Single intracranial calcifications

1. benign (“physiologic”)

a) choroid plexus: calcifications usually bilateral

b) arachnoid granulation

c) diaphragma sellae

d) dural: common locations include falx (falcine) tentorial, sagittal sinus

e) habenular commissure

f) petroclinoid or interclinoid ligaments

g) pineal: 55% of patients>20 yrs of age have a calcified pineal gland visible on plain skull X-ray

2. infection

a) cysticercosis cyst: single or multiple, see Neurocysticercosis

b) encephalitis, meningitis, cerebral abscess (acute and healed)

c) granuloma (torulosis and other fungi)

d) hydatid cyst

e) tuberculoma

f) paragonimiasis

g) rubella

h) syphilitic gumma

3. vascular

a) aneurysm, including:

● vein of Galen aneurysm

● giant aneurysm

b) arteriosclerosis (especially carotid artery in siphon region)

c) hemangioma, AVM, Sturge-Weber syndrome

4. neoplastic: calcifications usually suggest a more benign process

a) meningioma

b) craniopharyngioma

c) choroid plexus papilloma

d) ependymoma

e) glioma (especially oligodendroglioma, also astrocytoma)

f) ganglioglioma

g) lipoma of corpus callosum

h) pinealoma

i) hamartoma of tuber cinereum

5. miscellaneous

a) hematoma: ICH, EDH or SDH. Calcifications usually only when chronic

b) idiopathic

c) tuberous sclerosis

Meningioma

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subependymoma

Intracranial aneurysm: the presence of calcification (independent of aneurysm size) has been shown to increase the likelihood of poor outcome [1].

Arachnoiditis ossificans

Osler-Weber-Rendu syndrome; May show a well-demarcated homogeneous or mottled high density [2] (high density due to hematoma, calcification, thrombosis, hemosiderin deposition, alterations in BBB, and/or increased blood volume [3] with some form of contrast enhancement (around or within lesion) in 17 of 24 patients [4]. Surrounding edema or mass effect is rare (except in cases that have recently hemorrhaged).

Intracranial lipoma CT: Low density, may have peripheral calcification (difficult to appreciate on MRI) [5].

Tuberous sclerosis complex: MRI: Low signal in subependymal lesions may represent calcification.

Tectal glioma:Calcification on CT has been described in 9–25% [6] [7].

Finded in Ganglioglioma Pathology. Tumor calcification are best appreciated on CT and are seen in 30%. Frequently appears cystic on CT, but still may be found to be solid at operation. MRI: T1WI low to iso-intense, variable enhancement. T2 weighted image hyperintense. Calcifications appear as low signal on both.

Plain skull X-ray: calcification may be noted.

Asymptomatic meningiomas with calcification seen on CT and/or Hypointensity on T2 weighted image MRI appeared to have a slower growth rate [8].

see Primary Familial Brain Calcification.

Craniopharyngioma: Calcification: microscopically 50%. Plain X-ray: 85% in childhood, 40% in adults. Osteogenic factor Bmp2 may play an important role in the calcification of adamantinomatous craniopharyngioma ACP via autocrine or paracrine mechanisms. Given the presence of osteogenic markers (Runx2 and Osterix), craniopharyngioma cells could differentiate into an osteoblast-like lineage, and the process of craniopharyngioma calcification resembles that which occurs in osteogenesis/odontogenesis [9].

Radiographic detection of calcification in pituitary neuroendocrine tumor is relatively rare, and the clinical characteristics of pituitary neuroendocrine tumor with calcification remain unclear.

A total of 160 patients who underwent surgical resection of pituitary neuroendocrine tumors between February 2004 and December 2016 were reviewed. Eighty-one patients had hormone-secreting pituitary neuroendocrine tumors, and 79 patients had nonfunctioning pituitary neuroendocrine tumor. Among these 160 patients, cases with radiological calcifications on preoperative neuroimaging were included in this study, and clinical characteristics with intraoperative findings were analyzed, retrospectively.

pituitary neuroendocrine tumor with calcification on preoperative neuroimaging was observed in only nine cases (5.6%). The study population consisted of these nine patients with nonfunctioning pituitary neuroendocrine tumor (n = 5), mixed growth hormone and prolactin-secreting pituitary neuroendocrine tumors (n = 3), and a prolactinoma (n = 1). In 89% of cases (n = 8), calcified pituitary

neuroendocrine tumor was soft enough for suction despite the presence of a granular gritty texture intraoperatively. Besides, in a single patient (11%), evidence of hard thick capsular calcification was seen surrounding a soft tumor component; however, it did not interfere with adequate removal of the soft part, and tumor resection was possible in all cases without any complications.

pituitary neuroendocrine tumor presenting with calcification is relatively rare, but should be kept in mind to avoid making a wrong preoperative diagnosis. As not all pituitary neuroendocrine tumors with calcification are hard tumors, preoperative radiological calcification should not affect decision-making regarding surgical indications [10].

Dementia associated with brain calcification may occur in Down syndrome and some cases of Fahr's disease (bilateral striatopallidodentate calcinosis). Basal ganglia calcification may occur in Nasu–Hakola disease resulting from TREM2 mutations, and punctate calcification of subcortical and deep white matter may occur in adult-onset leukodystrophy with axonal spheroids and pigmented glia resulting from CSF1R mutations. Brain calcification in hypoparathyroidism or pseudohypoparathyroidism may occasionally be associated with cognitive impairment. All these diagnostic possibilities were excluded by the clinical and investigation findings.

Chance concurrence of two separate disorders (dual pathology), FTD and idiopathic brain calcification, might explain this case. However, a plausible unifying diagnosis for this phenotype is Kosaka–Shibayama disease, or diffuse neurofibrillary tangles with calcification (DNTC). This rare disorder of unknown aetiology is reported almost exclusively from Japan [11].

Case reports

A patient with Parkinson's disease (PD) who was examined as a candidate for DBS was initially rejected due to extensive brain calcifications. Upon second opinion and planning of trajectories she underwent successful surgery. Genetic analyses identified a mutation in SLC20A2, a gene known to cause brain calcifications, but no mutation known to cause PD was found [12].

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