. Histologically, these benign lesions are composed of a pathognomonic chondromyxoid matrix in an amorphous or nodular pattern as well as palisading spindle or epithelioid cells and varying proportions of fibrous stroma [24–26]. When present in the skull base, they are known to symptomatically encase cranial nerves. Lastly, exaggerated physiological calcifications can theoretically lead

to identification of brain stones in radiological examinations. Structures known to calcify include the pineal gland, choroid plexus, habenula, dura and arachnoid, falx cerebri, tentorium cerebelli, superior sagittal sinus, petroclinoid and interclinoid ligaments, arachnoid granulations and intra-axially the basal ganglia and cerebellum [9, 27]. Although physiological calcifications are usually considered of no clinical significance, the presence of pathology may be suspected in cases of abnormal increase in their usual size [e.g. normal choroid plexus calcification versus a choroid papilloma (Fig. 3) or carcinoma], location (e.g. displaced pineal gland due to tumour) or age at presentation [e.g. age-related basal ganglia calcification versus Fahr's disease in younger individuals (Fig. 9)] [8, 18, 19].

Dural calcifications and ossifications, especially of the falx cerebri, can also appear as "brain stones". They generally do not have any clinical significance and are often incidental findings during CT scans of the brain [28]. Dural calcifications, which are a result of calcium salt deposition, should not

Fig. 3 A 17-year-old female with seizures presented with a large calcified mass apparently arising from the left temporal lobe. The calcification pattern (mostly peripheral) and morphology ("popcorn appearance") strongly suggested a vascular lesion such as a cavernous malformation (cavernous angioma) or, less likely, a calcified haemangioma. Upon excision, the mass was later found to be a choroid plexus papilloma, shown in the axial NCECT in soft tissue (a) and bone windows (b) as well as the coronal NCECT reconstruction in soft tissue (c) and bone windows (d)
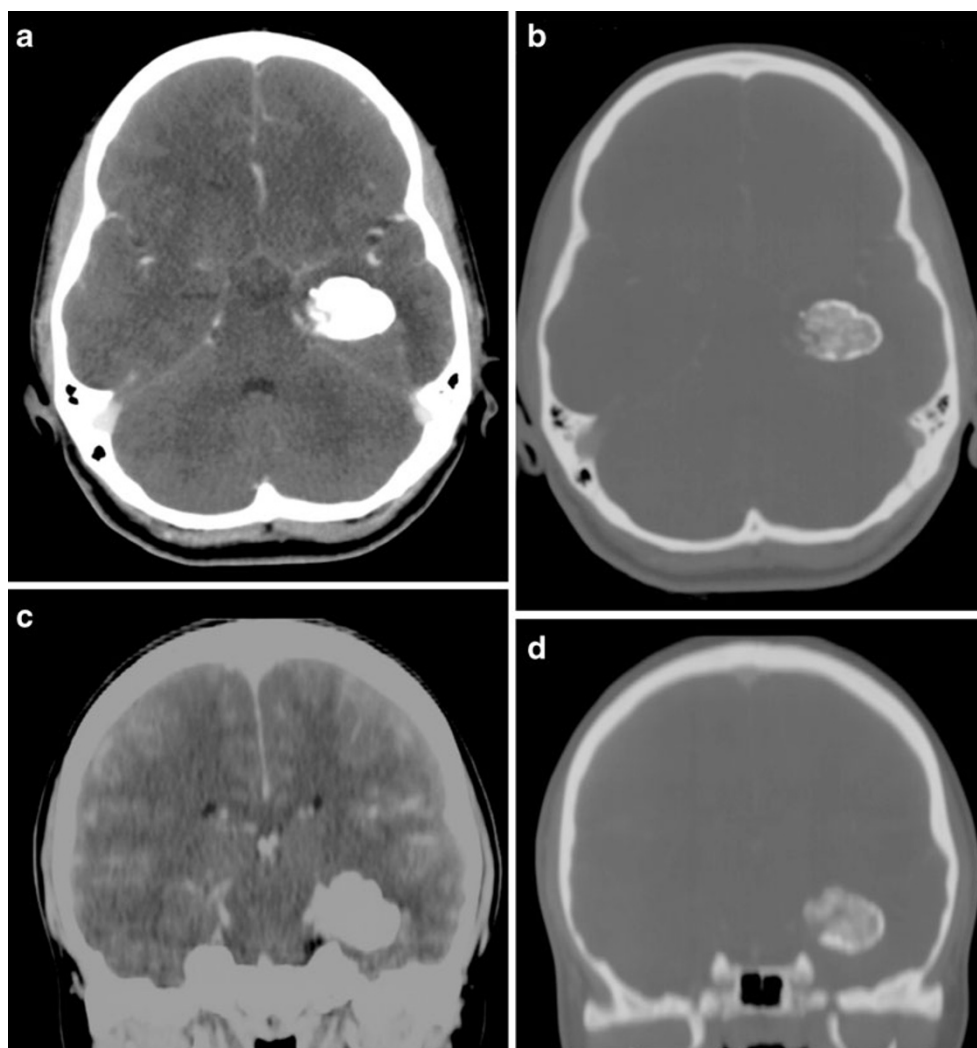
be confused with dural ossifications, which actually involves new bone formation [29]. As the falx is derived from multi-potential mesenchymal cells, they may become osteogenic after exposure to friction, haemorrhage or trauma [29, 30]. Ossifications in the falx cerebri are considered to be a rare phenomenon [9, 31, 32]. However, their incidence may be underestimated as it has been shown that a percentage of previously identified dense calcification on conventional radiography and CT may actually possess an outer shell of cortical bone surrounding an inner core of (fatty) bone marrow using modern high-resolution CT and MR imaging [28, 31, 32]. They can range in size from small islands of bone to large bony structures, can involve the entire falx [29, 33] and can be associated with certain endocrine and congenital disorders [30, 33].

Intra-axial

Intra-axial aetiologies of calcifications potentially resulting in formation of brain stones comprise a broad range of pathologies that can be subdivided into neoplastic, vascular, infectious, congenital and endocrine/metabolic [2, 8, 18, 34].

Neoplastic

Various intracranial tumours are associated with haemorrhage because of a combination of neovascularisation, intralesional arteriovenous shunt formation and rapid enlargement that outgrows the tumour blood supply resulting in necrosis, especially in those with high tumour grades. This condition then promotes disruption of intracellular calcium regulation, resulting in calcium deposition [23, 34, 35]. Tumours most likely to calcify and form brain stones include the slow-growing oligodendrogliomas, in which up to 90 % are known to calcify, resulting in typical dense clump-like calcifications (Fig. 4) [18, 23]. In some cases, the calcification can be punctate, ribbon-like or even peripherally located, and can extend into adjacent brain parenchyma. Low-grade astrocytomas are by far the most common glial neoplasms. However, only a small fraction of these tumours are known to calcify, with patterns ranging from linear, diffuse to punctate. Medulloblastomas are also known to calcify in about 10–20 % of cases [36]. Other brain tumours may calcify to various degrees such as pineal gland tumours, germ cell tumours, primitive neuroectodermal tumours (PNET), dysembryonic neuroectodermal tumours (DNET), gangliogliomas and pilocytic astrocytomas. Although rare, metastasic tumours to the brain can also calcify, often in the setting of treatment [37]. Combined with other imaging features, the presence, but not the pattern of calcification can narrow down the differential diagnosis of intracranial neoplasms. In the same context, the presence of calcification in tumours is not associated with the pathological grade of malignancy, but the eventual development thereof may suggest a favourable response to therapy [5, 38].

Fig. 4 A 68-year-old male with transient right-sided hemiparesis. This preoperative NCECT was done following discovery of suspicious lesions from a recent MRI (not available). Axial (a), sagittal (b) and 3D reconstruction (c) show a calcified lesion (*black arrows*) in the left post central gyrus. The entire lesion was surgically removed and was found to be a glioblastoma with an extensive oligodendroglioma component. Ninety percent of oligodendrogliomas are known to form dense, clump-like calcifications
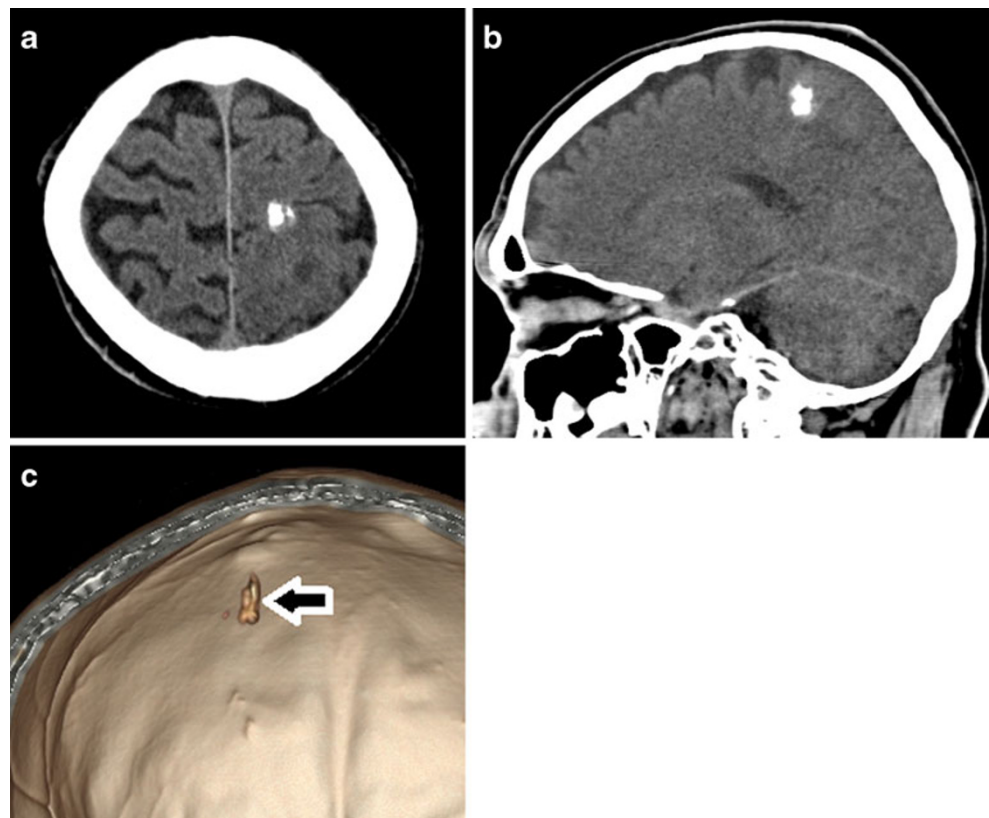
Fig. 5 Dystrophic calcification in a 73-year-old female with a prior history of intracranial bleed who presented with loss of consciousness. The NCECT scan **(a)**, close-up view **(b)** and 3D reconstruction **(c)** showed "brain stones" (*black arrows*) in the middle of the pathological brain regions. There is also an intraventricular drain present (*white arrows*). The subsequent appearance of calcifications in known post-traumatic or post-ischaemic areas of the brain is largely attributed to dystrophic calcification
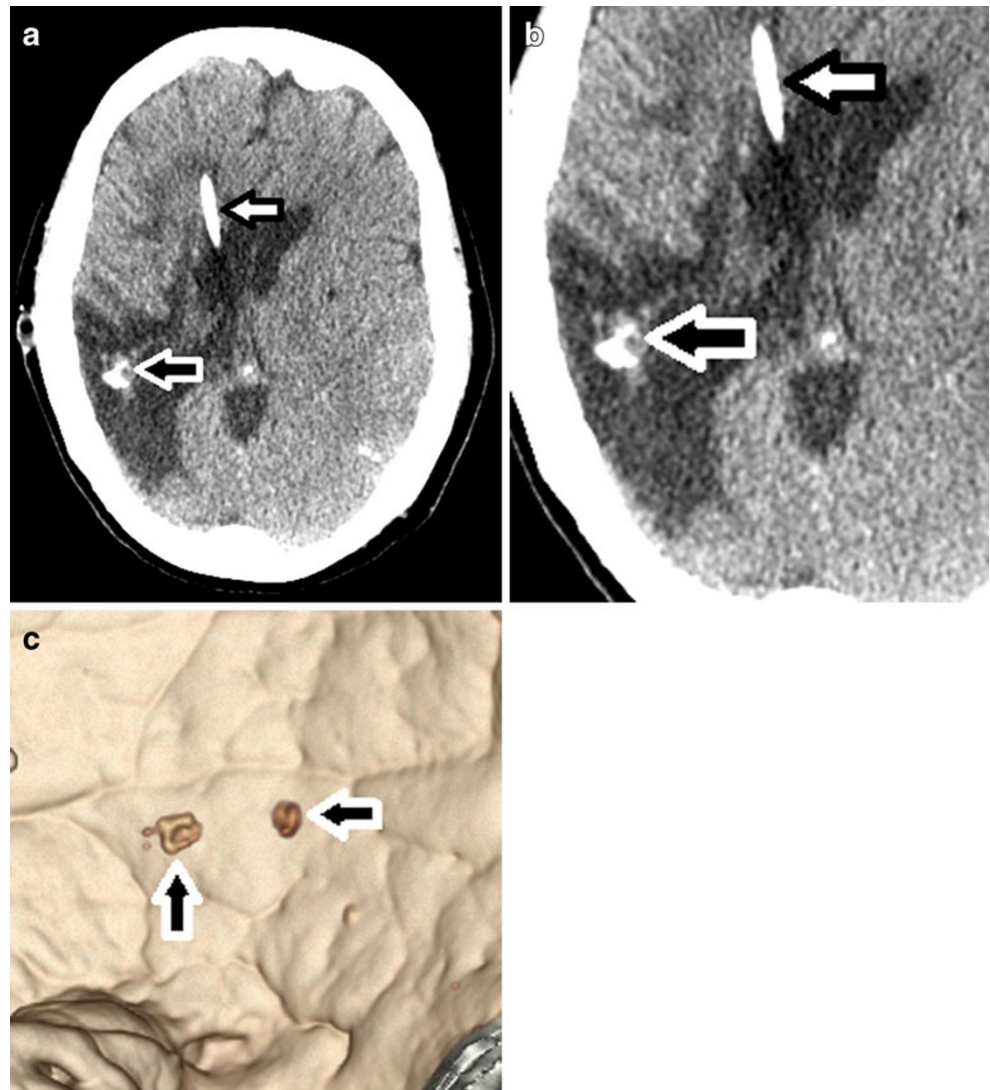

Fig. 6 Axial CT in bone windows **(a)** and 3D reconstruction **(b)** of a 33-year-old male with temporal lobe epilepsy. The large amorphous calcification seen at the base of the left temporal lobe in this NCECT represents a known case of an embolised AV malformation. Calcifications are known to appear in 25–30 % of cases in close conjuction with serpentine vessels or within adjacent brain parenchyma
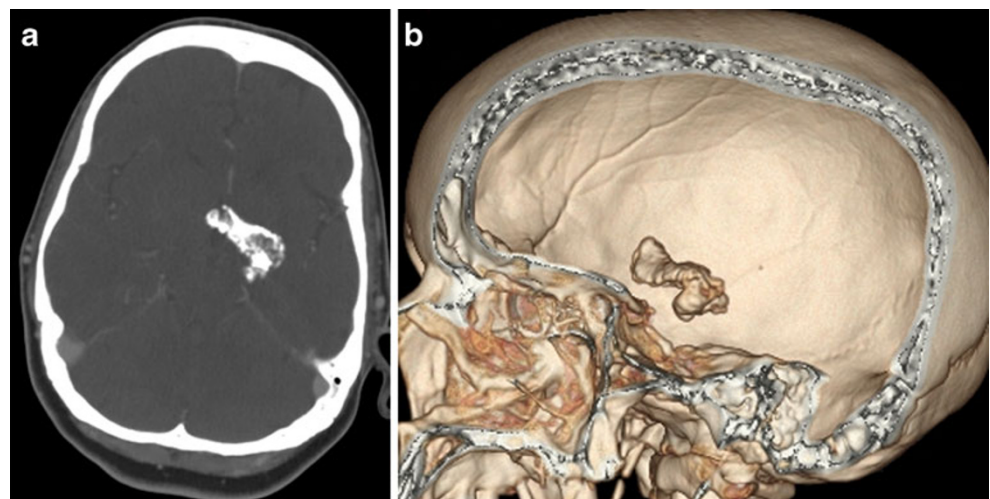
Vascular

Ever since Shafey first reported brain stones due to angiomatous (cavernous) malformations, vascular pathologies have been recognised as important causes of intracranial calcification in general [2]. Other vascular lesions that may result in the formation of intracranial calcifications and brain stones include arteriovenous malformations, dystrophic calcification in chronic infarction, chronic vasculitis and aneurysms (Fig. 5) [39]. In most situations, radiological features as well as location allow differentiation between these entities. Pathophysiological mechanisms leading to cal-

cification include chronic venous ischaemia and the formation of dystrophic calcification as a result of haemorrhage. Calcification in arteriovenous malformations (AVM) is seen in about 25-30 % of cases, mostly along the serpentine vessels, in adjacent brain parenchyma or within the nidus (Fig. 6). The incidence of calcification in cavernous malformations is higher, estimated to be around 40–60 % of cases, often with the typical "popcorn-ball" appearance [40]. Vasculitis leading to microinfarctions and subsequent dystrophic calcifications can be seen in a number of pathologies, such as in systemic lupus erythematosus [41]. The presence of a round or lobulated densely calcified mass in the suprasellar or parasellar

Fig. 7 A 60-year-old male known HIV patient presented with seizures. The solid calcifications on NCECT at the right frontal **(a)** left basal ganglia **(b)** and right parietal areas **(c)** represent sequela following treatment for documented toxoplasmosis. 3D reconstruction **(d)**. In congenital toxoplasmosis, calcifications have been shown to decrease in size or even completely resolve with treatment
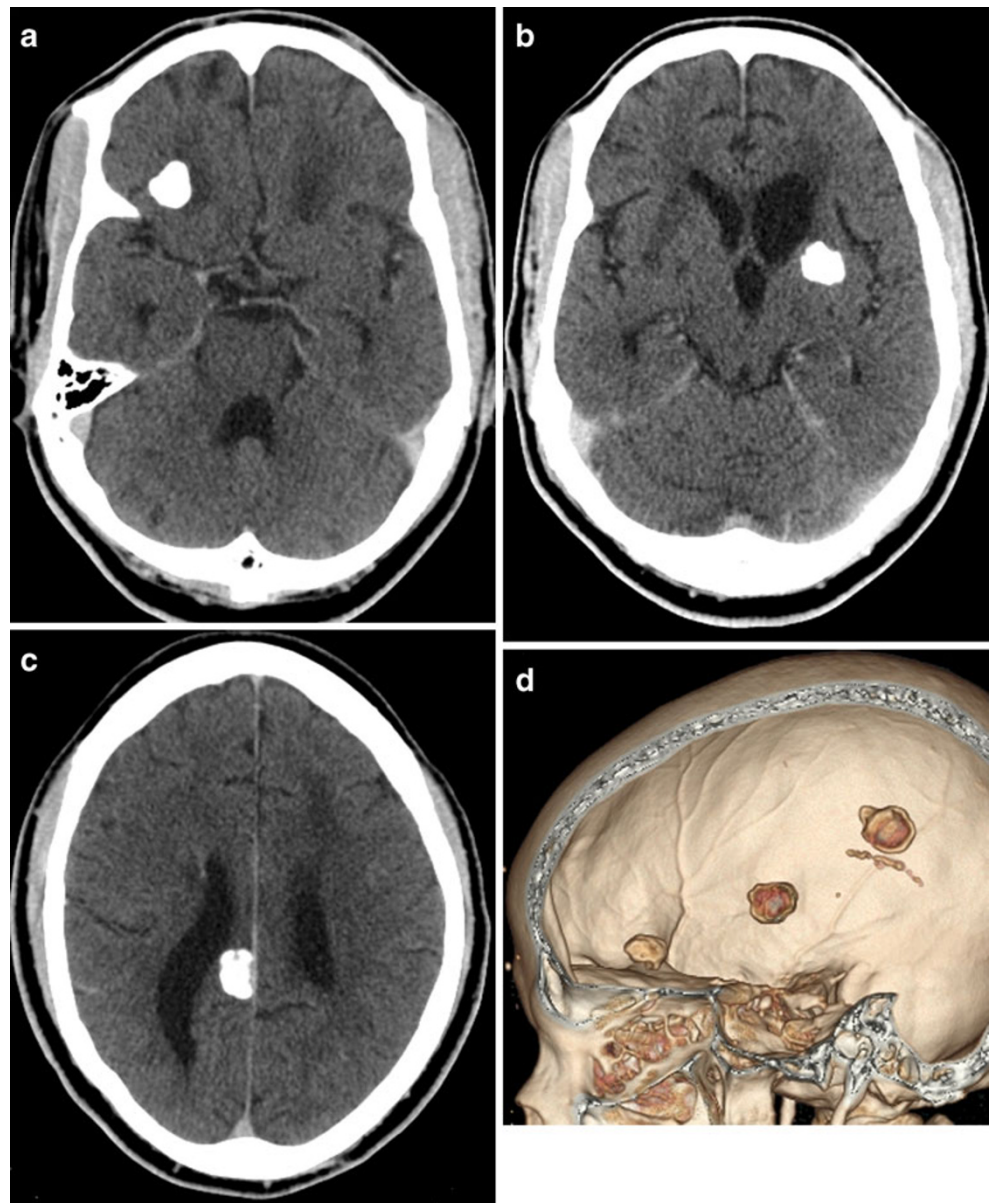
regions is highly suggestive of the presence of a calcified brain aneurysm. The calcification in this case involves the vessel wall, usually in larger aneurysms, and their presence can correlate with the extent of intraluminal thrombosis [42].

Infectious

Central nervous system infections leading to intracranial calcification are most often seen in congenital childhood infections,

particularly the "TORCH" group, which includes toxoplasmosis, rubella, cytomegalovirus and herpes simplex infections. As with vascular pathologies, the mechanism of calcification involved in these infections is dystrophic calcification. With congenital toxoplasmosis and cytomegalovirus infections, these calcifications are usually seen in the basal ganglia and periventricular areas, whereas with herpes simplex infections, the calcifications are located in the thalamic, periventricular and gyral regions [16]. The dense calcifications seen in patients with

Fig. 8 An 8-year-old male with a long complicated medical history was found to have hard-to-miss calcifications on the plain skull x-ray **(a, d)**. The NCECT **(b, e)** and 3D reconstructions **(c, f)** show a large amorphous calcification originating from the basal ganglia, extending into the right lateral ventricle of the patient diagnosed with tuberous sclerosis. Subependymal nodules associated with tuberous sclerosis can calcify with age, typically presenting with globular or sometimes ring-like calcification
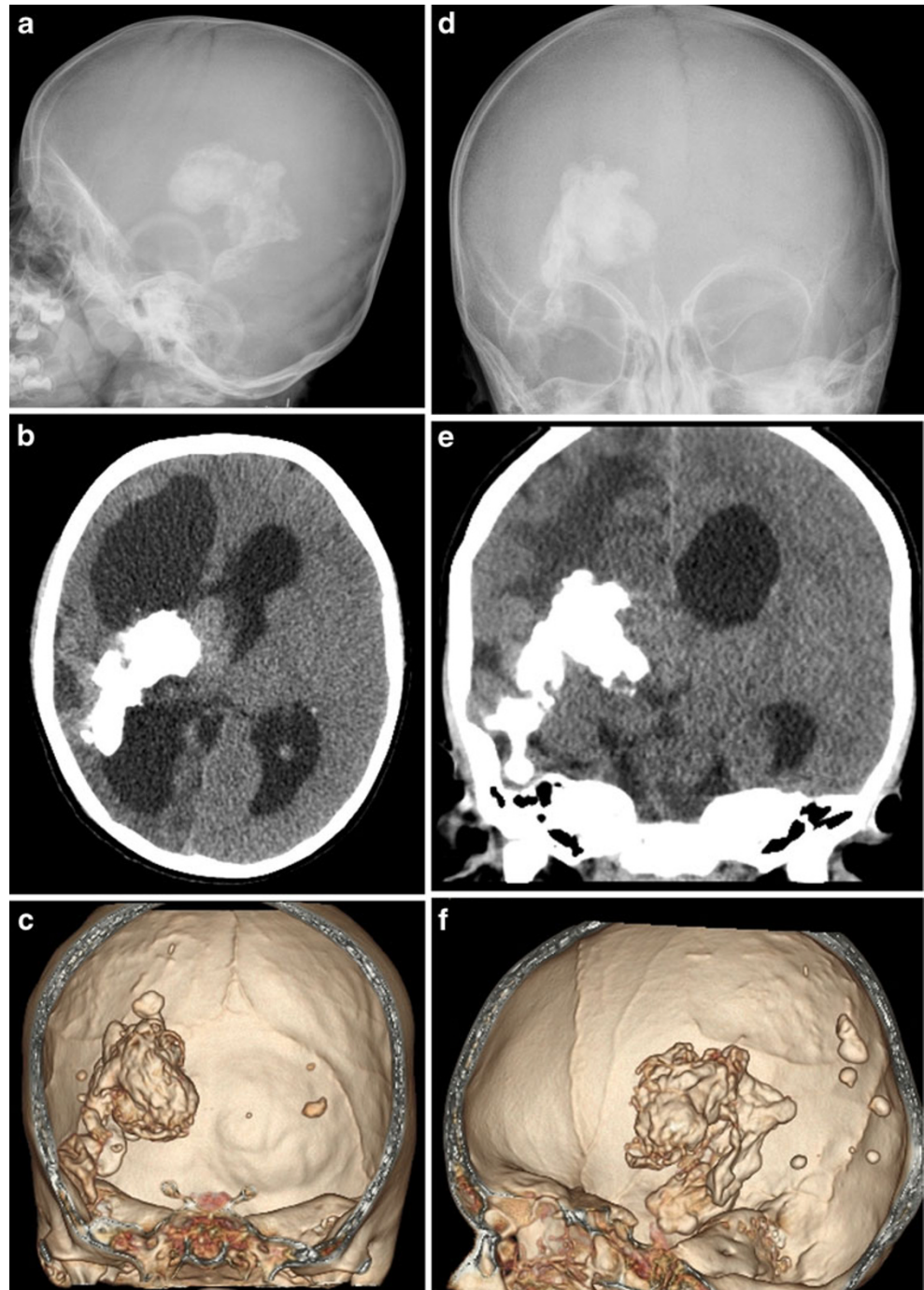
congenital toxoplasmosis can also have a random distribution, with lesion sizes that correlate with the duration of infection. Interestingly, treatment can result in shrinkage and even resolution of these calcifications (Fig. 7) [18, 40, 43]. Congenital rubella infections usually result in calcifications in the basal ganglia, periventricular regions and brainstem. Congenital herpes infection can lead to chronic encephalitis resulting in extensive encephalomalacia and coarse calcification in the remaining brain parenchyma.

Granulomatous infections such as tuberculosis as well as parasitic infections such as neurocysticercosis and cerebral hydatid cyst disease can also result in characteristic intracranial calcifications. Knowledge of a history of travel to endemic areas together with biochemical tests, clinical data and specific imaging findings can often lead to a confident diagnosis. Tuberculosis can result in intracranial calcification in 10–20 % of cases and is usually located supratentorially in adults and infratentorially in children. Calcified tuberculomas can range in size from about a centimetre to several centimetres and can appear as a "broken shell" or a dense centrally located lobulated calcification [6, 10, 44]. Neurocysticercosis is the leading cause of adult-onset seizures in less developed

countries, as shown by epidemiological studies, with no consensus on whether the intracranial calcifications are epileptogenic or not [45–47]. The calcifications represent dead larvae and have the appearance of a dense calcified cyst that sometimes contains a dense eccentric nodule. Lastly, cerebral hydatid disease is an extremely rare cause of intracranial calcification. The lesions are usually single, septated or multilocular and represent the dead parasite [48–50].

Congenital

Congenital conditions known to cause intracranial calcifications and brain stones include Sturge-Weber syndrome, tuberous sclerosis as well as lipomas and neurofibromatosis. Sturge-Weber syndrome calcifications are usually described as "double contoured curvilinear" or "tram-track", closely approximating the gyral patterns in the parietal and occipital regions. They can be bilateral in 15 % to 20 % of cases [18, 51]. On the other hand, the calcifications seen in the tubers associated with tuberous sclerosis can resemble the gyral pattern seen in Sturge-Weber syndrome (Fig. 8). These calcifications can occur in up to more than 50 % of cases [52, 53]. Subependymal nodules

Fig. 9 A 35-year-old female with a history of multiple shunt revisions was found to have symmetric calcifications in the cerebellum and basal ganglia on CT (a, b) and 3D reconstruction (c). Although not confirmed in this patient, the presence of symmetric calcifications in these regions in young patients is generally thought to be related to a possible diagnosis of Fahr syndrome or an endocrine disorder
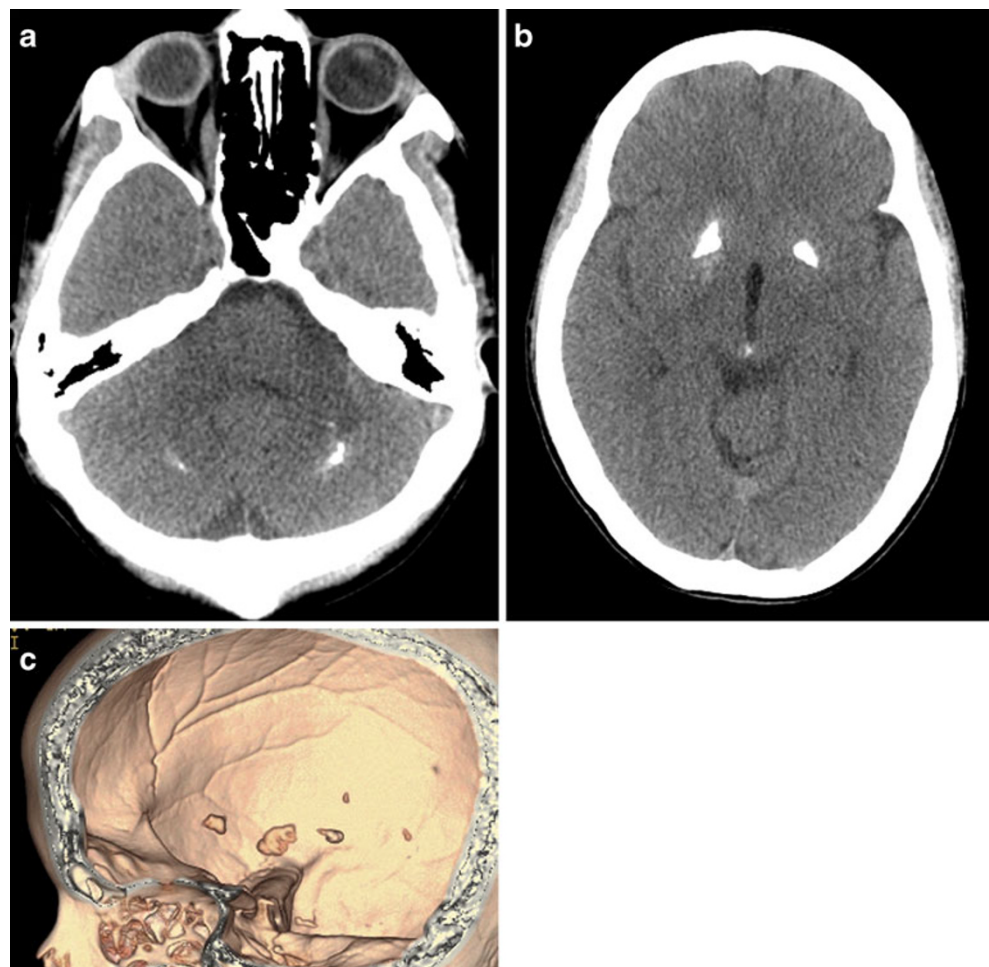
also associated with tuberous sclerosis can calcify with age, often presenting with globular or sometimes ring-like calcification. Neurofibromatosis type 2 can also present with calcifying subependymal nodules, although the calcifying lesions that are more commonly associated with them are the disease-related tumours such as meningiomas or non-tumoral lesions located in the choroid plexus, cerebral hemispheres or cerebellar hemispheres [54]. Lipomas are considered to be rare congenital benign malformations resulting from developmental aberrations involving the meninx primitiva. They are usually located in the midline, in close proximity to the corpus callosum, and are associated with other brain malformations in over 50 % of cases [55, 56]. Lipoma-associated calcifications are often seen particularly in interhemispheric locations as curvilinear along the capsule or as centrally located nodules. Up to half of the lipomas located in the suprasellar or interpeduncular areas are known to ossify. In one series, it was noted that no correlation exists between the presence of calcification and the location of the lipomatous lesions [56].

Endocrine/metabolic

Intracranial calcifications and brain stones due to endocrine and metabolic causes are generally the result of abnormalities in calcium homeostasis and characteristically symmetrically affect the basal ganglia, thalamus and cerebellum. Fahr's syndrome is an exception in that there are usually no accompanying abnormalities involving serum calcium levels or hormone levels. The basal ganglia, white matter and cerebellar calcifications in Fahr's syndrome may be minimal or massive, and have been demonstrated in pathological studies to be calcium deposits in capillary walls, small arterioles and veins including perivascular spaces, with studies showing a correlation between the amount of calcification and symptom severity (Fig. 9) [14, 57]. On the other hand, diabetes mellitus has also been shown to result in bilateral calcifications in the basal ganglia and gray-white matter interfaces presumably due to hypernatremia-induced endothelial cell damage with a relative lack of neurological symptoms [58]. Hypoparathyroidism, pseudohypoparathyroidism and hyperparathyroidism affect calcium homeostasis because of parathyroid gland dysfunction and abnormal parathyroid hormone levels, resulting in calcifications in the basal ganglia, dentate nucleus and subcortical areas with clinical symptoms ranging from seizures to tremors and parkinsonism [59]. Calcium chelation using bisphosphonates is currently being investigated as possible treatment for symmetric calcification-related neurological symptoms with promising initial results [57, 60].

Conclusion

Brain stones are more common than previously thought. Modern imaging modalities allow their identification in a wide range of clinical entities. Computed tomography is the mainstay in identifying and characterising brain stones while certain MRI sequences (gradient echo T2* and susceptibility-weighted imaging) are considered adjunctive. Brain stones may be extra- or intra-axial in location. Extra-axial brain stones comprise tumours and exaggerated physiological calcifications. Intra-axial calcifications can be classified under neoplastic, vascular, infectious, congenital and endocrine/metabolic aetiologies. Imaging findings combined with essential clinical information can help in narrowing the differential diagnosis, determining disease state or evaluating effect of therapy.

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