Original Research Article

Clinical evaluation of CT imaging in pathological intracranial calcification

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Abstract

Background: Intracranial calcification includes physiological and pathological calcification. Pathological intracranial calcification occurs in tumors, infections, and infestations, vascular pathology, metabolic and miscellaneous conditions. It is important to differentiate one from the other and can be done through various imaging modalities. Each pathology has a predilection for a particular site and has a particular pattern of calcification **Aim Of The Study:** To study the location, size, number, pattern, and incidence of intracranial calcification. To study the prognostic significance of intracranial calcification in a few cases. **Materials And Methods:** This is a prospective study *was done at* DR.Chandramma Dayananda Sagar institute of medical education and research Karnataka in the Division of Radiology and Imaging sciences between April 2020 to September 2020.Totally 50 patients with intracranial pathological calcification. The study was done to know the diseases that calcify. The pattern, size, site, calcifications were studied. **Results:** Out of the 50 cases, a maximum of 30 (60%) was due to infections, followed closely by tumors 17 (34%). The other causes contributed to 3 (6%) of which vascular was 2 (4%) and bilateral basal ganglia calcification 1 (2%). Out of the 29 cases of granulomas, Tuberculomas contribute to 9 cases (18%) and neurocysticercosis to 20 cases (40 %). Various gliomas which showed calcification were choroid plexus papilloma 2 cases (4%), ependymoma 1 case (2%), oligodendroglioma 1 case (2%), pilocystic astrocytoma I case (2%). **Conclusion:** Identification of calcification assures both the clinician and the patient alike regarding the favorable outcome. Calcification represented benignity, inactivity in the majority of the cases indicating control of or the death of a particular lesion.

Key Words: Intracranial calcification, vascular pathology, Tuberculomas

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INTRODUCTION

Neuroradiology plays a vital role in the evaluation of intracranial lesions. In addition to the location, size, and mass effect of these lesions, the radiologist can offer a differential diagnosis of tumor type based on imaging characteristics, like enhancement patterns, calcification, and the amount of surrounding edema./[1] Associated complications are usually evident on standard radiologic examinations.[2] Finally, the effects of therapy can be followed using the more recently developed functional imaging techniques, and the results of these studies can guide further treatment.[3] Calcification of the brain or supporting structures may represent past or present intracranial disease, or it may be merely a manifestation of normal degenerative or aging processes. The deposition of calcium salts in tissues other than bone is a pathological process known as calcification.[4] In calcification, the same salts concerned with ossification are deposited in tissue that is either dying or dead and from which cell structure has disappeared.[5] This is due to the inability of such cells to maintain a steep calcium gradient. The four components

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of many diseases of CNS that are important in the analysis of images and diagnosis are brain calcification, cerebral edema, intracranial pressure and hydrocephalus, and myelin/demyelination.[6] Calcification may occur in normal tissue as the result of being flooded with calcium (metastatic calcification), or it may represent the deposition of calcium in tissue, which is injured, degenerating, or dead (dystrophic calcification) As the presenting symptoms and signs of most of these conditions are very similar it is difficult to arrive at a diagnosis.[7] With the availability of imaging modalities like computed tomography, we can study the pattern, incidence of calcification, and other associated pathology of each condition and thus arrive at a diagnosis.[8]

MATERIALS AND METHODS

This is a prospective study *was done at* DR.Chandramma Dayananda Sagar institute of medical education and research Karnataka in the Division of Radiology and Imaging sciences between April 2020 to September 2020.Totally 50 patients with intracranial pathological calcification. The study was done to know the diseases that calcify. The pattern, size, site, calcifications were studied. The patients were referred to our department from other departments like Neurology, Neurosurgery, Medicine, Surgery, and Pediatrics, Most of the patients presented with headache, seizures, fever, dizziness, vomiting, etc. In all the cases the clinical history and a thorough clinical examination were done and the findings, blood and laboratory investigations, and the clinical diagnosis were recorded in the proforma. Those patients who showed evidence of calcification on computed tomography were included in the study. Patients in whom the conditions were incidental were also included. If the calcification was detected initially by CT, then the other investigation was carried out. Patients with physiological calcification were excluded from the study. The patients who were subjected to the study had clinical complaints like headaches, dizziness, convulsions, etc. All the cases were studied on PHILIPS COMPUTED TOMOGRAPHY system, which is a modified 3rd generation machine. Factors of l30kV and 70 mA were constant for all cases and factors of 11OkV and 50mA were used for infants. In the case of adult patients, the patients were kept nil orally for 4 hours before the procedure to avoid the complication of contrast administration such as vomiting. In infants and children aged 1 month to 6 years, sedation was usually required before the scan. The purpose of sedation was to avoid motion artifacts and to ensure a CT scan of diagnostic quality. From 6 years onwards the need for sedation generally decreased. A preliminary lateral program was done in all cases. Routine axial scans were performed on all cases, taking the orbitomeatal line as the baseline. 5mm slices were performed for the posterior fossa with 5mm table increment and 10mm slices for the supratentorial region with 10mm table increment with a scan time of 3 seconds were routinely employed. Thin sections were done wherever necessary and at the site of interest. A coronal scan was done whenever required. Coronal and sagittal reconstructions were done.

Method of collection of data: Patients who were reported to have calcification on plain film radiograph were subjected to CT scan examination and who were reported to have pathological calcification by the Professor were selected. In cases where calcification was detected, the following features of the calcifications were noted like HU values i.e., average attenuation in HU of the calcified lesion was taken, measurements i.e., size (maximum AP and Lateral diameters) of the calcified lesion were measured by cursor device on the console, pattern, number, location and distribution within the lesion and all these details were entered in the proforma.

RESULTS

TABLE 1: INCIDENCE OF CALCIFICATION ACCORDING TO AGE

SI NO.	AGE GROUP IN YEARS	NO. OF CASES	PERCENTAGE
1	<16	13	26
2	17 – 40	16	32
3	>40	21	42

In our study, the 50 cases were divided into three groups in the following manner, <16Years (pediatric age group), 17-40 years, and > 40 yrs. The maximum number of patients were in the age group of > 40 years – 21 cases (42%), Next was 17-40 years - 16 cases (32%) and lastly <16 years – 13 cases (26%) In this study of 50 cases of pathological intracranial calcification, the distribution according to sex in male is 32(64%) and female 18(36%).

TABLE:2 DIAGNOSIS

SL NO	DIAGNOSIS	NO OF CASES	PERCENTAGE
1	INFECTIONS	30	60
2	TUMORS	17	34
3	VASCULAR	2	4
4	BASAL GANGLIA CALCIFICATION	1	2

Table:2 Out of the 50 cases, a maximum of 30 (60%) was due to infections, followed closely by tumors 17 (34%). The other causes contributed to 3 (6%) of which vascular was 2 (4%) and bilateral basal ganglia calcification 1 (2%).

Table 3: INCIDENCE OF CLASSIFICATION IN INDIVIDUAL CONDITIONS

Sl No	Diagnosis	No of Cases	Percentage
1	GRANULOMA	29	58
2	TOXOPLASMOSIS	1	2
3	MENINGIOMA	9	18
4	GLIOMA	5	10
5	CRANIOPHARYNGIOMA	2	4
6	MEDULLOBLASTOMA	1	2
7	AVM	1	2
8	ANEURYSM	1	2
9	BILATERAL BASAL GANGLIA CALCIFICATION	1	2

Table :3 Out of the 50 cases of intracranial calcification 29 (58%) were due to granulomas and 1 case (2 %) was due to congenital toxoplasmosis. Meningioma was the most common tumor to calcify 9 cases (18%), gliomas 5 cases (10%), craniopharyngioma 2 cases (4%), and medulloblastoma 1 case (2 %). The various vascular conditions which demonstrated calcification were arteriovenous malformation 1 case (2%), aneurysm 1 case (2%), and. 1 case (2%) were of idiopathic bilateral basal ganglia calcification.

TABLE 4: INCIDENCE OF CALCIFICATION IN GRANUILOMAS

SL. NO.	GRANULOMA	NO. OF CASES	PERCENTAGE
1	TUBERCULOMA	9	18
2	NEUCYSTICERCOSIS	20	40

Table:4 Out of the 29 cases of granulomas, Tuberculomas contribute to 9 cases (18%) and neurocysticercosis to 20 cases (40 %).

TABLE 5: INCIDENCE OF CALCIFICATION IN GLIOMAS

SL. NO.	GLIOMAS	NO.OF CASES	PERCENTAGE
1	CHOROID PLEXUS PAPILLOMA	2	4
2	EPENDYMOMA	1	2
3	OLIGODENDROGLIOMA	1	2
4	PILOCYTIC ASTROCYTOMA	1	2

Table:5 Various gliomas which showed calcification were choroid plexus papilloma 2 cases (4%), ependymoma 1 case (2%), oligodendroglioma 1 case (2%), pilocystic astrocytoma I case (2%).

TABLE 6: INCIDENCE OF CALCIFICATION DETECTED IN RESPECTIVE LOCATION

LOCATION	NO. OF CASES	PERCENTAGE
SUPRATENTORIUM	47	94
INFRATENTORIUM	3	6

Table:6 Calcification is more common in the supratentorial as compared to infratentorial on CT. The distribution of calcification in the supratentorial and infratentorial was as follows, supratentorial 47 cases (94%) and infratentorial 3 cases (6%).

TABLE 7: PATTERN OF DISTRIBUTION OF CALCIFICATION ON CT

SL.NO.	DIAGNOSIS	AMORPHOUS	PUNCTATE	LINEAR	NODULAR
1	GRANULOMA				29
2	MENINGIOMA	6	2	1	5
3	GLIOMA	-	3	1	4
4	CRANIOPHARYNGIOMA	-	1	-	2
5	BILATERAL BASAL GANGLIA	1	-	-	-
6	TOXOPLASMOSIS	-	1	-	1
7	AVM	-	-	1	1
8	ANEURYSM	-	-	1	-
9	MEDULLOBLASTOMA	-	-	-	1

Table:7 On CT, the pattern of calcification that was seen was nodular which was most commonly seen in 43 followed by in the descending order, punctate and amorphous 7 each, linear 4. Few cases had more than one pattern of calcification within them.

TABLE 8: SIZE OF CALCIFICATION

SL.NO	DIAGNOSIS	<1cm	>1cm
1	GRANULOMA	26	3
2	MENINGIOMA	3	6
3	GLIOMA	1	4
4	CRANIOPHARYNGIOMA	1	1
5	TOXOPLASMOSIS	-	1

Table:8 Granulomas formed the major part of this study by 58%. We had 29 cases of calcified granulomas, of which tuberculomas were 9 (18%) and neurocysticercosis 20 cases (40%). There were 9 cases of tuberculoma with calcification in this study, 5 cases were female and 4 were male patients. All tuberculomas were in the supratentorial (9 cases). Also, a single lesion was seen in 7 cases and multiple in 2 cases. Following was the lobar distribution of the calcified tuberculomas in the descending order parietal 3, occipital, temporal, and frontal 2 each. The least HU value of the calcified lesion was 85HU and the maximum was 160 HU. Most of the cases i.e., 7 were below 1 cm in size, 2 were > 1 cm. The pattern of calcification was dense, homogeneously nodular in all the 9 cases on CT with sharp borders if less than 1cm and irregular borders in case of larger lesions.

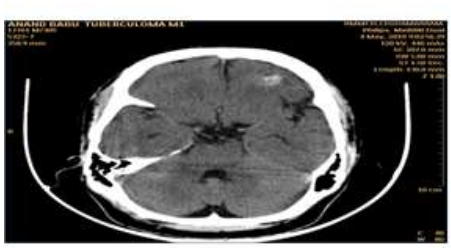

IMAGE 1

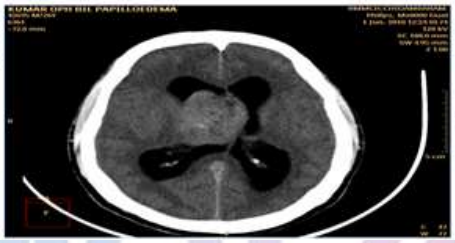

IMAGE 2

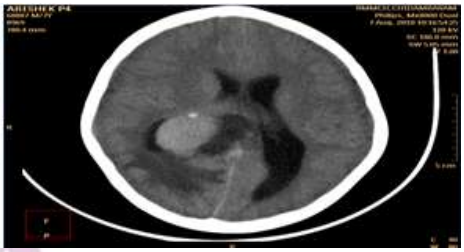

IMAGE 3

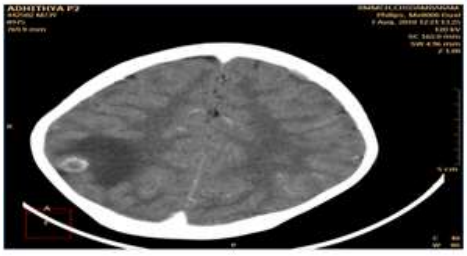

IMAGE 4

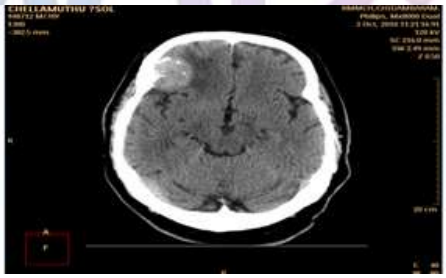

IMAGE 5

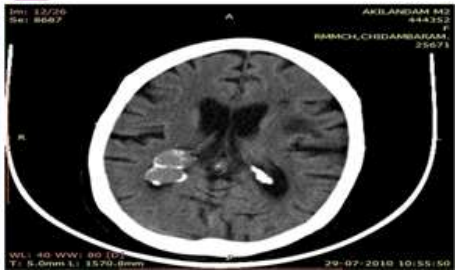

IMAGE 6

IMAGE 1: CT AXIAL SECTION OF TUBERCULOMA SHOWING HYPERDENSE LESION IN THE LEFT FRONTAL REGION**; IMAGE 2:** CT AXIAL SECTION OF MENINGIOMA SHOWING SOFT TISSUE DENSE MASS IN THE RIGHT PARASAGITTAL REGION WITH A HYPERDENSE FOCI**; IMAGE 3:** CT AXIAL SECTION OF PILOCYTIC ASTROCYTOMA SHOWING SOFT TISSUE DENSE MASS IN THE RIGHT PARIETAL REGION WITH HYPERDENSE FOCI AND PERILESIONAL EDEMA**; IMAGE 4:** CECT AXIAL SECTION OF TUBERCULOMA SHOWS A RING ENHANCING TARGET LESION WITH PERILESIONAL EDEMA; **IMAGE 5:** CT AXIAL SECTION OF MENINGIOMA SHOWS EXTRA AXIAL MASS WITH HYPERDENSE FOCI WITH MASS EFFECT, PERILESIONAL EDEMA**; IMAGE 6:** CT AXIAL SECTION OF CHOROID PLEXUS PAPILLOMA SHOWING SOFT TISSUE DENSE MASS WITH CALCIFICATION IN THE RIGHT OCCIPITAL HORN OF LATERAL VENTRICLE

DISCUSSION

Demonstration of calcification inside the cranium by CT imaging has always attracted the attention of clinicians. The incidence and etiology of pathological intracranial calcification in this area have been studied. Infectious causes like tuberculoma and cysticercosis and toxoplasmosis account for 60% (30 cases) of pathological calcification in this study similar to the study done by Dunn J I.[9] The second commonest cause was tumors accounting for 34% (17cases), followed by vascular 4% (2 cases) and pathological calcification of basal ganglia 2% (1 case). Calcification showed a male preponderance. Calcifications were more common in supratentorial as compared to infratentorial on CT. The percentage of calcification in the supratentorium and infratentorial are 94% and 6% respectively on CT.[10] Pathological intracranial calcification was more in adults 74% as compared to the pediatric age group 26%. Pathological

intracranial calcification in this study will be discussed under infections, tumors, vascular, and calcification of basal ganglia.[11] Infection causes like granuloma (tuberculoma and neurocysticercosis) and toxoplasmosis formed the commonest group - 60%. Among granulomas, tuberculoma contributed to 18% and neurocysticercosis to 40% similar to the study done by Golding R,[12] Out of 30 cases of IC calcification 9 cases were due to tuberculoma, which was detected on CT. Although the overall incidence of tuberculosis of the central nervous system has declined, it remains a disease process in our country and that is the reason why calcified tuberculomas formed part of this study. Of the 9 cases, 18% all were in the supratentorium.[13] Grainger RG, in their study, found about 60% were multiple and supratentorial and 40% were infratentorial Of the 9 cases, 4 cases had a tubercular focus in the chest which was evident on chest X-ray. 6 patients were started on antitubercular therapy and all of them were symptom-free after 4 to 6 months. The other 3 patients were treated symptomatically. Out of 50 cases of IC calcification, 20 cases were due to neurocysticercosis.[14] Most of the calcified lesions in our study were situated at the gray or the junction of gray and white matter. All 20 patients were treated with corticosteroids and albendazole and were symptom-free after the treatment. Toxoplasmosis is probably the most common cause of intracranial calcification in the neonate and only the congenital form causes intracranial calcifications.[15] The pattern of calcification in our study was punctate, nodular on CT and a similar pattern has been described by Jain DV et.al who says the pattern can be plaques, nodules, curvilinear and punctate. In 88.88% the tumor was in the supratentorium and in 11.12% the tumor was in the infratentorial.[16] The range of HU in diffuse calcification was around 92 HU. The HU range for nodular calcification was around 240 HU. According to our study, the distribution of tumor sites were the commonest being parasagittal 36.33% (3 cases) followed by convexity at 22.22% (2 cases), parasellar and posterior fossa 22.22% (2 each) The pattern of calcification in our study was linear in one, punctate and nodular in the other case similar to that described by Kieffer AS *et.al* [17] Calcification within the neoplasm is the hallmark of oligodendroglioma. The great majority of craniopharyngioma is densely calcified suprasellar masses lying within the suprasellar cisterns Kingsley HP[18] Calcification in craniopharyngioma is dense and extensive, it always extended to or beneath the diaphragm sellae. The calcification pattern in our study was nodular. Calcification is a less common feature of medulloblastoma as stated by Latovitzki N *et.al* [19]The pattern of calcification in our series was curvilinear. In this, the basal ganglia showed evidence of calcification. Following the identification, the patient was investigated extensively, despite which the cause was eluding and so it was concluded as idiopathic. According to his study in the very mild form, the calcification is linear bilaterally or punctate and EMI numbers are in the 40s detecting very early calcification and without mass effect on the lateral ventricles.[20] And as the calcification becomes more severe, this becomes more oval and there is no impression on the lateral ventricle. The EMI numbers are in the region of 60 or 70. In the severe form, the calcification is more extensive and need not be confined to the basal ganglia and there is an impression on the frontal horns.[21]

CONCLUSION

Pathological calcification is a marker of present or past intracranial disease. It is a radiological challenge to appreciate and demonstrate such markers. Earlier attempts to identify calcification through conventional modalities were not as much rewarding as it should have been because of the overlapping of the calvarial bones and low density of calcification as compared to the bone. With the advent of CT calcifications were not only easy to appreciate and demonstrate, but also the several morphological patterns have helped to correlate the types of calcification of the disease process. The findings suggest that CT analysis of intracranial calcification concerning most of the disease conditions was highly sensitive and specific with good repeatability and reliability

REFERENCES

1. Armington G, Osborn GA: Supratentorial Ependymoma: CT appearance. Radiology 1985; 157: 367 - 372.
2. Banna M: Review Article on Craniopharyngioma: based on 160 cases. BJR 1976: 49: 206 - 223.
3. Bilaniuk TL, Zimmerman AR: Computed Tomography of Brain Stem Gliomas in Children. Radiology 134; 89 - 95. Jan. 1980.
4. Cohen RC, Duchesneau MP: Calcification of the Basal Ganglia as visualized by Computed Tomography. Radiology 134; 97 - 99, Jan. 1980.
5. Damjanov I, Linder J (Edt.): Anderson's Pathology. 10,h edn.Vol. I. Mosby Year Book, INC, 1996.
6. Daniels LD. Williams LA: Differential Diagnosis of Intrasellar tumors by Computed Tomography. Radiology 141; 697 - 701. Dec 1981.
7. Danzinger Allan, Kalk JW; Computed Tomography in Basal GangliaCalcification. Clinical Radiology (1980) 31, 167 - 168.
8. Duffer KP. Klein MD; Calcification in Brain Stem Gliomas. Neurology 28; 832 - 834 Aug. 1978.
9. Dunn J I. Palmer SE: Toxoplasmosis. Seminars in Roentgenology, Vol XXXIII, No. 1 (Jan.), 1998; pp. 81 - 85.
10. Errest J.W, Floro Miraldi principles in computed tomography. Hagga J.R. Lanzaicri F.C.Sartoris D. J. Zarhouni E.An (Edr) in computed tomography and magnetic resonance imaging of the whole body,3rd
11. Fitz RC, Holgate CR: computed Tomography in craniopharyngiomas, Radiology 127;687-691, June 1978

12. Golding R, Peatfield CR; Computer Tomographic Features of Giant Intracranial Aneurysms. Clinical Radiology (1980)31,41- 48.
13. Gouliamos DA, Jimenez PJ: Computed Tomography and Skull.Radiography in the Diagnosis of Calcified Brain tumors. AJR 130;761 - 764, Apr. 1978.
14. Grainger RG, Allison DJ: Diagnostic Radiology and Textbook of Medical Imaging. 3rd edn., Vol. 3, Churchill Livingstone.
15. Grossman IR and Davis RK: Cranial Computed Tomographic Appearance of Chondrosarcoma of the Base of the Skull. Radiology 141:403 -408, Nov. 1981.
16. Jain DV, Talwar Al: Cysticercosis of the Brain: A CT Study. IJRI, 39: l, Feb. 1985,31 - 33.
17. Kieffer AS, Gold AHL: Intracranial Meningiomas. Neurology:| 19, Sep. 1969, 873 - 878.
18. Kingsley HP: The CT Scanner in Posterior Fossa Tumors of childhood. BJR 1979, 52, 769 - 776.
19. Latovitzki N, Clark C: Cerebral Cysticercosis. Neurology 28:Aug. 1978, 838 - 842.
20. Lee II.S, Rao C.V.G, Zimmerman R.A: Cranial MR1 and 3rd edn., McGraw Hill INC, 1992.
21. Locke ECl Biggers S; The Computed Tomographic appearance of Cerebral Cysticercosis in Adults and Children. Radiology 144: 819-823, Sep. 1982.

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