European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 09, Issue 07, 2022

MINERALIZING ANGIOPATHY IN STROKE PATIENTS OF PEDIATRIC AGE GROUP

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ABSTRACT

Background: Basal ganglia ischemic stroke accounts for a significant proportion of all childhood strokes. Trauma and bilateral basal ganglia calcification are its most frequent causes. It has been found that basal ganglia calcification is in fact the manifestation of mineralizing angiopathy of lenticulostriate arteries.

Objective: This study aims to evaluate the relationship between mineral calcification in the lenticulostriate arteries and acute infarcts in Indian population of age group new-born to 12 years.

Methods: A retrospective study of 23 children with basal ganglia infarcts detected on MRI was conducted at Dr. D.Y. Patil Medical Hospital, Pune.

Results: Maximum number of patients with evidence of basal ganglia infarcts belonged to the age group of 3-6 years. There was evidence of basal ganglia calcification in 40% patients. The presence of the calcification in the lenticulostriate arteries was confirmed on CT scan. MRI brain appeared to be less reliable in detection of calcific focus in the lenticulostriate arteries. However, calcification could be appreciated on phase sequences in a few patients.

Conclusion: Our study was able to provide evidence that mineralizing angiopathy of lenticulostriate arteries has a strong association with acute infarcts in the basal ganglia region in pediatric population.

Keywords: Pediatric stroke, Basal ganglia, mineralizing angiopathy, lenticulostriate arteries

INTRODUCTION

Childhood strokes form a rare entity, but can contribute significantly to pediatric morbidity and mortality. Unlike stroke in adults, whose main most common causes are atherosclerosis and hypertension, childhood stroke differs significantly in their etiology.
[1, 2].
Basal ganglia ischemic stroke accounts for a significant proportion of all childhood strokes. Trauma and bilateral basal ganglia calcification are its most frequent causes. Other causes include arteriovenous malformations, infection, cerebral vasculitis, hematologic causes such as sickle cell disease [3] and heart disease.
The lenticulostriate arteries arising from the initial part M1 of the middle cerebral artery, supply the basal ganglia. It has been found that basal ganglia calcification is in fact the manifestation of mineralizing angiopathy of lenticulostriate arteries [4]. Our study aims to establish the relationship between the presence of mineralizing angiopathy in the lenticulostriate arteries and infarcts in the basal ganglia region in pediatric population. This study aims to evaluate the relationship between mineral calcification in the lenticulostriate arteries and acute infarcts in Indian population of age group new born to 12 years.

METHODOLOGY

23 children with basal ganglia infarcts detected on MRI were selected. This study was conducted at Dr. D.Y. Patil medical hospital, Pune. MRI was done on Siemens Magnetom Vida 3T and Magnetom Avanto 1.5 T. CT images were taken on Philips ingenuity core (128 slices). Presence of calcification in unilateral/bilateral basal ganglia were checked on SWI sequence on 1.5 and 3Tesla MRI and follow up CT images were taken to confirm the presence of calcium in the lenticulostriate arteries of the affected region, wherever possible. MRI may not be completely trusted in this respect.
Complete history of the patient was taken for any antenatal infection, perinatal hypoxia, other major risk factors. It included vascular causes such as arterial dissections, moyamoya disease; cardiac conditions like congenital or acquired heart diseases; haematological disorders and prothrombotic states such as factor V Leiden mutation, protein C deficiency, protein S deficiency, antithrombin III deficiency, and homocystenemia.
Multiple investigations are included like chest radiograph, echocardiogram and blood profiles including PT-INR, Bleeding and clotting time profiles, blood counts and ESR.

RESULT

In our study, 23 patients, in the age group of newborns to 12 years, with infarcts in the basal ganglia region were selected. Number of male and female children which participated in the study was 12 and 11 respectively.
Maximum number of patients with evidence of basal ganglia infarcts belonged to the age group of 3-6 years.
Infarcts in basal ganglia were identified in all the 23 patients on CT and MRI with accuracy (100%).
Out of total number of subjects, 15 patients had acute infarcts and the remaining had chronic infarcts.
There was evidence of basal ganglia calcification in 6 patients out of the 15 patients of acute infarcts (40% patients). Table/figure 1 shows calcific foci in the basal ganglia region.
The presence of the calcification in the lenticulostriate arteries was confirmed on CT scan. Brain CT showed well-defined linear hyper attenuating lesions coursing vertically through the lower margin of the putamen, with HU value ranging between 55-90, suggestive of mineralization. (See Table/figure 1 and 2)

MRI brain appeared to be less reliable in detection of calcific focus in the lenticulostriate arteries. However, calcification could be appreciated on phase sequences in a few patients (See Table/figure 3). MR angiogram of brain appeared normal in all the cases (as seen in table/figure 4 and 5).
No other specific risk factor showed predominance in our study. Only 4 patients had history of trivial trauma before the episode.

DISCUSSION

There has been a lacuna in the knowledge of medical practitioners on stroke in pediatric age group. Over the years this knowledge has been improving consistently.
Occlusion of small penetrating arteries is the cause for basal ganglia infarcts [5]. In adults there is a strong association for such infarcts with atherosclerosis and hypertension. However in children these have been mainly as a result of arteritis or embolism [6]. One study done by Dusser et al. mentioned that many cases of basal ganglia infarcts had idiopathic etiology [7].
The term mineralizing angiopathy has been in literature since the 1960's to describe calcification in the basal ganglia on computed tomography [8-11].
Grant et al in the 1980's, first used the term lenticulostriate vasculopathy as not all the patient showed mineralization in the basal ganglia and thalami [10].
Recent study by Ramasamy S, Alagappan P, Venkataraman V. et al showed that majority of the strokes in the Indian pediatric population were arterial in nature. The most common artery to be involved were the lenticulostriate arteries [12].
The reason for calcification in basal ganglia still remains unfathomable. Calcification of lenticulostriate arteries on Computed tomography was observed in children as young as 6 months of age and this suggests a prenatal or early postnatal onset to such pathology.
Age wise distribution of patients of pediatric population that presented with acute infarcts in the basal ganglia region and participated in our study is shown in **Table/figure 6**.
Lenticulostriate arteries or anterolateral central arteries arise from the anterior portion of the circle Willis and supply the basal ganglia. They have been classified in literature as medial and lateral lenticulostriate arteries. Medial lenticulostriate arteries are considered to be arising from the A1 segment of the anterior cerebral artery and supply the globus pallidus and the medial part of the putamen. On the other hand, lateral lenticulostriate arteries arise from the proximal middle cerebral artery, usually the M1 segment but sometimes from the post bifurcation and M2 segment and supply the lateral portion of the putamen and external capsule as well as the upper internal capsule. **Table/figure 7** shows the anatomy of the lenticulostriate arteries.
Vasculopathy relating to the lenticulostriate arteries have been proposed due to various mechanisms. It includes both prenatal and postnatal causes. In prenatal we have perinatal asphyxia, respiratory diseases, congenital heart disease, foetal TORCH infection, chromosomal aberrations, congenital malformations, congenital metabolic acidosis, sialidosis, foetal hydrops. Neonatal polycythaemia, neonatal hypoglycaemia, bacterial meningitis, neonatal lupus erythematous and idiopathic causes have been attributed as the post-natal mechanisms.[7-11].
Lenticulostriate Vasculopathy pathologically show thickened walls which are hyper-cellular with intramural and perivascular mineralization (Table/figure 3). Vascular changes can be visualized to an extent in USG and are not easily visualized on CT or MRI.
A number of studies have found a relationship between trivial trauma like fall from bed, and mineralizing angiopathy [13-15]. This is probably due to the intimal trauma with thrombosis as its sequelae, arterial spasm (transient) or due to mechanical disruption to the flow in the perforating arteries.

None of the studies have been able to establish a strong relationship between infarcts in the basal ganglia region and lenticulostriate vasculopathy. We conducted this study for the same reason - to find if there is a relationship between them.
Our study was able to provide evidence between the presence of mineralizing angiopathy in the lenticulostriate arteries and infarcts in the basal ganglia region in pediatric population.

LIMITATIONS

Children with chronic infracts were not involved in this study.

REFERENCES

1. Bigi S, Fischer U, Wehrli E, Mattle HP, Boltshauser E, Bürki S, et al. Acute ischemic stroke in children versus young adults. Ann Neurol. 2011;70:245–54.

2. Toelle S, Avetisyan T, Kuyumjyan N, Sukhudyan B, Boltshauser E, Hackenberg A. Infantile basal ganglia stroke after mild head trauma associated with mineralizing.

3. Tsze DS, Valente JH. Pediatric stroke: a review. Emerg Med Int. 2011;2011:734506.

4. Goraya JS, Berry S, Saggar K, Ahluwalia A. Stroke after minor head trauma in infants and young children with basal ganglia calcification: a lenticulostriate vasculopathy? J Child Neurol. 2018;33:146–52.

5. Riela AR, Roach S. Etiology of stroke in children. J Child Neurol 1993; 8: 201-220.

6. Raybaud CA, Livet MO, Jiddane M, Pinsart N. Radiology of ischemic strokes in children. Neuroradiology 1985; 27: 567-578.

7. Dusser A, Goutieres F, Aicardi J. lschemic strokes in children. J Child Neurol 1986; 1: 131-136.

8. Wang HS, Kuo MF, Chang TC. Sonographic lenticulostriate vasculopathy in infants: some associations and a hypothesis. American Journal of Neuroradiology 1995;16(1):97-102.

9. Sisman J, Logan JW, Westra SJ, et al. Lenticulostriate vasculopathy in extremely low gestational age new-borns: Inter-rated variability of cranial ultrasound readings, antecedents and postnatal characteristics. Journal of Pediatric Neurology 2014;12(4):183-93.

10. Grant EG, Williams AL, Schellinger D, et al. Intracranial calcification in the infant and neonate: evaluation by sonography and CT. Radiology 1985;157(1):63-8.

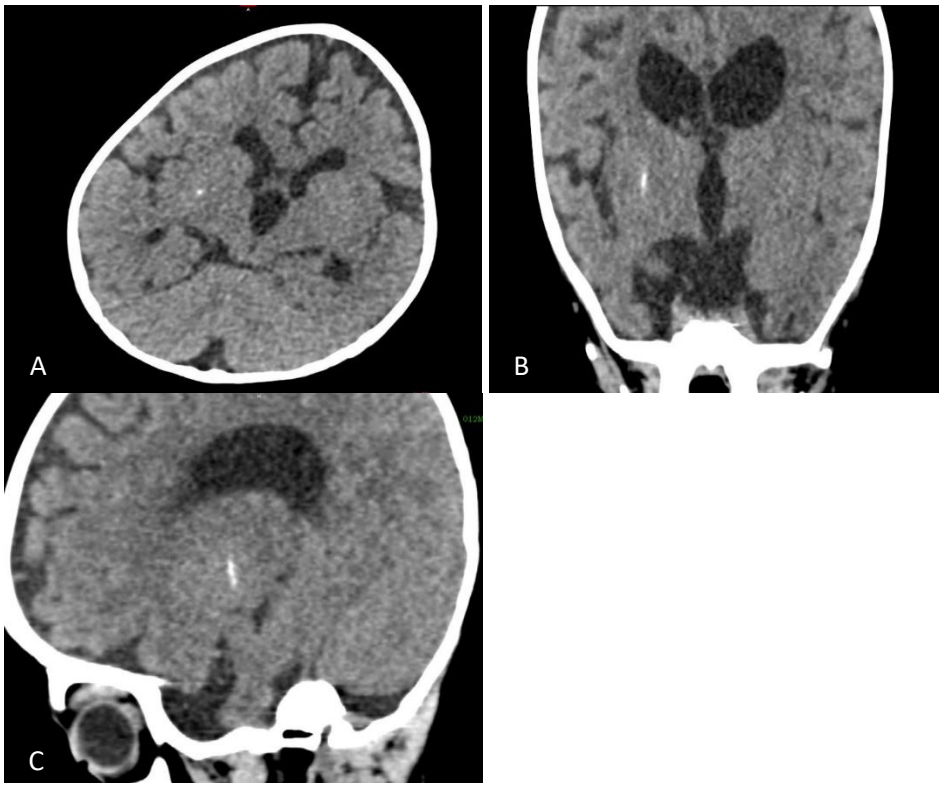
11. Teele RL, Hernanz-Schulman M, Sotrel A. Echogenic vasculature in the basal ganglia of neonates: a sonographic sign of vasculopathy. Radiology 1988;169(2):423-7.

12. Ramasamy S, Alagappan P, Venkataraman V, et al. Paediatric stroke- an analysis on artery of paediatric infarct- lenticulostriate arteries. J. Evolution Med. Dent. Sci. 2018;7(21):2550-2554,

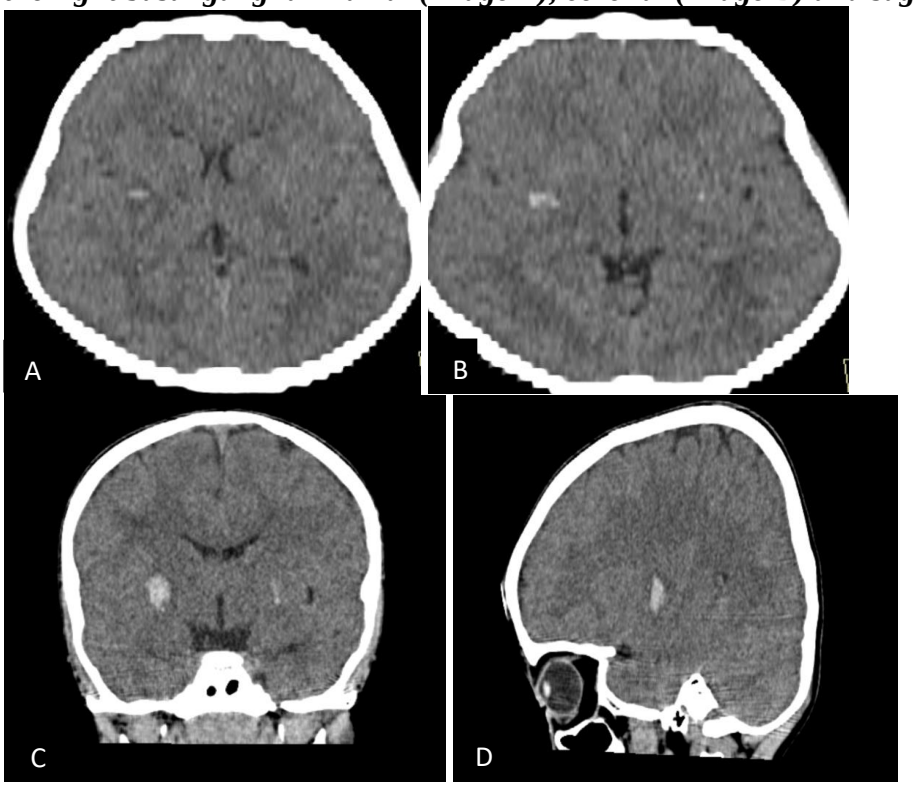
13. Lingappa L, Varma RD, Siddaiahgari S, et al. Mineralizing angiopathy with infantile basal ganglia stroke after minor trauma. Developmental Medicine & Child Neurology 2014;56(1):78-84.

14. Yang FH, Wang H, Zhang JM, et al. Clinical features and risk factors of cerebral infarction after mild head trauma under 18 months of age. Pediatric Neurology 2013;48(3):220-6.

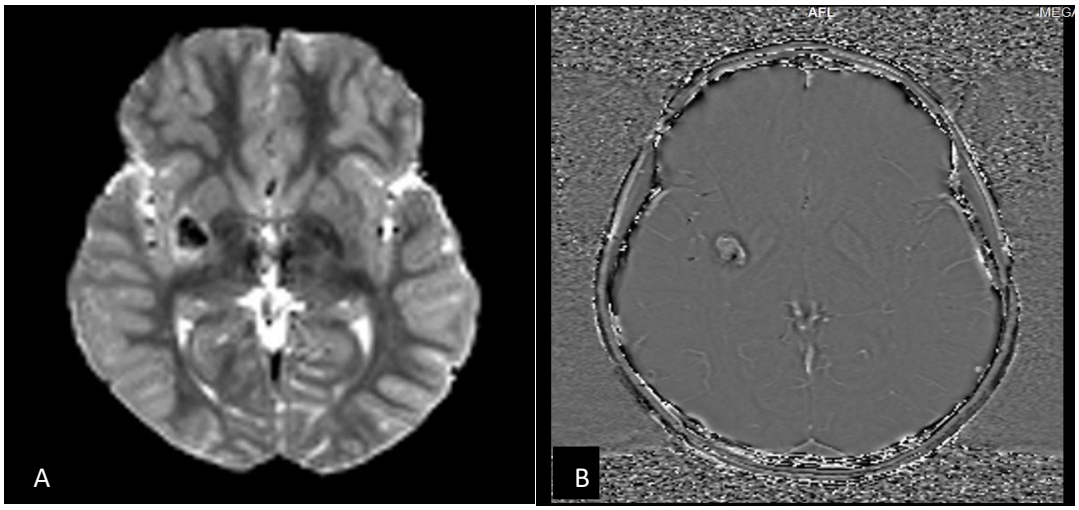
15. Yang ST, Lee WT, Hung KL, et al. Infantile basal ganglia stroke after mild head trauma. Neurology 2015;84(23):2381-2.



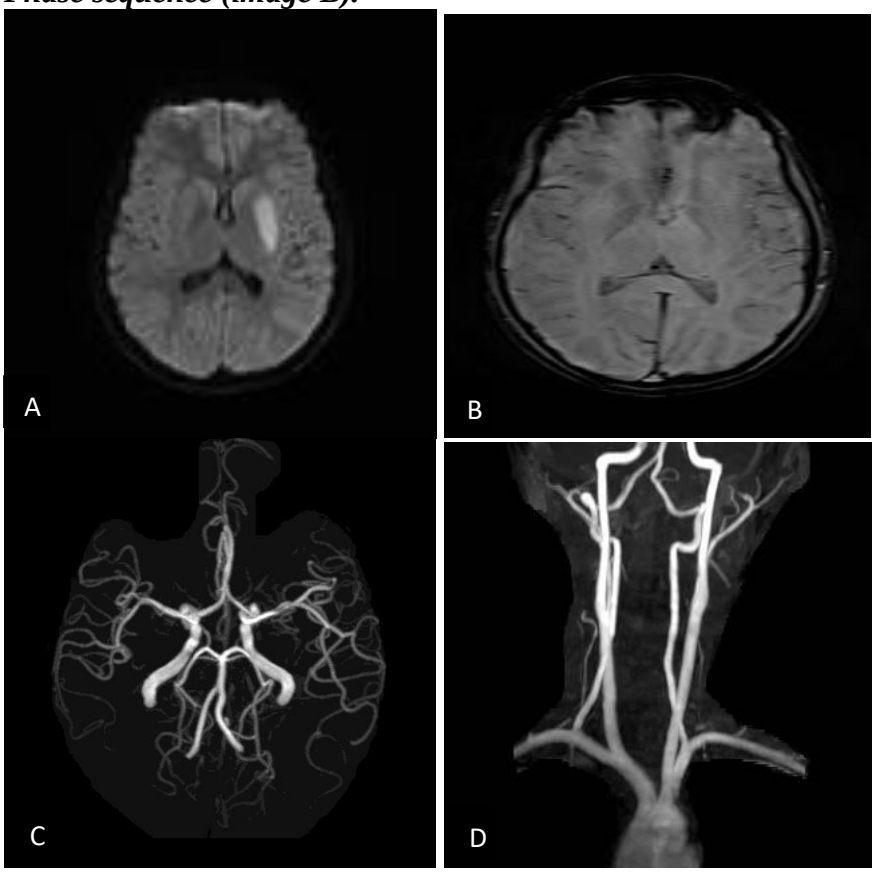
Figure/Table 1. NECT brain in a 1-year-old female child shows evidence of calcific foci in the right basal ganglia in axial (image A), coronal (image B) and sagittal (image C) planes.



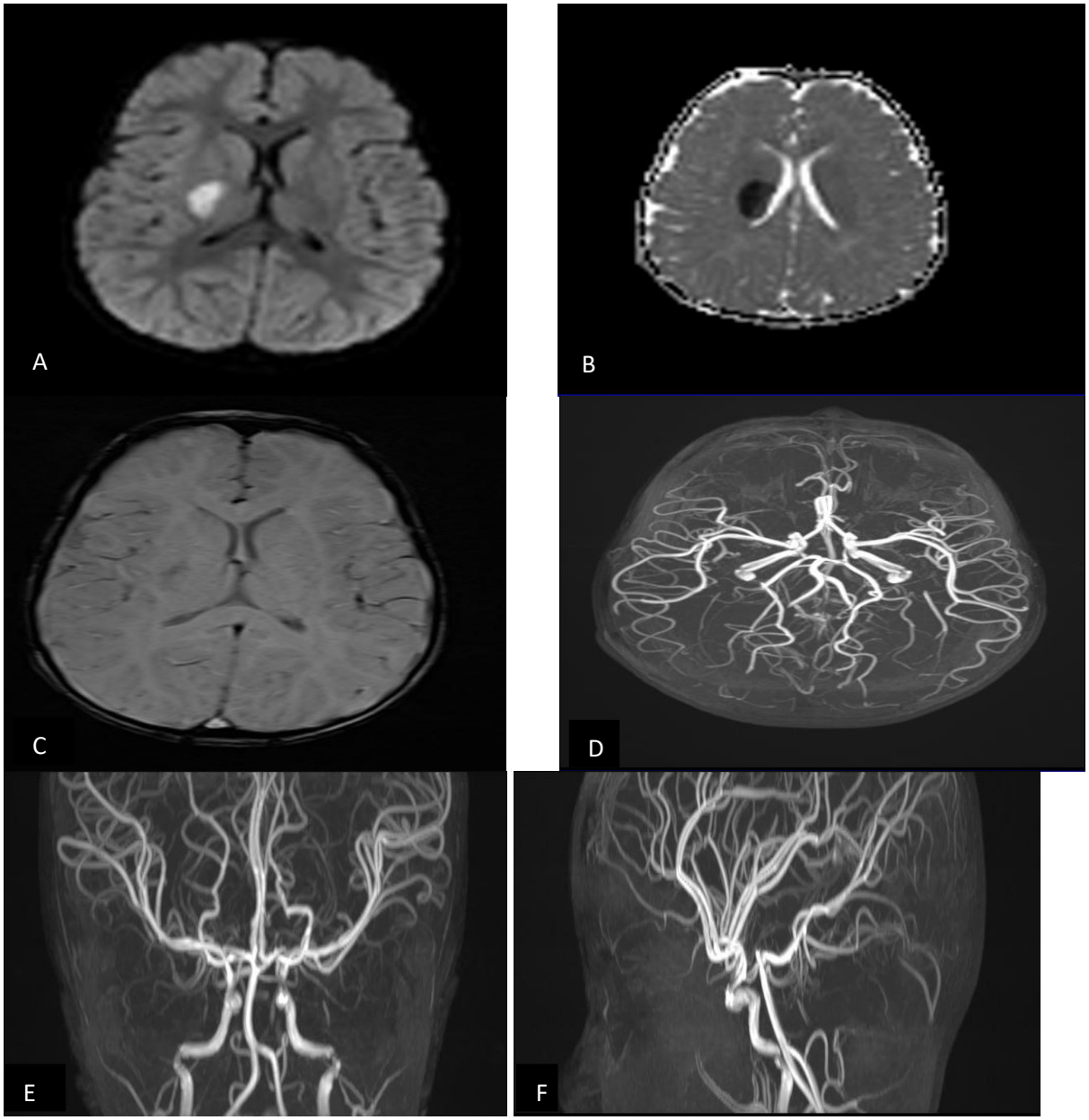
Figure/Table 2. NCCT brain of the same patient shows a hyperdense lesion in the right basal ganglia in axial (image A and B), coronal (image C) and sagittal (image D) planes.



Figure/Table 3. MRI brain of a7 year old male child shows an acute hemorrhagic infarct in the right basal ganglia (image A) with evidence of a calcific focus in the basal ganglia on Phase sequence (image B).



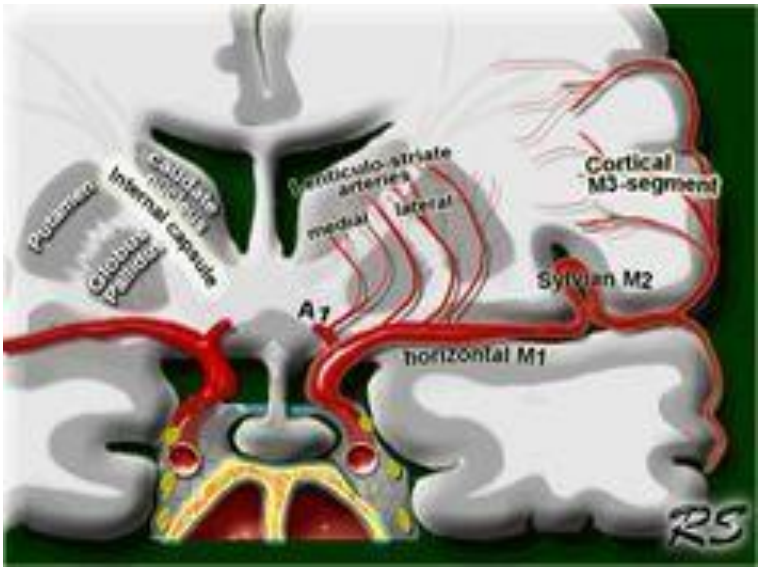
Figure/Table 4. MRI brain (DWI- image A; SWI- image B) showing an acute non-hemorrhagic infarct in the left basal ganglia in a 5-year-old male child. MR angiogram of brain(image C) and neck vessels(image D) appears normal.



Figure/Table 5. MRI brain of 2-year-old female child shows an acute infarct in the right basal ganglia on DWI (image A) and ADC (image B) sequences with blooming foci on SWI (image C) sequence suggestive of calcification in a lenticulostriate artery. Note that the MR angiogram in axial(image D), coronal (image E) and sagittal(image F) planes appears normal.

AGE OF THE PATIENT	*NUMBER OF PATIENTS*
0-3 years	*10*
3-6 years	*4*
6-9 years	*5*
9-12 years	*4*

Figure/Table 6- The age wise districation of the patients with acute infacrts in the basal ganglia region that were included in out study.



Figure/Table 7- Diagram depicting normal brain arterial supply showing normal lenticulostriate arteries. **This picture was reproduced from the Radiology Assistant website (http://www.radiologyassistant.nl) with permission from the author Dr Smithuis.**