Computed tomography patterns of intracranial calcifications: always easy to define?

Poster No.: C-1556  
Congress: ECR 2013  
Type: Educational Exhibit  
Authors: M. M. Serbanescu¹, G. Popa², I. G. Lupescu²; ¹Bucuresti/RO, ²Bucharest/RO  
Keywords: Calcifications / Calculi, Education, CT, Neuroradiology brain, Head and neck  
DOI: 10.1594/ecr2013/C-1556

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

1. To classify and outline the spectrum of entities that generate intracranial calcification.
2. To illustrate the CT appearance of intracranial calcification and underlay some typical aspects and sites that radiologists have to be familiar.

Background

INTRODUCTION
CT exam is the gold standard in detecting intracranial calcification.[1] A Hounsfield Unit (HU) of 100 or more in a soft tissue, on a non-enhanced CT, signifies a calcium-containing structure or a metallic object.[1]

Intracranial calcification may occurs physiologically[2,3] but they also indicate some central nervous system disorders such as:

- vascular,
- infectious,
- inflammator,
- tumoral,
- metabolic (endocrine), toxic cerebral insults and
- some congenital pathology.

ETIOLOGY

1. Physiologic. Associated with ageing (depending on location), physiologic calcification are extremely common incidental discovered, with almost no clinically significance.[2,3]

2. Posttraumatic. They are chronic sequels after ischemic or parenchymal hemorrhage from infarcts, trauma or surgery.[3] In post radiation or chemotherapy calcification appear long time after treatment and are much more common in young children[4].

3. Congenital disorders (phakomatoses) - are hereditary disorders with multiple central nervous system and cutaneous abnormalities. Calcifications are commonly reported in tuberous sclerosis and Sturge-Weber syndrome, but can also appear in basal-cell nevus syndrome and neurofibromatosis type I and II.[3]

Tuberous sclerosis is a multisystemic autosomal-dominant disorder with typical manifestation of the triad: seizures, mental retardation, and facial angiofibroma.[6]
Neurofibromatosis type 1 is a multisystem neurocutaneous disorder associated with increased incidence of different tumors, such as optic nerve glioma and plexiform neurofibroma.[2,3]

Neurofibromatosis type II (MISME - multiple intracranial schwannomas, meningiomas and ependymomas). The most common calcifications seen in neurofibromatosis type 2 are the ones associated with disease-related tumors, such as meningiomas or ependymomas. Nontumoral calcifications are mainly nodular calcifications of the cerebellum, simetric / often asymmetric calcifications of the choroid plexus and seldom cortical calcifications.[2,3,5]

Sturge-Weber syndrome also known as encephalotrigeal angiomatosis, is the only phakomatosis that is not associated with intracranial neoplasms.[8]


Intracranial atherosclerosis. The presence of calcifications in the arterial wall of large intracranial vessels should be mentioned in radiologist report because of their association with atherosclerosis. The carotid siphon (~60%) is the most commonly affected vessel, calcifications in the vertebral artery (~20%) and middle cerebral arteries (~5%) are less common.[2,3]

Other causes of vascular intracranial calcifications include:

Aneurysm: although patent aneurysms may contain mural calcification, partially or entirely thrombosed aneurysms commonly have calcification.[2]

Arteriovenous malformation: are arterio-venous shunting with no capillary bed interfering. They may contain dystrophic calcification along the tortuous vessels and within the adjacent parenchyma with prevalence of 25 - 30%. [11]

Cavernous malformation: are abnormal clusters of low-pressure blood vessels embedded in normal brain tissue.[2,3] Occasionally have been described intracranial calcification in developmental venous anomaly (DVA), and capillary telangiectasia. [7,12-14]

5. Infections. Patient with congenital infection present intracranial calcification with no specific appearance, often similar to any chronic brain injury (dystrophic calcifications). Calcifications in basal ganglia and the cortex are common features of all diseases that compose the TORCH syndrome (toxoplasmosis, other, rubella, cytomegalovirus, herpes simplex virus). Calcifications in patients infected with toxoplasmosis may resolve after treatment.[3,2,11]

The intracranial calcifications patterns in acquired infections, although not specific are extremely useful in making the correct diagnosis and evaluating disease progression.
Cysticercosis, tuberculosis, HIV and cryptococcus are the most common disease typically associated with calcifications.

6. Inflammatory disorders.

Systemic lupus erythematosus. In systemic lupus erythematosus cerebral calcifications has been seen in the basal ganglia, centrum semiovale, cerebellum and thalamus.[17]

Neurosarcoiosis. The neurosarcoiosis lesion appearance is nonspecific; granulomatous masses are seen as hyperdense nodules or calcification and involves the parenchyma, nerve, the leptomeninges, and dura mater.[2,3,18]

7. Tumors.

Intra-axial: oligodendroglioma, astrocytomas, medulloblastoma, ganglioglioma, DNET, metastases.[2,3,10,20]

Extra-axial: meningioma, pineal tumors, pituitary tumors, craniopharyngioma, epidermoid, dermoid, teratoma, colloid cyst, lipoma, metastases.[2,3,10,20]

Intraventricular: ependymoma, choroid plexus tumors, central neurocytoma, metastases.[2,3,10,20]

The most common intracranial neoplasms associated with calcifications are oligodendroglioma (70-90%), craniopharyngioma (50-80%), germ cell neoplasms, ganglioglioma (35-50%), meningioma (20-25%), choroid plexus papilloma (25%), medulloblastoma (20%), low grade astrocytoma (20%), and pilocytic astrocytoma (10%). Calcifications are rarely described in schwannomas, and dermoid and epidermoid tumor.[2,3]

Craniofaringioma is a benign tumor derived from Rathke pouch epithelium. Calcification is described the hallmark of a craniopharyngioma and occurs in about 90% of tumors.[2,3,7,22]

8. Metabolic/ endocrine pathologies.

Hyperparathyroidism/Hypoparathyroidism/Hypothyroidism/MELAS syndrome

Endocrine disorders involving calcium homeostasis are frequent associated with intracranial calcification. Most common locations are the basal ganglia, but also subcortical white matter, thalami and cerebellum.[22,23]

Fahr disease, a familial cerebrovascular ferro-calcinosis, is a rare condition starting in childhood presenting with progressive mental deterioration. It is characterised by extensive deposits of iron and calcium in the globus pallidus, dentate nuclei and subcortical white matter.[24]
IMAGING. Awareness of intracranial calcifications physiological localization, aspects and distribution, as well as the pathological conditions in which occurs, helps prevent errors and provides a proper differential diagnosis.

Imaging findings OR Procedure details

PATIENTS. We will illustrate the CT aspects of both physiological and pathological intracranial calcification, from a retrospective study between 2001 and 2012, that included 6882 patients evaluated by brain CT using both parenchyma and bone windows.

TECHNIQUES. Acquisitions were performed on a monoslice and a 16-row multislice CT, with 4.8 mm width slice, in sequential mode (using 2.4 mm brain and bone windows reconstruction), with and/or without contrast medium.

IMAGING FINDINGS. Most patients presented physiological intracranial calcification, choroid plexus (73%) and pineal gland (71%) calcification were predominant.

Pathological intracranial calcification may be found subependymal (tuberous sclerosis), leptomeningeal (cryptococcosis), dural (meningioma), in vascular wall (atherosclerosis), as well as in other intracranial structures such as: epiphysis, choroid plexus or in parenchymal grey/white matter.

1. Physiologic calcifications may appear as: hyperdense flat plaques (falx cerebri), laminar (dural, tentorial, petroclinoid ligament, superior sagittal sinus), curvilinear (habenula, epiphysis), faint punctate or have coarse conglomerated pattern (basal ganglia).[2]

The typical location are:

- the epiphysis (Fig. 1 on page 10) -- Pineal calcification (Fig. 2 on page 11) larger than 1 cm in diameter or patients less than 9 years of age should raise concerns for underlying tumor.[5]
- habenula (Fig. 1 on page 10),
- choroid plexus (Fig. 1 on page 10),
- tentorium (Fig. 1 on page 10),
- petroclinoid ligaments (Fig. 1 on page 10),
- falx cerebri (Fig. 3 on page 12),
• basal ganglia (Fig. 4 on page 13 and Fig. 5 on page 14)—In patients under 30 years of age, basal ganglia calcification may associate underlying pathologies and need careful clinical evaluation to rule out another etiology.
• sagittal sinus.[2,3]

2. Posttraumatic or postsurgical (Fig. 6 on page 15) calcifications may appear in the capsule surrounding chronic epidural and subdural hematoma.[3] (Fig. 7 on page 16 and Fig. 8 on page 17)

In post radiation or post chemotherapy calcifications appear delayed, and are much more common in young children.[4]

3. Congenital disorders (phakomatoses)

Tuberous sclerosis. *Imaging findings*:
• cortical/subcortical hamartomas. The subcortical tubers are usually supratentorial, and they calcify mostly in elderly patients. (Fig. 9 on page 18)
• subependymal nodules. Calcified subependymal hamartomas are found mostly along the lateral ventricles and may appear as localized projections into the ventricular cavity. (Fig. 9 on page 18)
• white-matter abnormalities.[7]

**Neurofibromatosis type I.** Non-enhanced *CT features*:
• enlarged optic nerve foramina and fissure (Fig. 10 on page 19) and dural calcification, explained by the association of different intracranial tumors, such as optic nerve glioma and plexiform neurofibroma.[7]

**Neurofibromatosis type II.** Intracranial calcifications are:
• non tumoral—mainly nodular calcifications of the cerebellum, symmetric or often asymmetric calcifications of the choroid plexus and seldom cortical calcifications or
• associated with disease-related tumors, such as meningiomas or ependymomas.[2,3,5]

**Sturge-Weber.** *CT features*:
• dense gyriform cerebral calcifications (Fig. 11 on page 20), often affect the parietal-occipital cortical areas or choroid plexus;
• diffuse high attenuation of the superficial and deep white matter, presumably due to microcalcifications;
• brain atrophy;
• thickening of the calvaria--as an indirect feature of loss of the brain substance;
• gyriform enhancement--reflecting pial angiomatosis.[7-10]

4. Vascular disorders

Intracranial atherosclerosis emphasized by the presence of linear or punctate arterial wall calcifications of large intracranial vessels (Fig. 12 on page 21), affecting mainly the carotid and middle cerebral arteries (Fig. 13 on page 22) and the vertebro-basilar system. (Fig. 13 on page 22)

Other causes of vascular intracranial calcifications are:

Aneurysm: thrombosed aneurysm commonly presents calcifications with rim-like and granular pattern. (Fig. 14 on page 23)

Arteriovenous malformation seen as:

• iso/hyperdense serpentine vessels;
• multiple curved (Fig. 15 on page 24) or punctate (Fig. 16 on page 25) vascular calcifications;
• and vascular tracks with prevalence peripheral location and strong enhancement.

Developmental venous anomaly represented by dilated medullary white matter veins with "medusa head" aspect. (Fig. 17 on page 26) CT features:

• occasional small punctate calcification;
• enhancing stellate tubular vessels converging in collector vein.[2,3,7]

Cavernous malformation are usually smaller than 3 cm, well defined hyperdense masses, without causing mass effect, sometimes partially calcified. After intravenous contrast administration there is little or no enhancement.[7] (Fig. 18 on page 27)

Vein of Galen aneurysms may appear on non-enhanced CT as mildly hyperdense venous pouch with wall calcifications, hydrocephalus and parenchymal calcification. [7,15]

5. Infections
**Congenital**

Cytomegalovirus and toxoplasmosis infections are commonly associated with hydrocephalus and randomly periventricular, subependymal, basal ganglia and cerebral cortical nodular calcifications.[3] Infection with immunodeficiency virus results in periventricular, frontal white-matter and cerebellar calcifications. Congenital herpes (HSV-2) infections present punctate or extensive gyral calcification, thalamic and periventricular calcification, also extensive cerebral destruction and multicystic encephalomalacia.[2,11]

**Acquired**

**Cysticercosis.** Typical appearance is that of a small calcified cyst with eccentric calcified nodule, representing the dead scolex. The most frequent calcifications locations are in the brain parenchyma, especially the gray-white matter junction and subarachnoid spaces in the convexities, ventricles, and basal cisterns.[2,3]

**Cryptococcosis** affects immunocompromised patients. Calcifications can be present in both the brain parenchyma and the leptomeninges.[3,16]

**HIV.** Calcifications may be seen in basal ganglia in patients with HIV encephalitis.[3]

**Tuberculosis.** Calcified parenchymal tuberculoma can occur in intracranial tuberculosis. The "target sign" formed by the calcified central nidus with peripheral ring enhancement is signifying tuberculoma.[7]

**6. Inflammatory disorders**

In **systemic lupus erythematosus** cerebral calcifications have been seen in the basal ganglia, centrum semiovale, cerebellum and thalamus.[17]

**Neurosarcoïdosis.** Lesions involve the parenchyma, leptomeninges and dura mater. The hallmark of neurosarcoidosis is the basal leptomeningeal involvement.[2,3,18]

**7. Tumors**

**Metastasis.** Intracranial calcification may occur from lung, breast, colon cancer (Fig. 19 on page 28) and osteogenic sarcoma.
Oligodendroglioma. Oligodendroglioma is the most common intracranial neoplasm associated with calcifications. The calcifications can be central or peripheral, punctate or ribbon-like, located within walls of intrinsic tumor vessels, and they may extend to the surrounding brain parenchyma. Cystic component may be present and the enhancement is variable.[2,3,7](Fig. 20 on page 29)

Astrocytoma. In diffuse low grade astrocytoma calcification are described as linear, punctate or multifocal, diffuse and may follow the white-matter tracts (more often in large tumors). Calcified chunks or nodules are present in most of the subependymal giant-cell astrocytomas and may be associate with other finding of tuberous sclerosis. Up to 20-25% of pilocytic astrocytomas have intratumoral calcification.[2,3,11]

Ependymoma. *CT features:*
- # presents as irregular shape mass in the 4th ventricle, extending in cisterna magna and cerebellopontine angle;
- calcifications (~50% of cases) ranging from small punctate foci to large masses;
- associated with hydrocephalus [7,10,22].

Craniopharyngioma. *CT features:*
- partially calcified, partial solid, cystic suprasellar mass in children,
- sometimes is associated with circle of Willis displacement.[2,3,7,22](Fig. 21 on page 30)

Meningiomas. About 25-30% of meningiomas are fully calcified on CT. The calcifications are either focal or diffuse (Fig. 22 on page 31), psammomatous, rim (Fig. 23 on page 32) or have radial pattern (Fig. 24 on page 33). Locations: 85 - 90% supratentorial [1], infratentorial or miscellaneous. [2,3,7]

*CT features:*
- extra-axial lesion ("inward buckling");
- isodense or hyperattenuating to parenchyma;
- underlying parenchymal edema;
- homogeneous and vigorously enhancement (Fig. 24 on page 33);
- "dural tail" sign;
- hyperostosis or osseous erosion.[7,19-21]

Causes of basal ganglia calcification:

- Metabolic-related:
Hypothyroidism may exhibit calcification in basal ganglia and cerebellum.

Hypoparathyroidism, either idiopathic or following thyroidectomy, is the most important cause because it may be treatable. Calcification involve the basal ganglia, the thalamus and the cerebellum.[2,3,25]

Hyperparathyroidism is associated with subcortical and basal ganglia calcification. [2,3,25]

Lead toxicity intracranial calcification are common confined to globus pallidus.[3,25]

Fahr disease, also known as bilateral striopallidodentate calcinosis, showing characteristic calcification in lateral globus pallidus.

Hallervorden Spatz disease is a neurodegenerative disease with hyperintensity in globus pallidus represented by brain iron accumulation. [2,25]

-Others causes:

Ischemic: carbon monoxide intoxication, birth anoxia (generally limited to globus pallidus).[18]

Congenital: trisomy 21

Chemotherapy: methotrexate

Radiation therapy. In post radiation therapy calcifications are commonly found in subcortical white-matter and basal ganglia in mineralizing microangiopathy (Fig. 25 on page 34) and in posterior white-matter areas in necrotizing leukoencephalopathy.[2,3]

Images for this section:
Fig. 1: F, 73 years NECT (a) Calcified bilateral petroclinoid ligaments. (b) Small calcification along the tentorium. (c) Bilateral coroid plexus (arrowheads), pineal and habenular (arrow) calcification.
Fig. 2: Different pattern of pineal gland calcification: (a) nodular, (b) curvilinear, (c) punctate.
Fig. 3: M., 69 years, NECT Linear calcified falx cerebri.
Fig. 4: Axial NECT in two patients. Different type of bilateral globus pallidus calcifications (a) coarse conglomerated and (b) punctate.
**Fig. 5:** Right caudat head dense calcification in a 55 years old male.
**Fig. 6:** Post surgery right frontal cortical (red arrows) and dural (white arrow) calcification in a patient with LMNH.
Fig. 7: NECT in a patient with hydrocephaly (star). Left posttraumatic dural calcification (after epidural hematoma)(arrow).
Fig. 8: 52 years old male with right chronic (hypodense) subdural hematoma (stars). NECT - small linear dural calcification (arrows)
Fig. 9: Multiple calcified subependimal hamartomas along the lateral ventricles and foramen of Monro in two different patient with tuberous sclerosis; (a,c) infratentorial small calcification (b,d) supratentorial partial calcified subcortical tuber (white arrow).
**Fig. 10:** Neurofibromatosis Type I; (a) NECT - Multiple small intracerebral calcification, widened optic nerve foramina (large white arrow), (b) MRI with Gd shows multiple intracerebral tumoral lesions (red arrows) little enhancement (green arrows) and enlarged optic nerves and optic chiasm (small white arrow).
Fig. 11: M, 55 y, presenting facial angioma and epilepsy. NECT reveal typical left fronto-parietal gyral calcification in Sturge-Weber Syndrome (red arrows); left fontal lobe atrophy associated (white arrow).
**Fig. 12:** Fig.14 Megadolico vertebro-basilar system and internal carotid artery with multiple small atherosclerotic calcified plaques.
Fig. 13: Important calcified plaques in the wall of verterbo-basilary system and bilateral internal carotid (red arrows), middle cerebral artery (white arrow). Extensive atherosclerosis. M, 80 years.
Fig. 14: Gigant left middle cerebral artery thrombosed aneurysm (red arrows). (a) NCET, Rim like wall calcification and granulous calcification; moderate edema developed in temporo-insular left region, (b) MRI and (c) CECT confirming partial thrombosed aneurysma; enhanced residual lumen (green arrow).
Fig. 15: Arterio-venous malformation. Right frontal lobe heterogeneous lesion: (a) with small hypodense central area, multiple serpenteses vascular calcification (white arrows), (b) intense enhanced vascular tracks located peripheral (red arrows).
Fig. 16: Angio-CT: MIP and source image - complex vascular malformation: aneurysm of the pericalosal artery and partially thrombosed saccular venous dilatation with peripheral calcifications (arrows).
Fig. 17: Developmental venous anomaly. Right cerebellar hemisphere heterogeneous lesion with small hypodense central area, multiple serpentine vascular calcification (red arrows), vascular tracks with "medusa head" sketch located peripheral with intense enhancement (white arrows).
Fig. 18: Cavernous malformation. (a) NECT Small hyperintens lesion, with punctate calcification in left periventricular white matter, near occipital lateral ventricle horn. (b) MRI. "Pop-corne" appearance with hipointense hemosiderin rim on T2-wi (green arrow); T1-wi with Gd - shows small venous malformation associated (white arrow).
Fig. 19: Massive calcifications in right frontal lobe colon cancer metastasis.
Fig. 20: Subcortical/cortical large left frontal oligodendroglioma (a) NECT, (b) MRI with Gd - heterogenous mass with nodular calcification (arrows) and cystic component (star)
**Fig. 21:** Obstructive craniofaringioma. Axial NECT (a)- Heterogenous suprasellar masse with periferal calcification (red arrows) and hipodens area; hydrocephaly associated(star); (b) CECT - Little enhancement (small white arrows) and mild displacement of Circle of Willis (large white arrow).
Fig. 22: Large left parasellar meningioma. (a) NECT heterogenous mass with diffuse calcification, (b) MRI with Gd- homogenous enhancement (white arrow).
**Fig. 23:** NECT - large left meningioma in two different patient with (a) circular calcification pattern and (b) radial calcification pattern.
**Fig. 24:** Meningioma. (a) NECT Large left meningioma with radial pattern calcification, (b) CECT - omogenous enhancement.
Fig. 25: Mineralizing microangiopathy. Small bilateral calcifications involving the thalamo-lenticular regions and the subcortical white matter (arrows).
Conclusion

1. Intracranial calcifications are relatively common and CT is the most sensitive method in their detection and proper location.

2. The presence of intracranial calcifications, their distribution and semiological appearance in association with the clinic and biological data and in particular cases the follow up of the patient, help to make an accurate diagnosis.

References


[25] Bindu Menon Similar calcifications of the brain on computed tomography, but different etiologies.

**Personal Information**

Serbanescu M. Magda