

**Original Article**

**Prevalence of incidental basal ganglia calcification on routine brain computed tomography**

Abdelrahman M. Radaideh, Duraid M. Jaradat, Fares H. Haddad

Princess Basma Teaching Hospital, Irbid, Jordan, and King Hussein Medical Center, Amman, Jordan

**ABSTRACT**

**Objective**

To assess the prevalence of incidental basal ganglia calcifications among patients having a brain computed tomography (CT) scan for non-related causes.

**Methods**

All brain CT scan performed at Princess Basma Hospital from February 2006 to July 2006 were retrospectively reviewed and interpreted by the same neuro-radiologist. The cohort was divided into two groups according to gender and three groups according to age groups. Blood was withdrawn only for those with brain calcifications for complete blood count, kidney function tests, calcium profile, vitamin D level and parathyroid hormone.

**Results**

A total of 1040 CT scans were included in the study; 552 were males. The overall prevalence of basal ganglia calcifications was 1.25 %.{ 0.72% for males vs.1.85% for females; Odds Ratio=2.57 (0.72-9.98), Relative Risk=2.55 (0.79-8.21), p=0.1}. The prevalence increased with increasing age; being 0.6% in younger age group vs. 2.4% in those above 60 years. Elevated parathyroid hormone was found in 6 patients; of whom only one proved to have low vitamin D<sub>3</sub> level.

## **Conclusion**

The prevalence of basal ganglia calcifications was quite low in this cohort and showed a steep increase with increasing age. High parathyroid hormone was one of the important biochemical abnormalities that warrant further evaluation and investigation in a large cross sectional cohort to identify direct cause-effect relationship. (Rawal Med J 2012;37:6-8).

## **Keywords**

Basal ganglia, basal ganglia calcification, parathyroid hormone.

## **INTRODUCTION**

The initial description of microscopic perivascular calcification within the basal ganglia was reported by Virchow and Bamberger in 1855.<sup>1</sup> Basal Ganglia Calcification (BGC) is a nonspecific finding in many medical conditions, including infectious, metabolic, and genetic syndromes. In addition, such calcification is observed as an incidental finding in approximately 0.7 to 1.2% of CT scans.<sup>2</sup> These incidental calcifications are usually benign and have no clear etiology, especially in patients over 60 years of age.<sup>3</sup> Majority of BGC are idiopathic in nature and disturbances of calcium metabolism are so rare that biochemical testing is performed only if indicated by other features.

Physiological intracranial calcification occurs in about 0.3-1.5% of cases, is asymptomatic and is detected incidentally by neuro-imaging.<sup>4</sup> Pathological BGC is due to various causes, such as metabolic disorders, infectious and genetic diseases and others.<sup>4-8</sup> Hypoparathyroidism and pseudohypoparathyroidism are the most common causes of pathological BGC.<sup>3,5</sup> Harrington et al reported BGC prevalence of 0.6% and out of those only two patients had parathyroid disorder.<sup>7</sup> CT scan is 5-15 times as sensitive as plain skull radiography in detecting intracranial calcification.<sup>7,9</sup> Other causes include mitochondrial cytopathy, infectious diseases such as cytomegalovirus, Epstein-Barr virus, toxoplasmosis, tuberculosis and acquired immunodeficiency syndrome.<sup>7,8</sup> This may also be seen in familial condition (Fahr's syndrome).<sup>10</sup> Magnetic Resonance imaging (MRI) may show much more extensive increased

signal on T1-weighted images than revealed by CT.<sup>9</sup> The aim of this study was to assess the prevalence of BGC among patients having a brain CT scan for non-related causes at our institution.

## **MATERIALS AND METHODS**

The study was conducted from February, 1<sup>st</sup> 2006 to July 1<sup>st</sup>, 2006 at Princess Basma Teaching Hospital in Jordan. All adult subjects who underwent brain CT for different causes were reviewed for the presence of BGC. Those known to have brain calcifications or had parathyroid dysfunction were excluded. The scans were reviewed and interpreted by the same neuro-radiologist. Scans were obtained using a CT unit Toshiba X vision GX with transverse sections of 5 mm thickness at an angle of 15-20° to the orbitomeatal line. Only those patients in whom CT showed BGC underwent history revision, clinical and laboratory investigations including a kidney function test, calcium profile, vitamin D level and parathyroid hormone (PTH).

Patients were divided into two groups according to gender and age by two decades. Verbal consent was obtained from all patients and the approval of Ethics committee of Prince Basma hospital was obtained prior to initiation of the study. The prevalence was calculated using simple percentages. Odd ratio and relative risks were calculated using chi square and Epiinfo 6 program. A p value of <0.05 was considered as statistically significant.

## **RESULTS**

Out of 1040 patients (552 males, 488 females), BGC were detected in 13 patients (9 females and 4 males) giving a total prevalence of 1.25%. The prevalence was higher in females vs. males {1.84% vs 0.72%, Odd Ratio=2.57 (0.72-9.98) and Relative Risk=2.55 (0.79-8.21), p=0.1}. The distribution of cases according to age groups and gender are shown in Table 1.

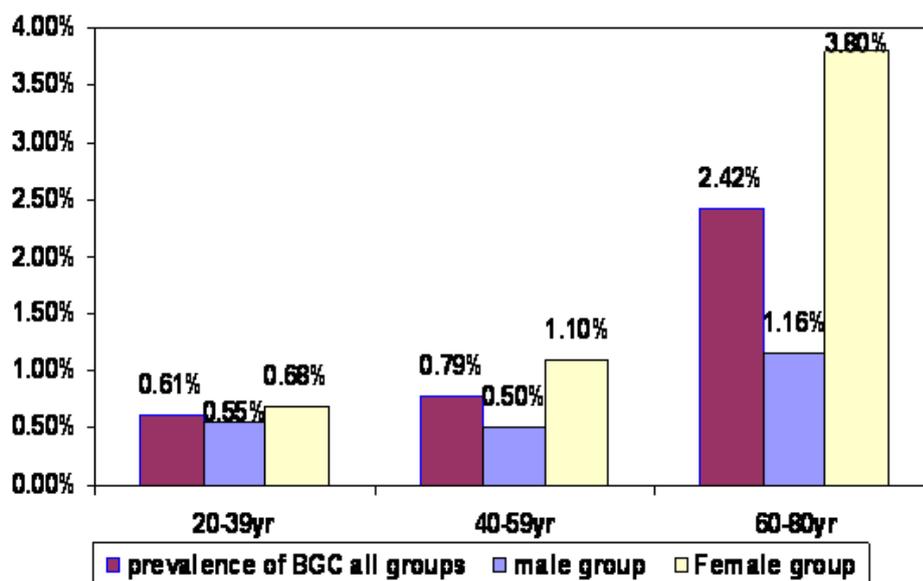
**Table 1. Distrubution and prevalence of BGC according to age and gender.**

Age group/ years	Number (males)	Males n=552	Female n=488	Total n=1040
<b>20-39</b>	327(179)	1	1	2
<b>40-59</b>	382(200)	1	2	3
<b>60-80</b>	331(173)	2	6	8
<b>Total*</b>	1040	4 (0.72%)	9 (1.84%)	13 (1.25%)

\*Odd Ratio =2.57(0.72-9.98). Relative Risk=2.55(0.79-8.21). p=0.1 male vs. females.

Bilateral brain calcifications were found 12 patients. Ten patients had calcifications in globus pallidus and in two patients calcifications were in the putamen (male 45 years old with persistent headache and female 33 years old with psychiatric disorder). Faint calcifications were found in 7 patients (4 females, 3 males), mottled calcification in 2 females and heavy calcifications in 3 patients (2 males and 1 female of the age group 60-80 years).

**Fig 1. Prevalence of basal ganglia calcification.**



The prevalence of brain calcification increased with increasing age (Fig 1). Although older females had more BGC than males (3.82% vs.1.16%), this did not reach statistical significance (p=0.11).

Six patients had elevation PTH between 90-230pg/ml (normal 10-69pg /ml); all of them had normal calcium and phosphorus profile. One female of 47 years age had raised alkaline phosphatase of 323 U/ L (normal up to 290) and low vitamin D level and was considered to have secondary hyperparathyroidism. No obvious cause for the elevated PTH was found in rest of patients.

## **DISCUSSION**

This is the first study of incidental intracranial calcification in our part of the world or Jordan. BGC were found in 13 patients out of 1040 giving a total prevalence of 1.25%; only one of the 13 patients had a documented metabolic abnormality which may have been a cause of pathological calcification. Hyperparathyroidism, either primary or secondary, may be associated with BGC in addition to old age and vitamin D deficiency,<sup>11</sup> thus more investigations must be done to identify these cases. Although Jordan is a sunny area nevertheless is a known country for vitamin D deficiency and this should be considered a top differential diagnosis.<sup>12</sup> In rare cases, idiopathic BGC syndrome consisting of bilateral BGC, neuropsychiatric abnormalities, disturbances of movement and normal calcium and phosphorus metabolism should be considered.<sup>13</sup>

CT scan is 5 to 15 times more sensitive in detection of BGC than conventional skull radiographs.<sup>9,14</sup> In younger age patients, unilateral BGC should be reevaluated for extra pyramidal symptoms and for localized pathological process.<sup>7</sup> In our series, none of our patients exhibited such a condition. The cause of secondary hyperparathyroidism in the single patient who was found to be vitamin D deficient without any evidence of malabsorptive disease, suggest that all other patients with raised PTH, vitamin D should be tested to rule out its deficiency.<sup>11,12</sup>

Legido et al reviewed 6,428 head CT scans in pediatric group and found BGC in 48 (1.1%) patients.<sup>15</sup> Neurologic symptoms were common in children of all groups, but could not be correlated to BGC related. Calcium and phosphorus metabolism was evaluated in 19 patients and was abnormal in only one.<sup>15</sup> Our prevalence rate was similar to that shown in other studies<sup>5,6,16,17</sup> and that females had a non-significantly higher BGC. It also showed that the BGC rates increased with increasing age. This, along with the risk of having parathyroid dysfunction, is also similar to those reported in the literature.<sup>16</sup> The limitations of study include no long term follow up of those patient with lesions and no calcium profile, parathyroid hormone or vitamin D level were tested in all patients.

## CONCLUSION

We found that the prevalence of basal ganglia calcifications was quite low in this cohort and showed a steep increase with increasing age. Vitamin D abnormalities with associated secondary hyperparathyroidism should be in the differential diagnosis.

Correspondence: Fares Halim Haddad. Email: haddf@hotmail.com Rec. Date: Oct 08, 2011 Accept Date: Jan 10, 2012
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