Original Article

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Study on CT Evaluation Intracranial Calcifications

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Abstract

Background: Deposition of calcium is a common process which is seen in both age related and disease related processes. While other imaging modalities are also there which may aid in identification of intracranial calcium deposition, CT remains most reliable and sensitive means in their identification. With this, we aim to identify and determine the calcium deposition in various brain structures in accordance to their location, incidence, size, number and pattern of calcification. **Subjects and Methods:** This is a prospective study of 100 patients with intracranial physiological and pathological calcifications over a period of 18 months. Unenhanced CT of the brain was performed for these patients in the axial plane, complemented by coronal & sagittal 3D reconstruction with contrast study in selected cases. The patients were referred to our department and from other departments mainly department of Medicine and Pediatrics. **Results:** Intracranial calcifications both physiological and pathological are more common in older age. The Choroid plexus has highest incidence in the sample data by 81 %(81 cases). The highest incidence of pathological calcification is found in the age group of more than 40 years with infection's (granulomas) having the highest incidence of 36.62%. **Conclusion:** With advent of CT, calcifications were not only easy to appreciate and demonstrate but also several morphological patterns have helped to correlate the types of calcification of the disease process.

Keywords: Intracranial calcifications, Computed tomography, Radiological imaging, Brain.

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Introduction

In day to day practice of radiology calcification of intracranial structures are most routine findings seen on CT, because plain CT of the head is most favored imaging modality for initial assessment of patients presenting with any neurological problem (weather acute or chronic).

CT scan of the head is classically performed to identify haemorrhage, calcifications, infarction, tumors and bone trauma. Computed tomography (CT) is an unquestionable modality for identification, characterization and localization of intracranial calcifications. This study aims to evaluate intracranial physiological and pathological calcifications in all age groups.

Intracranial calcification is detected 9 to 15 times more frequently with computed tomography (CT) than with conventional skull radiography.[1] A number of factors including slice thickness; window width and level may affect the detectability of calcification on CT.[2]

Intracranial calcifications are classified primarily into six broad categories depending upon their etiology and pathogenesis:

1) Physiological
2) Congenital disorders (Phakomatoses)
3) Vascular diseases
4) Infections
5) Tumors
6) Metabolic

1) Age-related physiologic and neurodegenerative calcifications

Intracranial physiologic calcifications are unaccompanied by any evidence of disease and have no demonstrable pathological cause.[3] They are frequently due to calcium and occasionally iron deposition in the vasculature of different brain structures. Choroid plexus, pineal gland, habenula, basal ganglia, falx, tentorium, petroclinoid ligaments and sagittal sinus are among the most common site for physiological calcification. According to Weins and Stenbeg,[4] pineal calcification is found in 25% of individuals over 10 years of age. 60–70% of indiviuals above 50 years of age have pineal gland calcification. 13% of adults show calcification of habenula. To compare, only 28% of choroid plexus calcification was detected by conventional radiography and it was 75% by CT imaging in adults above 40 years of age. Only 7-9% of adults showed calcification of sagittal sinus and falx cerebri.

2) Congenital disorders

The phakomatoses are a broad group of neurocutaneous disorders affecting structures arising from ectoderm. Classically, calcifications are expressed in tuberous sclerosis and Sturge-Weber syndrome but neurofibromatosis and basal-cell nevus syndrome can also show calcification.

Sturge-Weber syndrome show calcification located in the cerebral cortex adjacent to the vascular malformations of leptomeninges, and it is usually appear after the patient attains 2 years of age. Calcifications are frequently seen in parietal and occipital cortex and are gyriform / curvilinear in appearance. Frontal lobe and/or bilateral involvement can present with extensive calcifications. Areas of cortical atrophy presents with calcifications.

3) **Vascular calcifications**

Walls of large intracranial arteries are common site for calcifications and should be stated in the report because they are frequently associated with atherosclerosis. Other causes of calcification related with vascular pathology, like vascular malformations and aneurysms should also be kept in mind. AVMs have a prevalence of 25-30% and comprise dystrophic calcifications along the vessels and within the bordering cerebral cortex.[5]

4) **Infections**

a) Congenital: TORCH syndrome (toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus), Cytomegalovirus and toxoplasmosis infections produce calcification in periventricular and subependymal areas. Congenital HIV infection is associated with periventricular frontal white-matter and cerebellar calcifications.[6]

b) Acquired infections: Neurocysticercosis, neurotuberculosis, HIV and cryptococcus comprise the major acquired infections typically affecting intracranial structures presenting with calcifications. In neurocysticercosis, it is dead larva which is calcified (granular- nodular stage) and it classically appear as small, calcified cyst with a calcified nodule located eccentrically, that corresponds the dead scolex. In brain parenchymal lesion, a "target sign" is often seen as a central nidus of calcification bordered by a ring of enhancement is highly indicative of a tuberculoma.[7]

5) **Tumors**

a) Intra-axial tumors

Subependymal giant-cell astrocytomas are benign tumors which show calcified nodules which are subependymal in location. Nearly 25% of pilocytic astrocytomas show intratumoral calcification of eccentric nodule. The highest incidence of calcification amongst all brain tumors is noted among oligodendrogliomas, as nearly 90% of them show calcium deposition. Typically found within walls of tumor vessels, calcification can be in centre or periphery of tumor, and can be punctuate or ribbon like.

b) Extra-axial tumors

Nearly 20 – 69% of meningiomas show calcification8. These calcifications can range from focal or diffuse, and can be coarse or sand like, sometimes even rim. Meningiomas in children if associated with calcification, there is higher probability that they could be of more aggressive subtypes of meningiomas.[9]

Calcifications of pineal gland are very common, larger pineal calcifications are highly suggestive of neoplastic etiology.

c) Intraventricular tumors

Intraventricular ependymomas classically calcify, range from punctuate to mass-like calcifications2. Ependymomas located in posterior fossa show small, round calcifications in nearly 50% cases and they show the highest incidence of calcification amongst the posterior cranial fossa tumors.[10]

6) **Endocrine/metabolic/idiopathic**

Fahr disease/syndrome is an uncommon degenerative disorder, results from metabolic disturbances and is characterized by widespread deposition of calcium in bilateral basal ganglia that can result in progressive dystonia, Parkinson disease, neurological and psychiatric symptoms. It is often associated with faulty transport of iron with resultant damage of tissue involved and extensive calcium deposition. Bilateral basal ganglia, cerebellar dentate nuclei, cerebral white matter and internal capsule are the frequent site of metastatic deposition.[11]

A brief knowledge of physiological age related intracranial calcification helps in spot on and early detection of pathological intracranial calcification on CT. The recognition of pathologic intracranial calcifications based upon how they appear and areas of their distribution by CT, narrows the differential diagnosis. The present study was conducted to improve accuracy of diagnosis of intracranial calcifications and to differentiate between physiologically age related and pathological intracranial calcifications.

Subjects and Methods

This is a hospital based study, includes the 100 patients who were referred to department of radiology for CT scan of brain during study period of 18 months. The study protocol was approved from ethical committee of institution.

Inclusion criteria:

1. Patients of all age groups presenting with symptoms like fever, head ache, vomiting, seizures, behavioural disorders, dizziness and neurological deficits of sudden onset.
2. Asymptomatic patients with incidental detection of intracranial calcifications on routine computed tomographic (CT) scanning done for other diagnosis

Exclusion criteria:

1. Known cases of skull bone trauma.
2. Contrast enhanced Computed tomograms of brain.
3. Known cases of previous head surgery.
4. CT scans compromised by streaky or motion artefacts.
5. Lens calcifications were excluded from this study as it lies outside cranial cavity.

The data will be collected by means of a standard proforma , meeting the objectives of the study through personal interview with the patients after taking informed consent. Equipment used was "GE Brivo CT 385 16 slice spiral CT machine" – is a helical multi slice scanner with multi-planar reconstruction and multi-planar volume reconstruction techniques. Available gantry tilt: +/- 30 degree.

Reconstruction matrix: 512 x 512. Display matrix: 1024 x 1024.

Slice thickness: 0.625, 1.25, 2.5, 3.75, 5, 7.5mm.

Available imaging modalities for intra cranial calcification:

Plain film modalities: a deposit of calcium has to be fairly heavy before it can be shown by plain film. X-rays attenuation is greater, so calcification appears as white areas on film.

Computed tomography: with use of CT, even minor degree of calcifications can be detected. Calcium absorbs more x-rays than surrounding tissues and therefore appears as a denser area in images.

Magnetic resonance imaging: calcification has a variety of appearances which is at times baffling. So it is better to carry out a CT examination if one is looking mainly for calcification.

Simple radiography, computed tomography (CT), magnetic resonance imaging (MRI) and, for infants, sonography all help physicians in the diagnosis of intracranial calcifications, but CT has a high sensitivity in diagnosis because of the hyperdense signals of calcifications in this tomography.[12]

Computed tomography is a veritable imaging modality for best identification and characterization of any intracranial calcifications. Intracranial calcification is visualized 9 to 15 times more frequently with computed tomography (CT) than with conventional skull radiography. A number of factors including slice thickness; window width and level may affect the detectability of calcification on CT.

Examination of the subjects

After positioning patient in supine position for CT scan, volumetric acquisition of data was undertaken. Taking the orbito-meatal line as reference axis, the plane of section was parallel to this line. Using slice thickness of 5 mm, scans were performed from the orbito-meatal line to the vertex with slice interval of 10 mm and reconstruction was done to 1.25mm. Automated multiplanar reconstruction was performed and images were displayed in coronal, sagittal and volume rendered images apart from axial views. The exposure settings used were 120 kVp and 80 to 100 mAs.

The densitometry is used with Hounsfield Unit (HU) to differentiate various soft tissues in the cranium. The average densities of calcifications were 100-120 HU. The other common pathology which has high density is acute haemorrhage with HU values in range of 60-80 and can be effortlessly distinguished from calcifications having HU values 100 and above.

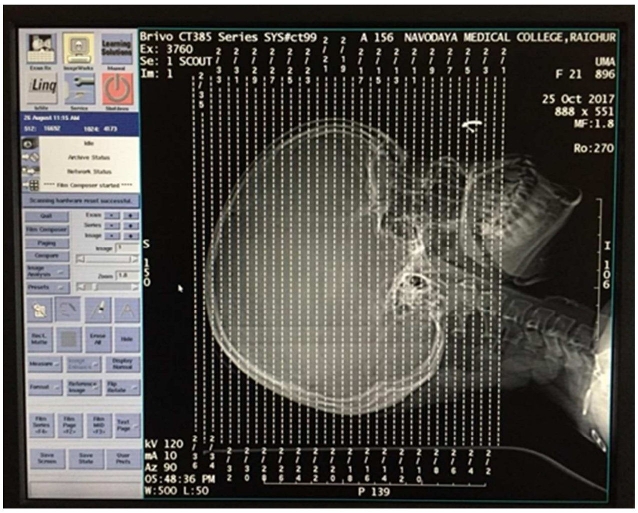

Figure 1: Section and planning of CT brain.

The regions looked for normal physiological calcifications were choroid plexus, pineal gland, habenula, ligaments (petro-clinoid, interclinoid), dura, dural folds (falx and tentorium), basal ganglia and venous sinuses. They were further evaluated for their size in millimetres, numbers and pattern of calcification (nodular, linear/punctate or amorphous). Patients with physiological calcifications generally do not have any symptoms. Other presenting with symptoms (fever, headache, vomiting, seizures, behavioural disorders, dizziness and neurological deficit of sudden onset) and calcifications at locations other than physiological were placed in pathological calcifications.

In all cases, systematic studies of the brain were performed in axial sections complemented by coronal and sagittal views in selected cases.

Statistical analysis:

Data entered in MS excel sheet and analysed by using statistical package of social sciences (SPSS version 22.0). Both descriptive and inferential statistics were applied for statistical analysis of the data in our study. A p value of ≤ 0.05 was considered as statistically significant.

Results

Majority of were in the age group of 10-74 yr and it was found that 23% of them were in the age group of above 65 years. While 10 to 24 years age group contributed to 14%, 25 to 34 yrs to 20%, 35- 44yrs to 13% and 45-54 yrs to 14% of the patients studied.

In our study choroid plexus calcification has the highest incidence closely followed by pineal gland.

Choroid plexus and pineal gland calcifications were highest in age group of above 65 years. Incidence increases with age. Incidence of intracranial physiological calcification was more common in males than females.

The patients were divided on the basis of type of calcifications. Both age related physiological and pathology associated calcifications can co-exist. In the present study, 57 (57%) cases showed only physiological calcification whereas 43 (43%) of them were detected with some pathology related with calcifications and were therefore included in pathological calcification group. Total incidence of pathological calcifications in these 43 cases was 71 nos.

Table 1: Distribution of Physiological Calcification According to Size and Pattern of Calcification

Sl. no.	Location	Avg size (in mm)	Pattern of calcification
1	Choroid plexus	7-8mm	A + N
2	Pineal gland	5-6mm	N
3	Habenula	3-4mm	N
4	Falx	4-5mm	N

The size range of calcifications was from 4 mm to 8 mm with most calcifications having an average size of 4 mm to 5 mm. The largest calcified area was choroid plexus calcification having average dimension of 7 mm to 8 mm. Over all nodular calcification was found in most of the locations. A: amorphous and N: nodular calcification.

Table 2: Incidence of Total Cases with Pathological Intracranial Calcification According to Age

Age Group In Years	No Of Cases	Percentage	χ2	P value
< 10	0	0	45.73	0.01 (S)
10- 40	6	8.5		
>40	65	91.5		
Total	71	100		

Highest incidence of pathological calcification is found in the age group of more than 40 years (91.5) and the result is statistically significant. In turn, reflects as the increase in pathological calcifications with increasing age.

Table 3: Incidence of Calcifications

Diagnosis	Number	Percentage
Infections		
Tuberculoma	21	80.77
Neurocysticercosis	03	11.54
Toxoplasmosis	02	7.69
Vascular disorder		
Atherosclerotic Wall	15	78.95 %
AVM	02	10.53 %
ANEURYSMAL	02	10.53 %
Tumor		
Meningioma	08	61.54 %
Glioma	02	15.38 %
Craniopharyngioma	02	15.38 %
Medulloblastoma	01	7.69 %
Phakomatosis		
Tuberous Sclerosis	03	50.00 %
Sturge weber syndrome	02	33.33 %
Nf 2	01	16.67 %

Among the patients with pathological calcifications, most common pathology was infection which was noted in 26 cases (36.6%). Vascular lesions associated calcifications were seen in 19 cases, tumor was detected in,[13] pathological bilateral basal ganglia in 7 cases and congenital phakomatoses in 6 cases.

Among calcifications associated with infections, calcified tuberculoma was most common among infections, majority of which were single focal calcifications. Multiple calcified granulomas were common in neurocysticercosis. 2 case of calcification was also seen with congenital toxoplasmosis infection. NCC: Neurocysticercosis.

Among calcifications associated with vascular disorders, atherosclerotic calcification was most common pathology. The carotid artery was most affected location, followed by the vertebral artery and basilar artery. Incidence of AVM and aneurysm was limited to 2 cases each. AVM: arterio-venous malformation.

Among tumors, meningioma was most commonly associated with calcification in our study. The most common location was parasagittal with 2 cases along sphenoid ridge. Calcification associated with glial tumors was seen in 2 cases of oligodendroglioma. Calcification was also noted in craniopharyngioma and medulloblastoma in our study.

Among phakomatoses, calcification was seen with tuberous sclerosis, Sturge- Weber syndrome and NF 2. In tuberous sclerosis calcified subependymal nodules were noted along the lateral ventricle. Gyral calcification were seen with Sturge- Weber syndrome. NF 2: Neurofibromatosis type 2.

Table 4: Distribution based on pattern of calcifications.

Sl. no.	Diagnosis	Amorph Ous	Puncta Te	Line Ar	Nodul Ar
1	Granuloma	00	00	00	24
2	Meningioma	01	02	00	05
3	Glioma	00	01	00	01
4	Craniopharyngioma	00	01	00	01
5	Bilateral Basal Ganglia	03	01	00	03
6	Toxoplasmosis	00	01	00	01
7	Avm	00	00	00	02
8	Aneurysm	00	00	02	00
9	Phakomatoses	00	00	03	03
10	Medulloblastoma	00	00	00	01

Nodular type of calcification was the most common pattern among pathological calcification in our study.

Discussion

In our study, the most common incidence and sites of intracranial physiological calcification areas are 81% in the choroid plexus scored high degree of calcification followed by pineal gland by 73 %, Habenula had 20 % of calcification and Tentorium & falx had 14%.

According to Kiroglu et al, choroid plexus calcifications are a common finding and are usually in the atrial portion of the lateral ventricles. Calcifications were not commonly seen in the third or the fourth ventricle.[13] Similarly, our study also demonstrated most choroid plexus calcifications to be in the lateral ventricles. The infrequent finding of unilateral choroid plexus calcifications could influence certain clinical settings where there is suspicion of pathology.

The study results are similar to Alves G et al, demonstrated increasing prevalence of both choroid plexus and pineal gland calcifications however, choroid plexus calcifications more prevalent than pineal calcifications.[14]

The result was similar to the study done by Kendall and Cavangh report the same increase in calcifications in old age with an incidence of Tentorium cerebelli, sagittal sinus and falx cerebri calcifications were most common in old age too, and pineal, habenular, and choroid plexus calcifications also increase at older age15. In our study, intracranial physiological calcification also increases with age. Choroid plexus and pineal gland calcifications were highest in age group of above 65 years with incidence of 23 cases each in our study. In our study incidence of falx calcification was 14%.

Choroid plexus calcification is known to be associated with pineal gland calcification16. In our study, the commonest calcifications noted were choroid plexi and pineal glands 81 (81% of total population) and 73 (73 % of total population) respectively. This choroidal plexus calcification predominance has been reported by other authors17 (Menon & Harinarayan et al). However a reversal of this pattern was noted by other studies,[18,19] (Daghighi et al and Admassie & Mekonne). The physiologic calcifications of the choroid plexus are very common after the age of 40 years.

Admassie and Mekonne 74 reported an overall incidence of normal pineal gland calcifications of 72.0% and that of choroid plexus 43.3%19. Similarly, the results obtained by M.H. Daghighi S et al, the most common sites of intracranial physiological calcification areas are the following: 71.0% pineal calcification, 66.2% choroid plexus calcification, 20.1% habenular calcification, 7.3% tentorium cerebelli, sagittal sinus or falx cerebri calcifications, 6.6% vascular calcification, 0.8% basal ganglia calcification and 0.9% lens and other non-defined calcifications.[18]

The pattern of choroid plexi calcification in this study was symmetrical and bilateral in 100% of positive cases of intracranial calcifications. Such calcification increased with age. Choroid plexi calcifications are known to occur in all ventricles, most commonly in the glomus within the atrium of lateral ventricles near foramen of Monro. Other sites are telachoroidea of third ventricles, roof of fourth ventricle along foramen of Luschaka (Dahnert, 2003, p237)20. In this study, all the calcifications were in the atria of lateral ventricles.

According to our study, Choroid plexus calcification had the highest with much higher incidence of 81 %. Weins J, Stenbeg and Daghighi S,[4,18] obtained an incidence of 66.2 % calcification in the choroid plexus which is less when compared to our study.

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. In our study habenula shows an incidence of 20 % which is on the higher aspect as compare other studies.

The average size of calcification involving choroid plexus was 7-8 mm and that of pineal gland was 5-6mm. While calcifications at habenula and falx measured 3-4mm and 4-5mm on an average respectively.

The pathological calcification was seen in 43% (total incidence 71) of the patients and increases with age with 88.4 % of patient's shows an age of over 40 years which shows an increase in pathological calcification increases with age.

The most common cause of pathological calcification in our study was Infections which were associated with 36.62% of the patients. The other reasons of the pathological calcifications were Tumors 18.31%, vascular disorders 26.70%, disorders of basal ganglia 9.86 %, Phakomatoses 8.45%. The most common reason of pathological intra cranial calcifications were granuloma with prevalence in 33.8% of cases while the second most common reason was atherosclerotic calcification found in 21.13% cases.

According to Kiroglu et al. calcification of the intracranial arteries associated with primary atherosclerosis is more frequent in elderly people. Other causes of vascular calcifications include aneurysm, arteriovenous malformation (AVM) and cavernous malformation10.

In our study 24 patients showed granulomatous calcification. Among the patient with granuloma, the tuberculosis is found in 87.50% of the patients. Tuberculosis results in calcified parenchymal granulomata in 10% to 20% of patients; meningeal calcifications are much less common. HIV encephalitis is associated with basal ganglia calcification21. Cryptococcus affects immunocompromised patients and calcifications can be seen in both the brain parenchyma and the leptomeninges.[22]

Incidence of calcified granuloma associated with neurocysticercosis was limited to 3 cases in our study.

Intracranial calcifications can also be seen in rare idiopathic disorders such as Fahr's disease. This disease shows characteristic calcifications in basal ganglia predominantly involving lateral globus pallidus. Other involved areas are thalami, cerebral white matter and dentate nuclei of cerebellum. In our study bilateral basal ganglia calcification did find in 7 cases.

Conclusion

The distribution and appearance of pathological from normal intracranial calcifications is usually different but many a times overlapping and confusing. Familiarity with CT findings is important for proper diagnosis and management of cases.

In the present study, all patients with pathological calcifications also had physiological calcifications. So our work has led us to conclude that normal physiological calcifications in CT have a documented location where they usually occur and follows an order unlike pathological calcifications. Several pathologic conditions involving the brain are associated with calcifications and the recognition of their appearance and distribution helps narrow the differential diagnosis.

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