Cerebral Calcifications as a Differential Diagnosis of Psychiatric Disorders
Amir Mufaddel¹, Ossama T. Osman², Ghanem Al Hassani¹

1 Introduction

It is of vital importance to differentiate psychiatric symptoms secondary to organic causes from primary psychiatric disorders. Early diagnosis of the primary organic etiology is necessary for early intervention and for avoiding side effects of using long term psycho-tropic medications. This will be particularly helpful if the underlying cause is treatable and its treatment can lead to improvement in psychiatric symptoms. Organic psychiatric disorders are more likely if the patient is presenting with first episode of psychiatric symptoms, prominent cognitive symptoms or with clinical features that are not typical for functional psychiatric disorder. Several physical causes can contribute to the etiology of organic psychiatric disorders. Examples of such conditions include neurological conditions, infectious diseases, constipation, dehydration, pain and vascular causes.

Organic psychiatric disorders can present with different pictures including:

1. Delirium
2. Dementia
3. Other organic mental disorders: those are classified in ICD-10 as organic hallucinosis, organic catatonic disorder, organic delusional or schizophrenia-like disorder, organic mood disorder, organic anxiety disorder and organic personality disorder (Mufaddel et al., 2014b).

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Table 1 summarizes the differential diagnosis of psychiatric symptoms that can occur due to organic conditions. The table includes organic conditions that can lead to cerebral calcifications. It is important to consider that there are many other organic conditions that can present with psychiatric symptoms, but this chapter discusses only those associated with cerebral calcification.

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<tr>
<th>Psychiatric Condition</th>
<th>Possible Etiological Conditions with Cerebral Calcification</th>
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| Mood changes/Depression | **Intra-axial:** Temporal lobe tumors  
 Parietal lobe tumors  
 Thalamic lesions  
 Craniopharyngioma  
 Infections (brucellosis, toxoplasmosis)  
 Hypoparathyroidism  
 Fahr’s disease  
 Tuberous sclerosis  
 **Extra-axial calcifications:**  
 Frontal lobe meningioma  
 Gorlin –Goltz syndrome |
| Psychotic symptoms | **Intra-axial:**  
 Hypoparathyroidism  
 Fahr’s disease  
 Infections (influenza virus, HSV-1, congenital rubella)  
 **Extra-axial calcifications:**  
 Frontal lobe meningioma  
 Gorlin –Goltz syndrome |
| Personality changes | Extra-axial calcifications (Frontal lobe tumors such as meningioma) |
| Autism spectrum disorders | Tuberous sclerosis  
 Infections (congenital rubella, |
| ADHD | Sturge-Weber syndrome  
 Neurofibromatosis |
| Mental retardation | Tuberous sclerosis  
 Sturge-Weber syndrome  
 Neurofibromatosis  
 Craniopharyngioma.  
 Infections: Congenital toxoplasmosis, CMV infection |
| Dementia | Vascular lesions  
 Hypoparathyroidism |

**Table 1**: Differential diagnosis of psychiatric conditions that can be associated with calcified cerebral lesions.
Radiological investigations are useful tools to exclude organic pathology in patients presenting with psychiatric symptoms. One of the possible radiological findings that can indicate presence of current or previous organic pathology contributing to the clinical psychiatric presentation is the presence of cerebral calcifications which can occur in a wide range of conditions with different etiologies. Calcifications can occur as physiologic, dystrophic, congenital or vascular calcifications. For psychiatric patients who present with cerebral calcifications, the location of calcification and the clinical psychiatric and systemic presentations are important in establishing a final diagnosis (Mufaddel & Al Hassani, 2014b).

Intracranial calcifications are frequent findings on radiological brain examinations. It is sometimes difficult to conclude whether such calcifications are of clinical significance or are just incidental findings particularly when the lesions are not clearly explaining the clinical picture. This is particularly applied when the presenting complaint is of only psychopathological nature.

In this chapter, the differential diagnosis of brain calcifications will be discussed in relation to the possible psychiatric presentations reported in the literature.

Based on their location, cerebral calcifications can be divided into extra-axial and intra-axial calcifications (Table 2). Intra-axial calcifications occur within the brain parenchyma; and extra-axial calcifications are external to the brain parenchyma. Examples of structures involved in extra-axial calcifications are: falx cerebri and the pineal gland. Intraventricular calcifications are discussed as intra-axial calcifications but they are sometimes considered as a third type of cerebral calcification. Structures commonly involved in intra-axial clarifications are the basal ganglia and the cerebellum. Causes of intra-axial calcifications include neoplasms (e.g. oligodendrogliomas and astrocytomas), vascular causes (e.g. angiomatous malformations and aneurysms), Infectious (e.g. congenital childhood infections, and parasitic infections such as neurocysticercosis and cerebral hydatid cyst disease), congenital causes (e.g. tuberous sclerosis); and endocrine/metabolic causes (e.g. hypoparathyroidism, and hyperparathyroidism) (Celzo et al., 2013; Makariou & Patsalides, 2009).

Both acquired and congenital infections can lead to intracranial calcifications. Example of infections causing calcifications includes TORCH infections (toxoplasmosis, other, rubella, cytomegalovirus, herpes simplex virus). Metabolic disorders that affect calcium homeostasis can lead to calcifications predominantly involving the basal ganglia. Inflammatory lesions, such as sarcoidosis and tumors, may also lead to cerebral calcifications (Celzo et al., 2013; Makariou & Patsalides, 2009).

The basal ganglia calcifications are usually seen in the globus pallidus, the head of the caudate nucleus, and the putamen and they commonly occur in middle-aged and the elderly subjects. Brain calcifications are interpreted as incidental findings of no significance by some clinicians. However, individuals with brain calcifications, particularly those below the age of 30 years, should be carefully evaluated for underlying etiologies (Celzo et al., 2013; Makariou & Patsalides, 2009).
### Extra-axial Calcifications

**Structures Involved:**
- Falx cerebri
- The pineal gland
- Choroid plexus
- Habenula
- Dura and arachnoid
- Tentorium cerebelli
- Superior sagittal sinus
- Petroclinoid and interclinoid ligaments
- Arachnoid granulations

**Causes:**
- Meningiomas
- Dural osteomas
- Calcifying tumours
- Exaggerated physiological calcifications

### Intra-axial Calcifications

**Structures involved:**
- Basal ganglia
- Cerebellum

**Causes:**
- Neoplastic
  - Oligodendrogiomas
  - Astrocytomas
  - Medulloblastomas
  - Other primary brain tumours.
  - Metastatic tumours
- Vascular
  - Angiomatic malformations
  - Arteriovenous malformations
  - Dystrophic calcification in chronic infarction
  - Chronic vasculitis
  - Aneurysms
- Infectious
  - Congenital childhood infections, particularly the ‘TORCH’
  - Tuberculosis
  - Parasitic infections such as neurocysticercosis and cerebral hydatid cyst disease
- Congenital:
  - Sturge-Weber syndrome
  - Tuberous sclerosis
  - Lipomas
  - Neurofibromatosis
- Endocrine/metabolic
  - Diabetes mellitus
  - Hypoparathyroidism
  - Pseudohypoparathyroidism
  - Hyperparathyroidism
- Idiopathic/genetic:
  - Familial idiopathic basal ganglia calcification.

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**Table 2:** Differential diagnosis of extra- and intra-axial cerebral calcifications.
2 Extra-axial Calcifications:

Extra-axial cerebral calcifications are commonly caused by meningiomas, dural osteomas, calcifying tumours, and physiological calcifications (Celzo et al., 2013). Some rare conditions with multi-system involvement can also be associated with extra-axial calcifications such as that occurring in Gorlin-Goltz syndrome with characteristic falx-cerebri calcification (Mufaddel et al., 2014a). Anatomical locations for extra-axial calcifications are shown in Figure 1.

![Figure 1: Anatomical locations for extra-axial calcifications.](image)

Based on etiology, the differential diagnosis of extra-axial calcification in relation to psychiatric presentations is discussed below.

2.1 Extra-axial Neoplasm

Frontal meningiomas can compress the frontal lobes externally, and if they are large in size they can lead to personality and intellectual changes. They are sometimes seen by psychiatrists before the diagnosis of meningioma is established due to the nature of their psychiatric symptoms. Delayed diagnosis of frontal meningiomas presenting with psychiatric features has been reported in several cases presenting with longstanding history of visual hallucinations and personality change. Other presenting symptoms can occur such as headache and visual loss; and such patients are sometimes misdiagnosed as conversion disorder (Panzer et al., 1991). Some patients may suffer from headache preceding or during the psychiatric symptoms and others may later develop epilepsy. Other psychiatric presentations of frontal meningiomas include symptoms resembling depression, anxiety, hypomania, and schizophrenia. Surgical treatment was associated with improvement of symptoms and even improved level of functioning in some cases.

Severe psychiatric symptoms and epilepsy have also been reported as presenting
symptoms in patients with huge osteoma in the anterior cranial fossa (Hudolin et al., 1961).

### 2.2 Gorlin-Goltz Syndrome

Gorlin-Goltz syndrome is an autosomal dominant syndrome with multiple and diverse clinical features that involve the nervous system, skin, eyes, endocrine system, and bones. In 1960, Gorlin and Goltz reported the triad that characterized the diagnosis of Gorlin-Goltz syndrome, the triad included the presence of nevoid basal cell carcinoma, kerato-cystic odontogenic tumors in the jaws, and bifid ribs. There are several other clinical features of this syndrome including calcification of the falx cerebri, facial milia, palmar and plantar epidermal pits, spine and rib anomalies, relative macrocephaly, medulloblastomas, frontal bossing, cleft lip and/or palate, ocular malformation, and developmental malformations (Gorlin & Goltz, 1960; Casaroto et al., 2011). Gorlin-Goltz syndrome is diagnosed by the presence of either two major criteria or one major and two minor criteria. The major criteria:

1. Multiple (>2) basal cell carcinomas (or one if under 20 years of age).
2. Odontogenic keratocysts of the jaws proven by histopathology.
3. Three or more palmar or plantar pits.
4. Bilamellar calcification of the falx cerebri.
5. Bifid, fused or markedly splayed ribs.
6. First-degree relatives with nevoid basal cell carcinoma.

The minor criteria:

1. Macrocephal
2. Frontal bossing, cleft lip/palate, pectus, and syndactyly of digits.
3. Sprengel deformity, pectus, and syndactyly of digits.
5. Ovarian fibroma
6. Medulloplastoma

Cases with complicated multiple clinical presentations have been reported with history of pleomorphic psychiatric features, basal cell carcinoma, low vitamin-D level, high parathyroid hormone levels, and extensive calcification along falx cerebri and around the cerebellar vermis. Possible presenting psychiatric symptoms include irritability, aggressive behavior, labile mood, hallucinations, paranoid delusions, and transient cognitive impairment (Mufaddel et al., 2014a). Figures 2, 3 & 4: show palmar pits, falx cerebri calcification, & cerebellar vermis calcifications in a patient with Gorlin-Goltz syndrome who presented with psychiatric symptoms.
Figure 2: Palmar pits in a patient with Gorlin-Goltz syndrome (Mufaddel et al., 2014a).

Figure 3: Falx cerebri calcification in a patient with Gorlin-Goltz syndrome (Mufaddel et al., 2014a).

Figure 4: Cerebellar calcification in a patient with Gorlin-Goltz syndrome (Mufaddel et al., 2014a).
3 Physiologic calcifications

Physiologic cerebral calcifications are likely if they are not associated with any evidence of disease and have no demonstrable pathological cause. They most commonly occur in the pineal gland, habenula, choroid plexus, basal ganglia, falk, tentorium, petroclinoid ligaments and sagittal sinus. The size of calcification and age at presentation should be considered before concluding that the calcification is physiologic (Kıroğlu et al., 2010).

4 Intra-axial calcifications

Intra-axial calcifications have several etiologies including neoplasm, vascular causes, infections, congenital disorders, and endocrine/metabolic causes. They could also be idiopathic such as that occurring in Fahr’s disease. Anatomical locations for intra-axial calcifications are shown in Figure 5. Based on etiology, the differential diagnosis of intra-axial calcification in relation to psychiatric presentations is discussed below.

![Figure 5: Anatomical locations and causes of intra-axial calcifications.](image)

4.1 Neoplastic Causes

Tumors that are commonly associated with intracranial calcifications include oligodendrogliomas, astrocytomas, craniopharyngiomas, meningiomas, pineal gland tumors and
ependymomas. In some instances, the presence of calcification and its pattern can be pathognomonic in some tumors such as oligodendrogliomas and craniopharyngiomas (Makariou & Patsalides, 2009; Celzo et al., 2013).

In some cases, psychiatric symptoms can be the first presenting symptoms of brain tumours in the absence of neurological signs. For example, incidental MRI findings of thalamic tumor have been reported in patients presenting only with psychiatric symptoms. Patients may present with mood change, psychotic symptoms, panic attacks, personality changes, or memory problems. (Moise & Madhusoodanan, 2006). In other cases, neurological signs can be minimal and the psychiatric symptoms are more prominent. Example of these is parietal lobe tumors which can present with depression accompanied by minimal neurological. (Madhusoodanan et al., 2004). Therefore, radiological investigations are necessary for early detection of possible brain tumors in individuals presenting with psychiatric symptoms particularly in those presenting with new symptoms, atypical presentation or treatment-resistant psychiatric symptoms.

No clear association could currently be confirmed between the nature of psychiatric symptoms and the location of tumor or its histological type (Madhusoodanan et al., 2007). However, mood disorders and schizophrenia-like conditions can be related to right and left hemispheres dysfunctions, respectively. Lesions located in the temporal lobes are commonly associated with depression (Uribe, 1986).

Cranioopharyngiomas are locally invasive and are frequently recurrent and they are associated with neurological and endocrinological dysfunction. Most studies regarding psychiatric sequale of craniopharyngiomas were conducted in patients who have been treated for the tumor. Children treated prior to the age of 18 years had an overall neuro-behavioral dysfunction in 57% of cases including social impairment (41%), school dysfunction (35%), and emotional/affective dysfunction with primarily depressive symptoms (40%) (Zada et al., 2013).

4.2 Vascular Disorders

Vascular calcification can be due to atherosclerosis, aneurysm, arteriovenous malformation or cavernous malformation. Atherosclerotic calcifications could be related to cognitive dysfunction and to brain changes on MRI examination. Larger volumes of calcification are associated with lower cognitive scores and smaller total brain volumes (Bos et al., 2012).

Vascular disorders are associated with developing neurodegenerative conditions such as Alzheimer’s disease, multiple sclerosis and Huntington’s. Cerebrovascular disease may also lead to vascular type of dementia which is the second commonest cause of dementia. It usually occurs in the seventh and eighth decades with a relatively acute onset and it might follow stroke. It may be difficult to establish a clear diagnosis of vascular dementia in the absence of history of stroke or localizing neurological signs.

High risk of dementia is associated with larger calcification volume in all vessels, except in the coronary vessels. Also extra-cranial calcification of carotid artery is significantly associated with a higher risk of dementia. Similarly, this finding remains also significant for Alzheimer’s disease (Bos et al., 2014).
Alzheimer’s disease is primarily related to the hippocampus as brain tissue develops neuro-degeneration with characteristic tau and amyloid protein deposits. Clinical features of Alzheimer’s disease are memory impairment with gradual onset and continuing decline. Aphasia, apraxia, agnosia and disturbances in executive functioning may occur. Findings on CT or MRI characteristically show hippocampal atrophy and ventricular enlargement. Microscopic neuropathological features of neurofibrillary tangles and senile plaques (amyloid plaques) are cardinal diagnostic features.

One of the hypotheses aimed to explaining the pathological changes in Alzheimer’s disease suggest possible breakdown of blood brain barrier (BBB) following traumatic brain injury. BBB leakage can occur secondarily to abnormal brain activity with associated increase in the number of endothelial caveolae, leading to transcytosis of plasma proteins and reduction of tight junction proteins (Franzblau et al., 2013).

4.3 Infections

Both acquired and congenital infections can be associated with cerebral calcifications as well as psychiatric symptoms which can be either acute or chronic symptoms. Psychiatric symptoms can be the initial clinical presentation of systemic and central nervous system infections, and they can occur in the absence of neurological symptoms in some disorders as in some cases of viral encephalitis. Mood symptoms can occur secondary to brucellosis or toxoplasmosis. Late-onset neuropsychiatric complications, occurring several years following the infection, have also been reported such as in the case of subacute sclerosing panencephalitis due to measles. Some Infectious diseases are thought to have possible etiological role for major psychiatric disorders such as schizophrenia (e.g. Influenza virus and HSV-1), (Mufaddel et al., 2014b).

The most common acquired intracranial infections that are typically associated with intracranial calcifications include: cysticercosis, tuberculosis, HIV and cryptococcus infections (Makariou & Patsalides, 2009).

Congenital anomalies are caused by perinatal infections in 2% to 3% of cases. TORCH (Toxoplasmosis, Other, Rubella, Cytomegalovirus (CMV), and Herpes infections), are among the most common infections that can lead to congenital anomalies (Stegmann & Carey, 2002).

Congenital toxoplasmosis usually presents with the classic triad of chorioretinitis, hydrocephalus, and intracranial calcifications. Other variety of symptoms can also occur and systemic manifestations may include fever, hepatomegaly, splenomegaly, jaundice, lymphadenopathy, anemia and abnormal spinal fluid (Halonen & Weis, 2013). Congenital toxoplasmosis can lead to brain and eye tissues abnormalities including destruction or remodeling of the white substance and blockade of the aqueduct of Sylvius by infected necrotized foci which may further calcify. Sequelae of congenital toxoplasmosis include mental retardation, psychomotor abnormalities, seizures, deafness, microcephalus, and hydrocephalus (Robert-Gangneux & Dardé, 2012).

Congenital cytomegalovirus (CMV) infection is one of the most common viral causes of congenital infections with incidence that varies between 0.15% and 2.0% (Gaytiant et al., 2002). CMV infection can be associated with mental retardation, cerebral palsy,
psychomotor retardation and sensorineural hearing loss. These complications are often irreversible even with antiviral treatment. Abnormal CT findings were reported in 70% of symptomatic children with CMV with intracerebral calcifications being the most common finding. Most of those who had abnormal CT scan findings during neonatal period (90%) developed at least one long-term sequel. About 50% of children with CT abnormalities had an IQ < 50, (Boppana et al., 1997). Other radiological features include enlarged ventricles, white matter abnormalities, polymicrogyria, cysts, structural changes, and extensive encephalopathy (Cheeran et al., 2009).

Few studies have been conducted to investigate psychiatric manifestations in individuals with congenital rubella. There are conflicting results and views regarding the association between congenital rubella and developing autism and mental retardation (Chess, 1971). Congenital rubella is commonly associated with CNS manifestations including hearing loss and psychomotor retardation. Psychiatric manifestations have been reported in up to 50% of cases (Rorke, 1973). One study investigated the radiological findings in adult deaf patients with schizophrenia-like symptoms and documented prenatal rubella virus infection and compared them with controls. The study concluded that there are abnormal white matter lesions which may correspond to neurovascular lesions but they do not appear to be directly related to schizophrenia-like symptoms (Lane et al., 1996). Calcifications due to congenital rubella are commonly located in the periventricular white matter, basal ganglia, and brain stem (Kiroğlu et al., 2010).

Neonatal herpes simplex encephalitis is accompanied by early rapid atrophic changes which may be evident in the 3d week. Late findings may include cortical atrophy and calcification with variety of distributions ranging from punctate to an extensive gyral pattern with possible involvement of the cerebellum (Noorbhesht et al., 1987). Case control studies suggest a significant association between presence of maternal antibodies to HSV2 glycoprotein gG2 and developing subsequent psychotic illness (Buka et al., 2001).

4.4 Congenital Disorders

Cerebral calcification in congenital disorders is frequently seen in Sturge-Weber syndrome, tuberous sclerosis, neurofibromatosis, intracranial lipoma, Cockayne and Gorlin syndromes.

4.4.1 Tuberous sclerosis

Tuberous sclerosis is an autosomal dominant condition that presents with adenoma sebaceum, epilepsy, retinal Phakomas, subungualfibromata, white skin patches, shagreen-skin and cognitive impairment. It can be associated with multiple tumours in kidneys, spleen and lungs. Cognitive impairment is often severe and learning disability occurs in 38%-64% of cases (Gelder et al., 2004; Gillberg et al. 1994; Kumar et al., 2005; Webb et al., 1991).

Autism and broadly-defined features of pervasive developmental disorders are common in patients with tuberous sclerosis. One study suggested a prevalence of 24% and 19% respectively. Autistic features were found more common in females than males.
with tuberous sclerosis (Hunt & Shephred, 1993).

Epidemiological studies have shown that individuals with tuberous sclerosis complex had mental retardation and autistic-like pervasive developmental disorders with a prevalence of 50-60% and 43-86% respectively. On the other hand, children with autism have tuberous sclerosis complex existing in 1% of cases (Harrison & Bolton, 1997).

There are few established medical causes of autism spectrum disorder. One of these causes is tuberous sclerosis which is a unique neurogenetic model for investigating the brain basis of the syndrome (Bolton et al., 2002).

Factors that may have greater likelihood of developing an autism spectrum disorder in children with tuberous sclerosis include: mutation in the TSC2, early-onset infantile and presence of an epileptiform focus in the temporal lobes (Bolton, 2004).

Other psychiatric symptoms include psychosis, anxiety and depression. Childhood-onset mood disorders were also reported in some cases (Chopra et al., 2006).

4.4.2 Sturge-Weber syndrome

Sturge-Weber Syndrome (encephalofacialangiomatosis) is a congenital disorder with exceptional familial occurrence. There is capillary malformations involving the skin, eye and the brain. Skin involvement usually presents with port-wine naevus involving one side of the face in the distribution of a fifth nerve division. Brain involvement presents with leptomeningeal angioma which tends to involve the parietal and occipital lobes (Kumar et al., 2005). Eye involvement may lead to glaucoma. Computed tomography and contrast-enhance MRI are helpful in the diagnosis of brain involvement. Radiological features are common in patients with Sturge-Weber syndrome who have facial involvement. These are commonly involving the frontal and parietal lobes and less commonly occipital lobe brain involvement. When the port-wine nevus is bilateral, there will be a greater risk of brain involvement (Crosley & Binet, 1978; Taly et al., 1987).

Neuropsychiatric features that are commonly associated with Sturge-Weber syndrome include epilepsy, cognitive symptoms, attention deficit hyperkinetic disorders, headache, hemiparesis, and visual field defects (Lo et al. 2012).

Small study including 16 patients with Sturge-Weber syndrome has shown that they have psychiatric diagnoses including mood disorder (31%), disruptive behaviour (25%), and adjustment disorder (25%). Substance-related disorders were most frequently found in adults (67%), (Turin et al., 2010).

Epilepsy, hemiparesis, mental retardation and ocular problems were found the most common and the most severe presentations in one series of 55 patients with Sturge-Weber syndrome. Cerebral lesions were associated a progressive course during childhood. Surgical treatment has shown benefits in controlling seizures but it has poor benefits in hemiparesis and intellectual deficits (Pascual-Castroviejo et al., 2008).

4.4.3 Neurofibromatosis (NF)

Neurofibromatosis (Von Recklinghausen’s disease) was described in 1882 by Friedrich
Daniel Von Recklinghausen. It is characterized by multiple skin neurofibromas and pigmentation due to neuroectodermal abnormality. It has several clinical symptoms involving the skin, nervous system, bones, eyes and other sites. There are two clinical types of NF including peripheral type (NF1) and central type (NF2) (Kumar et al., 2005).

NF1 is more frequent than NF2 and presents as subcutaneous, soft, and sometimes pedunculated tumors which increase in number. The gene responsible for NF1 is located on the long arm of chromosome 17 (17q11.2) which encodes a protein called neurofibromin which is expressed in neurons, Schwann cells, oligodendrocytes and astrocytes. Learning disabilities may occur due to NF1 mutation (Antônio et al., 2013).

Recent studies suggest slight increase in the frequency of mental retardation in children with NF1 (North et al., 2002).

Psychiatric and cognitive disorders are more often encountered in patients with NF1 than NF2. Specific learning disabilities exist in only 20% of children with NF1; and their neuropsychological profile indicates deficits in perceptual skills (visuospatial and visuoperceptual), executive functioning, and attention (sustained and switching). Difficulties in sustained attention were present in 63% of children with NF1, with 38% fulfilling criteria for attention deficit-hyperactivity disorder (Hyman et al., 2005).

The abnormal gene responsible for NF2 is located on chromosome 22(q122), and the abnormal protein is merlin or schwannomin which is a cytoskeletal protein. Several tumors may occur in NF2 such as meningioma, acoustic neuroma, lexi-form neuroma, glioma and cutaneous neurofibroma (Evans et al., 2000).

### 4.5 Metabolic Causes

Basal ganglia and subcortical calcifications may occur in patients with chronic renal failure and secondary hyperparathyroidism. Calcification due to hypoparathyroidism typically involve the basal ganglia, thalami, and the cerebellum. Cerebral calcifications are more common pseudohypoparathyroidism than idiopathic hypoparathyroidism. Hypothyroidism can be associated with basal ganglia and cerebellar calcifications (Makariou & Patsalides, 2009). Psychiatric features in hypoparathyroidism include depression, anxiety, emotional lability, confusion, and psychosis (Hossain, 1970).

Cognitive deficits commonly occur in patients with chronic hypoparathyroidism with positive correlation between symptoms of cognitive dysfunction and the presence of cerebral calcification (Kowdley et al., 1999). Neuropsychological dysfunctions were in 35.5 % of patients with idiopathic hypoparathyroidism; and they correlated with illness duration, female gender, and serum calcium but not with intracranial calcifications (Aggarwal et al., 2013).

Elderly patients with hypoparathyroidism and associated calcifications can be misdiagnosed as cases of senile dementia. Symptoms of dementia that occur due to hypoparathyroidism are treatable and should be considered in the differential diagnosis of dementia (Katsidzira et al., 2010).
4.6 Familial Idiopathic Basal Ganglia Calcification (Fahr’s disease)

Fahr’s disease is a rare neurodegenerative disorder which is characterized by the presence of symmetrical and bilateral calcification of the basal ganglia. Calcifications were also reported in other brain regions such as dentate nucleus, thalamus and cerebral cortex. Familial and non-familial cases of the disease have been reported, with predominantly autosomal-dominant fashion. Neuropsychiatric features and movement disorders are the common presenting clinical features (Mufaddel & Al Hassani, 2014). The clinical features of Fahr’s disease are shown in Table 3, and the criteria for the diagnosis are shown in Figure 6.

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<tr>
<td>Radiological Findings</td>
<td>Bilateral symmetrical calcifications of basal ganglia and dentate nucleus. Other sites of calcifications: thalamus, Centrum semi-ovale, cerebellum and cerebral white matter.</td>
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Table 3: Clinical presentations of Fahr’s disease (Mufaddel & Al-Hassani, 2014).

References


Figure 6: Criteria for the diagnosis of Fahr’s disease (Mufaddel & Hassani, 2014).


Bos, D., Vernooij, M.W., de Bruijn, R.F., Koudstaal, P.G., Hofman, A., et al. (2014). Atherosclerotic calcification is related to a higher risk of dementia and cognitive decline. Alzheimers Dement


