

Intracranial calcifications on CT

Yılmaz Kiroğlu, Cem Çallı, Nevzat Karabulut, Çağatay Öncel

ABSTRACT

Computed tomography (CT) is very sensitive for detection and localization of intracranial calcifications. We reviewed in this pictorial essay the diseases associated with intracranial calcifications and emphasized the utility of CT for the differential diagnosis.

Key words: • calcification, physiologic • calcification, pathologic • computed tomography

Intracranial calcifications can be classified mainly into 6 groups based on their etiopathogenesis: age-related and physiologic, congenital, infectious, endocrine and metabolic, vascular, and neoplastic (Table).

Age-related physiologic and neurodegenerative calcifications

Intracranial physiologic calcifications are unaccompanied by any evidence of disease and have no demonstrable pathological cause (1). The most common sites include the pineal gland, habenula, choroid plexus, basal ganglia, falx, tentorium, petroclinoid ligaments and sagittal sinus. Calcification of the pineal gland is seen in two-thirds of the adult population and increases with age. Pineal calcification over 1 cm in diameter or under 9 years of age may be suggestive of a neoplasm (2). Habenula has a central role in the regulation of the limbic system and is often calcified with a curvilinear pattern a few millimeters anterior to the pineal body in 15 % of the adult population. Choroid plexus calcification is very common finding, usually in the atrial portions of the lateral ventricles (Fig. 1). Calcification in the third or fourth ventricle or in patients less than 9 years of age is uncommon. Basal ganglia calcifications are usually idiopathic incidental findings that have a 0.3–1.5% incidence and increases with age. They usually demonstrate a faint punctuate or a coarse conglomerated symmetrical calcification pattern (1) (Fig. 2). Calcifications of the falx, dura mater or tentorium cerebelli occur in about 10% of elderly population. Falcian calcifications usually have a characteristic appearance pattern as dense and flat plaques and are usually seen in the midline of the cerebrum (2) (Fig. 3). Dural and tentorial calcifications are usually seen in a laminar pattern and can occur anywhere within the cranium (Fig. 4). Petroclinoid ligament and sagittal sinus calcifications are common age-related degeneration sites and usually have laminar or mildly nodular patterns (1, 2) (Fig. 5).

Congenital calcifications

This condition is frequently seen in Sturge-Weber syndrome (SWS), tuberous sclerosis (TS) and intracranial lipoma, but rarely in neurofibromatosis (NF), Cockayne (CS) and Gorlin syndromes (GS). SWS is a rare disorder consisting of a port-wine nevus in the distribution of the ophthalmic branch of the trigeminal nerve and nervous system malformations. The syndrome results from malformation of the cerebral vasculature located within the pia mater. This malformation leads to venous hypertension and subsequent hypoperfusion of the underlying cortex, causing chronic cerebral ischemia, parenchymal atrophy, enlargement of the ipsilateral choroid plexus and calcification. Calcification in SWS demonstrates a characteristic linear cortical pattern (3) (Fig. 6). TS is an autosomal dominant disorder characterized by mental retardation, epilepsy and adenoma sebaceum. Intracranial lesions in TS consist of subependymal hamarto-

From the Departments of Radiology (Y.K. ✉ drkiroglu@yahoo.com, N.K.), and Neurology (Ç.Ö.), Pamukkale University Faculty of Medicine, Denizli, Turkey; and the Department of Radiology (C.Ç.), Ege University Faculty of Medicine, Izmir, Turkey.

Received 22 January 2009; revision requested 25 July 2009; revision received 27 July 2009; accepted 28 July 2009.

Published online 18 December 2009
DOI 10.4261/1305-3825.DIR.2626-09.1

Table. Intracranial calcifications

Age-related and physiologic	Pineal gland, habenula, choroid plexus, falx cerebri, tentorium cerebelli, dura mater, petroclinoid ligament, sagittal sinus
Congenital	Sturge-Weber syndrome, tuberous sclerosis, neurofibromatosis, lipoma, Cockayne syndrome, Gorlin syndrome
Infectious	TORCH diseases, granulomatous infections, chronic viral encephalitis
Endocrine and metabolic	Fahr disease, hypothyroidism, hypoparathyroidism, hyperparathyroidism, pseudohypoparathyroidism, post-thyroidectomy
Vascular	Primary atherosclerosis, cavernous malformation, arteriovenous malformation, aneurysms, dystrophic in chronic infarction and chronic vasculitis
Neoplastic	Oligodendroglioma, craniopharyngioma, germ cell neoplasms, neurocytoma, primitive neuroectodermal tumor (PNET), ependymoma, ganglioglioma, dysembryonic neuroectodermal tumor (DNET), meningioma, choroid plexus papilloma, medulloblastoma, low grade astrocytoma, pilocytic astrocytoma, pinealoma, pinealoblastoma, schwannoma, dermoid, epidermoid, calcified metastases (osteogenic sarcoma, mucinous adenocarcinoma)



Figure 1. Bilateral choroid plexus (arrowheads), pineal (arrow) and habenular calcifications (dashed arrow) on axial CT.

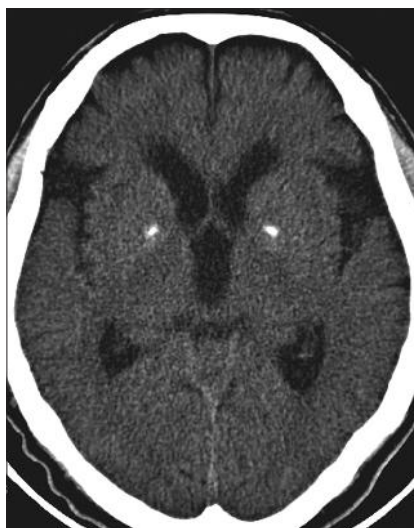


Figure 2. Calcifications in bilateral basal ganglia.

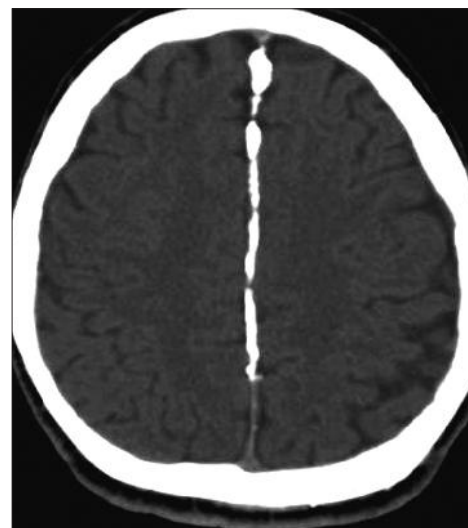


Figure 3. Calcified falx cerebri on the anteroposterior axis.



Figure 4. Calcifications along the tentorium cerebelli.



Figure 5. Calcified bilateral petroclinoid ligaments (arrows).



Figure 6. Characteristic gyral calcifications in Sturge-Weber syndrome associated with atrophy of the right frontal and temporal lobes.

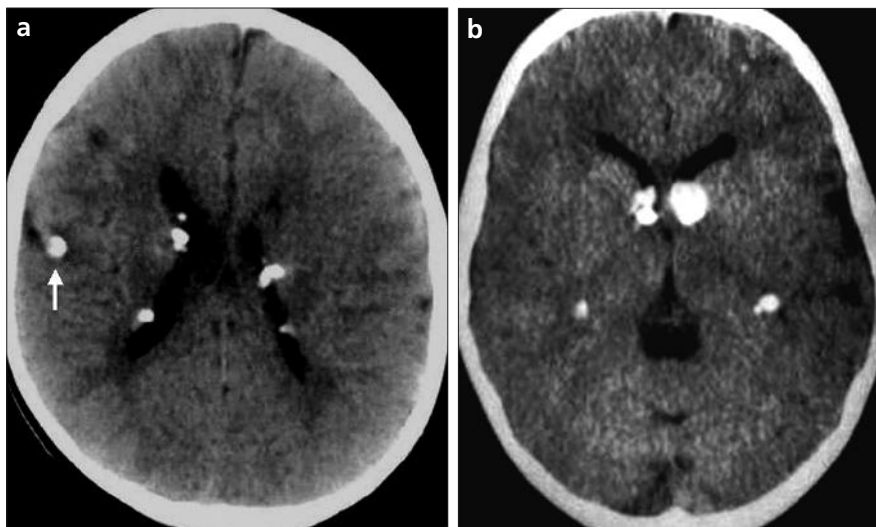


Figure 7. a, b. Periventricular calcifications of subependymal nodules in two different patients with tuberous sclerosis. Subcortical calcified tuber (*arrow*) in the right parietal lobe (**a**).

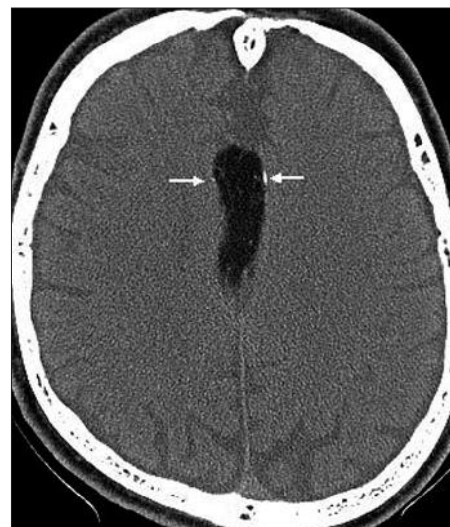


Figure 8. Intracranial lipoma in the midline of cerebrum with thin focal calcifications (*arrows*).

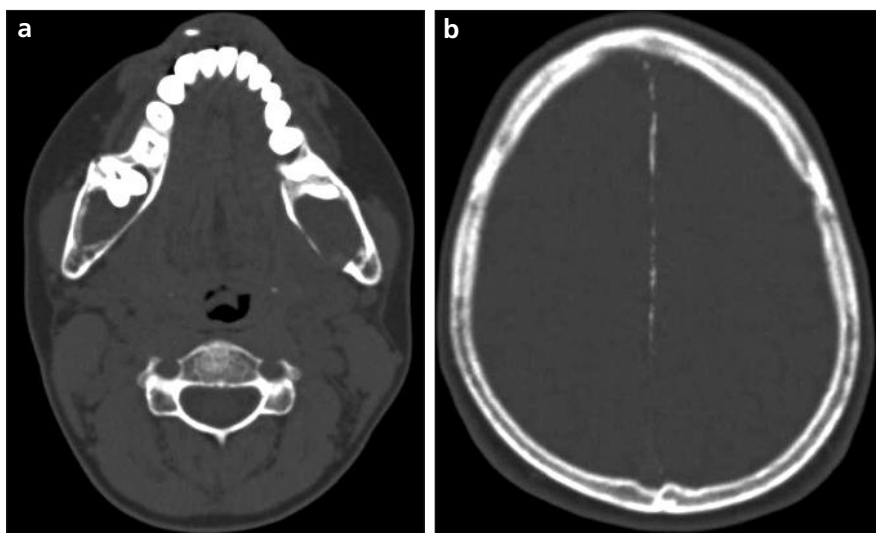


Figure 9. a, b. Dentigerous keratocysts in both sides of the jaw (**a**) and midline falx calcification (**b**) in Gorlin's syndrome.

mas, subcortical tubers, giant cell tumors, and white matter lesions. Calcifications of the subependymal nodules are pathognomonic and commonly located along the caudothalamic groove and atrium. Subcortical tubers are calcified more often in elderly patients (4) (Fig. 7). NF1 is an autosomal dominant disorder characterized by gliomas, dysplasias and hamartomas. Intracranial hamartomas in NF1 are frequently seen in the location of globus pallidus, but they rarely calcify (5). Intracranial lipomas are benign congenital malformations, which progress asymptotically. Approximately 80–90% of intracranial lipomas are located at or

near the midline. A curvilinear or focal pattern of calcifications is frequently seen in their capsules and surrounding parenchyma (6) (Fig. 8). CS is an autosomal recessive disorder, which shows progressive encephalopathy including intracranial calcifications and white matter lesions. Calcifications are commonly in coarse pattern and frequently involve the subcortical white matter, basal ganglia and dentate nuclei. GS is an autosomal dominant tumor predisposition syndrome that consists of multiple basal cell carcinomas of the skin, odontogenic keratocyst of the jaw, various skeletal abnormalities, and lamellar falx calcification (7) (Fig. 9).

Infectious calcifications

A large number of infectious agents may involve the fetal central nervous system in utero. The most common of them are "TORCH" agents including toxoplasmosis, rubella, *Cytomegalovirus* (CMV) and *Herpes simplex virus* (HSV). Congenital HSV infections demonstrate extensive cerebral destruction, multicystic encephalomacia and scattered calcifications (8). Congenital toxoplasmosis is commonly associated with hydrocephalus and randomly nodular calcifications in periventricular, basal ganglia and cerebral cortical areas (Fig. 10). Congenital rubella is usually associated with meningitis, ventriculitis and subsequent ventriculomegaly. Calcifications are commonly located in the periventricular white matter, basal ganglia, and brain stem (9). CMV is the most common cause of congenital infections and frequently associated with microcephaly, chorioretinitis, and intracranial calcifications. Calcifications in congenital CMV infections are commonly seen in the periventricular and subependymal sites (8) (Fig. 11).

Chronic phase of viral encephalitis may feature widespread encephalomalacia and calcification in the residual parenchyma as a rare infectious cause of intracranial calcifications (9).

Calcification in intracranial tuberculosis and fungal opportunistic granulomatous infections is also quite rare. A "target sign" representing a central nidus of calcification surrounded by a ring of enhance-



Figure 10. Axial unenhanced CT shows extensive bilateral thalamic and periventricular calcifications associated with dilated ventricular asymmetry in congenital toxoplasmosis.

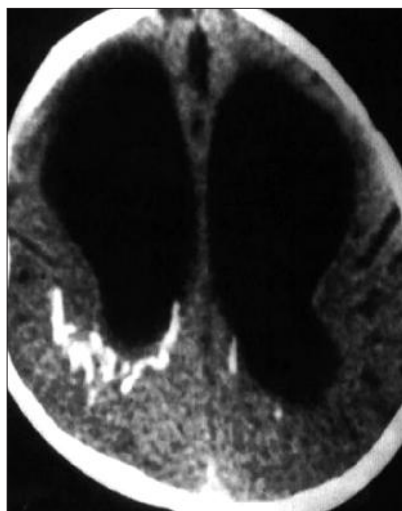


Figure 11. Extensive periventricular and subependymal calcifications with marked hydrocephaly in congenital *Cytomegalovirus* infection.

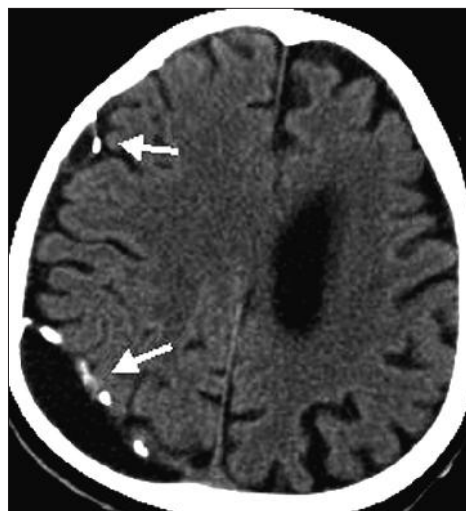


Figure 12. Unenhanced CT image shows low-density epidural effusions with calcified walls (arrows) adjacent to the right parietal lobe.

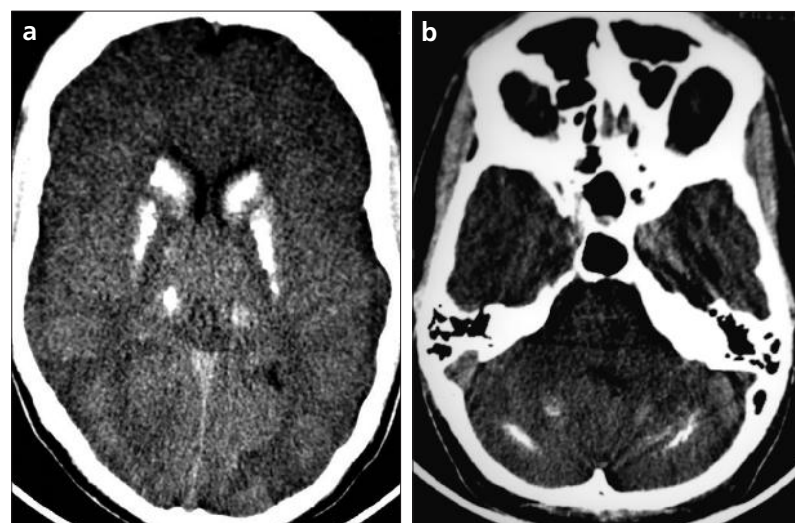


Figure 13. a, b. Dense calcifications in the bilateral corpora striatum and thalami (a), and in the cerebellar parenchyma (b) associated with Fahr disease.

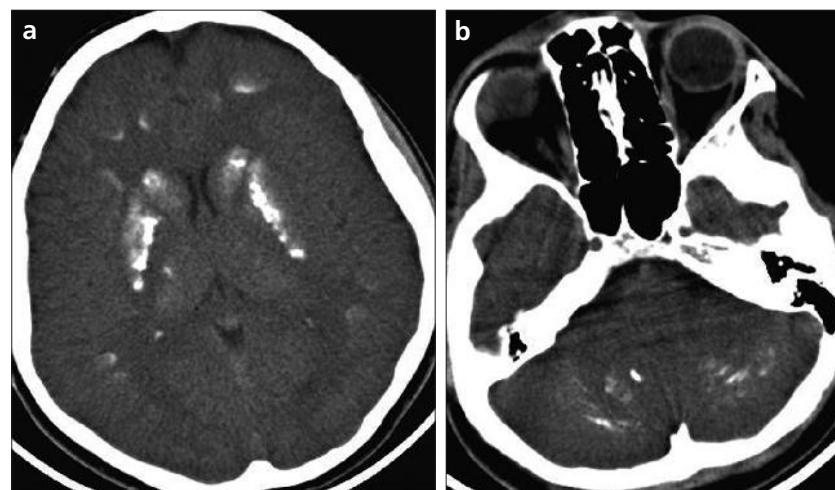


Figure 14. a, b. Extensive calcifications in the bilateral corpora striatum and peripheral cerebral subcortical areas (a), dentate nuclei and cerebellar parenchyma (b) associated with hypoparathyroidism.

ment is strongly suggestive of a tuberculoma (8, 10). Although subdural and epidural empyemas are common complications of intracranial infections, they may rarely demonstrate calcified meninges and low density effusions in the chronic phase of the illness (Fig. 12).

Calcifications related with hormonal and metabolic disorders

Fahr disease is a rare degenerative neurological disorder characterized by extensive bilateral basal ganglia calcifications that can lead to progressive dystonia, parkinsonism and neuropsychiatric manifestations. It is associated with defective iron transport resulting in tissue damage with extensive calcification. Metastatic deposition is frequently located in the bilateral basal ganglia, dentate nuclei, cerebral white matter, and internal capsule (11) (Fig. 13). Fahr patients are usually asymptomatic in the first two decades of life, despite the presence of intracranial calcifications.

Function of the parathyroid hormone is primarily maintaining the plasma calcium levels. Hormonal disturbance of the parathyroid glands including hypoparathyroidism, hyperparathyroidism and pseudohypoparathyroidism may lead to intracranial calcifications. Calcium accumulation is demonstrated primarily in the bilateral basal ganglia, dentate nuclei, and peripheral subcortical white matter sites (12) (Fig. 14).

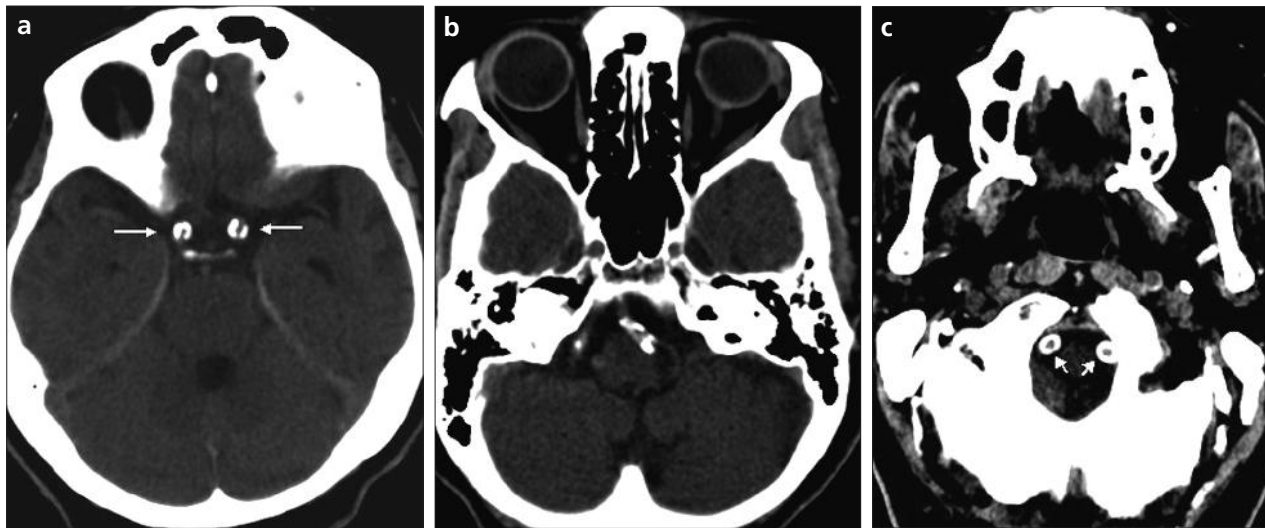


Figure 15. a–c. Primary atherosclerotic calcified plaques are demonstrated on the walls of the internal carotid (a) and bilateral vertebral arteries (arrows) (b, c).



Figure 16. Unenhanced axial CT scan shows a giant aneurysm with a rim-like calcified wall arising from the anterior communicating artery (arrow). Calcification in the right middle cerebral artery walls (arrowhead), surrounding edema, and hydrocephalus with intraventricular hemorrhage (dashed arrow) are also seen.

Vascular calcifications

Calcification of the intracranial arteries associated with primary atherosclerosis is more frequent in elderly people. The highest prevalence of intracranial artery calcification is seen in the internal carotid artery (60%), followed by the vertebral artery (20%), middle cerebral artery (5%), and basilar artery (5%) (13) (Fig. 15).

Other causes of vascular calcifications include aneurysm, arteriovenous malformation (AVM) and cavernous malformation. Although patent aneurysms may contain mural calcification, partially or completely thrombosed aneurysms commonly have calcifications (14) (Fig. 16). AVMs may contain dystrophic calcifications along the serpentine vessels and within the adjacent parenchyma with a prevalence of 25–30% (13) (Fig. 17). Cavernous malformation is a benign vascular hamartoma that is frequently calcified in a "popcorn-ball" fashion (15) (Fig. 18).

Neoplastic calcifications

The most common intracranial neoplasms associated with calcifications (16) are oligodendroglioma (70–90%), craniopharyngioma (50–80%), germ cell neoplasms (dysgerminoma, seminoma, teratoma, choriocarcinoma; 60–80%), pineal neoplasms (pineoblastoma, pineocytoma; 60–80%), central neurocytoma (50–70%), primitive neuroectodermal tumor (PNET) (50–70%), ependymoma (50%), ganglioglioma (35–50%) (Fig. 19),

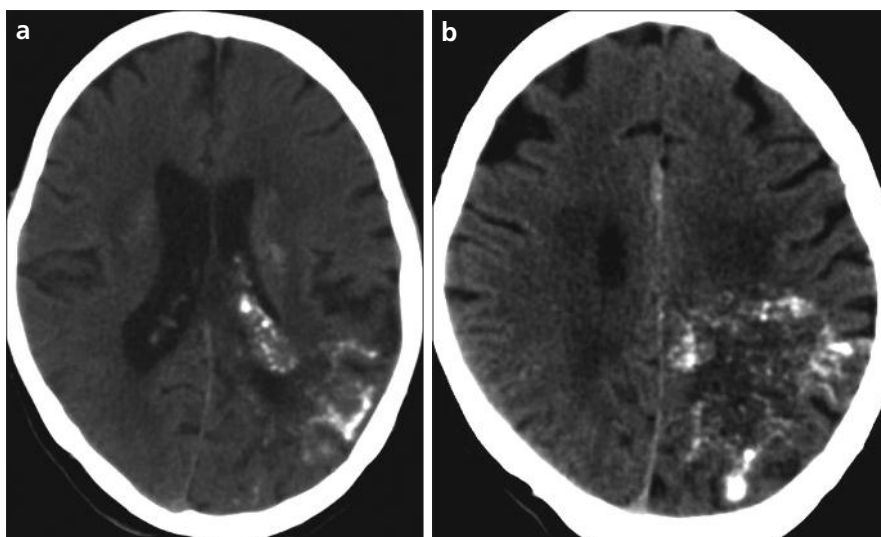


Figure 17. a, b. Extensive serpentine calcifications in the parietooccipital cerebral parenchyma and left choroid plexus extend from the deep periventricular region towards the cortex with accompanying atrophy in a large arteriovenous malformation.

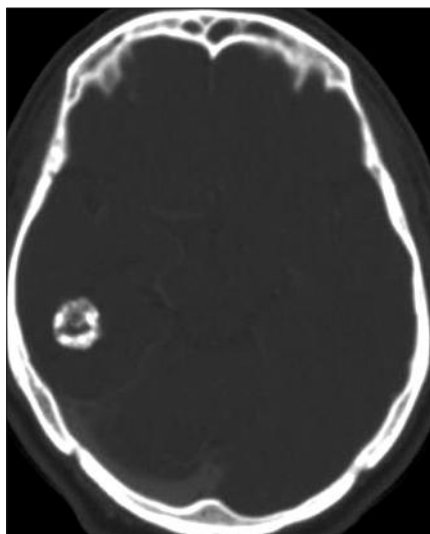


Figure 18. Cavernous malformation in a "popcorn-ball" appearance with a thick and dense rim calcification pattern in the right parietal lobe.

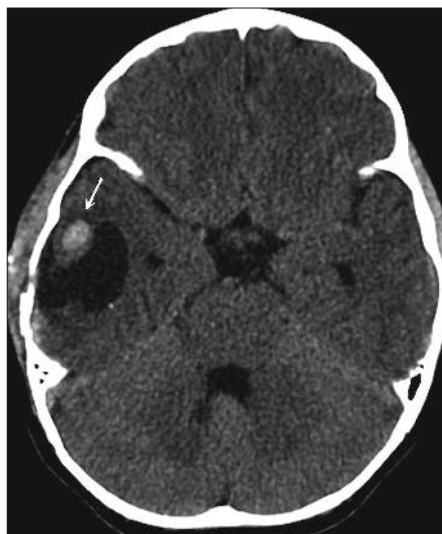


Figure 19. Ganglioglioma in the right temporal lobe as a partially cystic, cortex-based mass with nodular calcification (arrow).

dysembryonic neuroectodermal tumor (DNET) (20–36%), meningioma (20–25%), choroid plexus papilloma (25%), medulloblastoma (20%), low grade astrocytoma (20%), and pilocytic astrocytoma (10%). Calcifications are rarely seen in schwannomas, and dermoid and epidermoid tumors.

Evaluation of neoplastic calcifications along with the patient age, tumor localization and calcification pattern may contribute to the radiologic differential diagnosis of intracranial neoplasms. Oligodendrogliomas are usually located in the frontal lobe and are calcified in a nodular and clumped pattern (Fig. 20). Craniopharyngioma is characterized by a suprasellar mass that is calcified in an amorphous and lobulated pattern (Fig. 21). A dural based tumor in an elderly patient, with



Figure 20. a, b. Oligodendrogliomas in two different cases. First is in the right frontal lobe and represents a partially calcified cortex-based mass (a) while the second is located in the left periventricular area (b). Calcifications of both masses are in a nodular and clumped pattern.

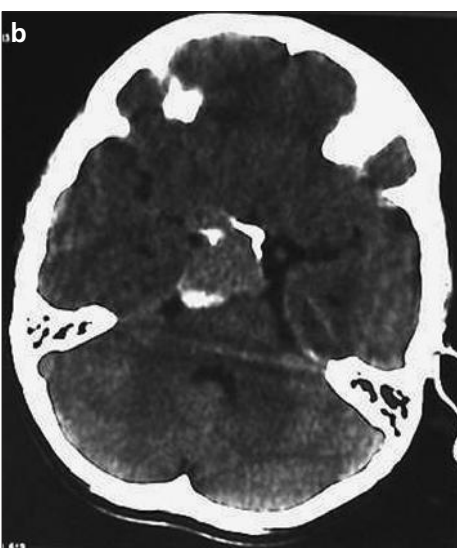


Figure 21. a, b. Craniopharyngiomas in two different patients in sellar and suprasellar regions with amorphous calcifications.

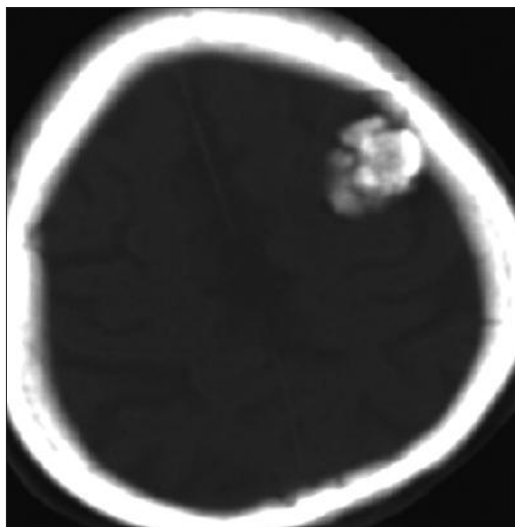


Figure 22. Bone-window unenhanced CT image demonstrates a meningeoma as a dural-based cortical mass with dense globular calcification in the left frontal lobe.



Figure 23. Teratoma in a newborn patient in the right cerebral hemisphere with coarse anterior calcifications.

a variable pattern of calcifications including diffuse, focal, sandlike, sunburst, and globular, strongly suggests a meningioma (17) (Fig. 22). A cortex-based cystic mass with a mural calcific nodule in a young patient may suggest a ganglioglioma. A heterogenous, calcified mass in the pineal region in a young patient may suggest a germ cell neoplasia (Fig. 23). Calcifications are fairly rare in metastases with the exceptions of osteogenic sarcoma and mucinous adenocarcinoma. Metastases treated by radiotherapy or chemotherapy may also develop calcification (2).

References

1. Kieffer SA, Gold LH. Intracranial physiologic calcifications. *Semin Roentgenol* 1974; 2:151-162.
2. Deepak S, Jayakumar B. Intracranial calcifications. *J Assoc Physicians India* 2005; 53:948.
3. Wasenco JJ, Rosenbloom SA, Duchesneau PM, Lanzieri CF, Weinstein MA. The Sturge-Weber syndrome: comparison of MR and CT characteristic. *AJNR Am J Neuroradiol* 1990; 1:131-134.
4. Lin DD, Barker PB. Neuroimaging of phakomatoses. *Semin Pediatr Neurol* 2006; 1:48-62.
5. Arts WF, Van Dongen KJ. Intracranial calcified deposits in neurofibromatosis. *J Neurol Neurosurg Psychiatry* 1986; 11:1317-1320.
6. Yilmaz N, Unal O, Kiyimaz N, Yilmaz C, Etlik O. Intracranial lipomas. *Clin Neurol Neurosurg* 2006; 4:363-368.
7. Kantarci M, Ertas U, Sutbeyaz Y, Karasen RM, Onbas O. Gorlin's syndrome with a thin corpus callosum and a third ventricular cyst. *Neuroradiology* 2003; 6:390-392.
8. Sze G, Lee SH. Infectious disease. In: Lee SH, Rao KCVG, Zimmerman RA, eds. *Cranial MRI and CT*. 4th ed. New York: Mc Graw-Hill, 1999; 453-517.
9. Hedlung GL, Boyer RS. Neuroimaging of postnatal pediatric central nervous system infections. *Semin Pediatr Neurol* 1999; 4:299-317.
10. Alkan A, Parlak M, Baysal T, Sigirci A, Kutlu R, Altinok T. En-plaque tuberculomas of tentorium in a pregnant women: follow-up with MRI. *Eur Radiol* 2003; 5:1190-1193.
11. Acou M, Vanslembrouck J, Deblaere K, Bauters W, Achten E. Fahr disease. *JBR-BTR* 2008; 1:19.
12. You JS, Kim HJ, Chung SP, Park S. Intracranial bilateral symmetric calcification in hypoparathyroidism. *Emerg Med J* 2008; 3:162.
13. Chen XY, Lam WW, Ng HK, Fan YH, Wong KS. The frequency and determinants of calcification in intracranial arteries in Chinese patients who underwent computed tomography examinations. *Cerebrovasc Dis* 2006; 21:91-97.
14. Tomandl BF, Köstner NC, Schempershofe M, Huk WJ, Strauss C, Anker L, Hastreiter P. CT angiography of intracranial aneurysms: a focus on post processing. *Radiographics* 2004; 3:637-655.
15. Metoki T, Mugikura S, Higano S, Ezura M, Matsumoto Y, Hirayama K, Takahashi S. Subcortical calcification on CT in dural arteriovenous fistula with cortical venous reflux. *AJNR Am J Neuroradiol* 2006; 5:1076-1078.
16. Rabinov JD. Diagnostic imaging of angiographically occult vascular malformations. *Neurosurg Clin N Am* 1999; 3:419-432.
17. Rees J, Lee SH, Smirniotopoulos JG. Primary brain tumors in adults. In: Lee SH, Rao KCVG, Zimmerman RA, eds. *Cranial MRI and CT*. 4th ed. New York: Mc Graw-Hill, 1999; 261-341.