# "MINERALISING ANGIOPATHY IN YOUNG CHILDREN: AN OBSERVATIONAL STUDY."

**BY**

**REG NO. BS0120005**

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# LIST OF ABBREVIATIONS:

| | |
|---|---|
| MA | Mineralizing angiopathy |
| LSV | Lenticulostriate vasculopathy |
| CMV | Cytomegalovirus |
| US | Cranial ultrasound |
| MCA | Middle cerebral artery |
| ACA | Anterior cerebral artery |
| HIV | Human immune deficiency virus |
| CT | Computed tomography |
| SLV | Sonographic lenticulostriate vasculopathy |
| ROI | Region of interest |
| PROM | Premature rupture of membranes |
| NICU | Neonatal intensive care unit |
| HU | Hounsfield units |
| H/O | History of |

# ABSTRACT

**Background:** Angiopathy is the disease of the arteries, veins, and capillaries. Two types of angiopathies namely microangiopathy and macroangiopathy are reported in literature. Mineralizing angiopathy (MA), which has a fair prognosis and is one of the primary culprits of stroke affecting the basal ganglia in infants and young children after a slight fall. Mineralizing angiopathy may be the unidentified aetiology of paediatric stroke. Further research is required to understand the mechanisms driving mineralizing angiopathy and the long-term outlook for such patients.

The objective of the study was to study the prevalence of Mineralizing Angiopathy in young children and to study the association perinatal infections with Mineralizing Angiopathy.

**Materials and methods:** One-year observational study was done in the department of radio diagnosis at the KLES DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI.

Children aged 6 month to 12 years of age, who are undergoing a computed topographic evaluation of the brain for any clinical indication were included in the study. The Total children included in the study were 180. All the scans were done with a 128 slice CT machine.

**Results:** Prevalence of mineralizing angiopathy in the study population (children aged between 6 months to 12 years) was 29.67%. The mean age of the children was 5.68 years. Majority of the MA cases belonged to the age group of 1-5 years.

In the present study among the cases of MA Majority of them had 6-10 calcific foci in 37% of the cases and the mean foci of calcification in cases were 9.43

Five of the patients of MA had also underwent MRI examination which did not show evidence calcifications or blooming on SWI sequence. MRI is not sensitive for detection of MA

The average HU of the calcific foci was +60.47 HU.

Visualization of the linear foci of calcification along the lenticulostriate vessels was demonstrated in coronal reconstruction in 25.93% of the cases.

Only 5 cases (8.9 %) of the cases of MA (54cases) had presented with stroke after minor trauma/fall.

The mean duration of labour was longer in cases of MA the duration of mean duration of PROM was obtained from history in the MA group was 18.74hr.

The average number of calcifications and average HU of the calcifications was more when prolonged duration of PROM.

In the present we have found the history PROM and NICU admission and duration of labour had a statistically significant relationship with MA, which states that there is a possible association between perinatal infections and mineralizing angiopathy. Any site of infection in the brain during the intranatal and peripartum period can predispose to MA.

**Conclusion:** The diagnostic utility of imaging modalities like MRI is limited in children with MA, but CT has higher utility in picking up foci of calcification and demonstration of linear calcifications in coronal reconstruction

The prevalence of mineralizing angiopathy in our study was 29.67%. Majority of the cases of MA belonged to the age group of 1 to 5 years

Mineralizing angiopathy has got association with perinatal infections (history of PROM and NICU admissions) during delivery.

Minimal number of the cases of MA present with stroke when they have a fall in the vulnerable period of 1 to 5 years.

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# INTRODUCTION

Angiopathy is the disease of the arteries, veins, and capillaries. Two types of angiopathies namely microangiopathy and macroangiopathy are reported in literature[1]. The walls of small blood arteries thicken and become so weak in microangiopathy that they bleed, leak protein and impair blood flow[1].

A frequent cause of subcortical strokes in infants following minor trauma is mineralizing angiopathy involving lenticulostriate arteries. CT scans of the affected area frequently reveal calcification[2].

Mineralizing microangiopathy is a disorder that typically affects children as a side effect of cranial radiotherapy and chemotherapy. It is characterised by parenchymal cerebral calcifications[3].

The corticomedullary junction regions, lentiform nuclei of the basal ganglia, and the dentate nucleus of the cerebellum are among the typical sites affected by the disorder, which can impact many different parts of the brain[3]. Thalamostriate vasculopathy and mineralizing angiopathy are other names for lenticulostriate vasculopathy (LSV) [4].

Numerous infectious etiologies, such as cytomegalovirus (CMV), rubella, amniotic infection, streptococcus sepsis, and syphilis beni, have been related to LSV[5].

# OBJECTIVES

**AIM AND OBJECTIVES OF THE STUDY:**

In view of the above findings further the dissertation work is taken up to study

- **PRIMARY OBJECTIVE:** To study the prevalence of Mineralizing Angiopathy in young children who have undergone a CT scan at KLE's Dr Prabhakar Kore hospital.
- **SECONDARY OBJECTIVE:** To study the association perinatal infections with Mineralizing Angiopathy.

**NEED FOR THE STUDY:**

Mineralizing angiopathy (MA), which has a fair prognosis and is one of the primary culprits of stroke affecting the basal ganglia in infants and young children after a slight fall, is increasingly recognised. With a mostly unknown origin, this disorder causes lenticulostriate artery calcification in the internal capsule and basal ganglia.

It is thought to be believed that the anatomical and developmental properties of the origin and course of lenticulostriate arteries during infancy are what contribute to the lenticulostriate arteries' increased vulnerabiliy to thrombosis and injury following minor head trauma.

Mineralizing angiopathy may be the unidentified aetiology of paediatric stroke. Thin-section spiral CT with multiplanar reconstructions may be helpful for kids who experience localised impairments following minimal trauma. Further research is required to understand the mechanisms driving mineralizing angiopathy and the long-term outlook for such patients.

# REVIEW OF LITERATURE

Lenticulostriate vasculopathy (LSV) has confounded radiologists and neonatologists ever since it was originally identified by Grant et al. in 1985 [6]. Grant et al initial report on cranial ultrasound (US) noted "branched echogenicities in basal ganglia; punctate calcifications near the angle of the right lateral ventricle" in a second twin born during a 35-week pregnancy.

The first to record "branching echogenicities" in a newborn's basal ganglia were described by Grant et al., while the first to use the term "vasculopathy" to characterise the 12 cases they documented by Teele et al.[7]

Furthermore, the branching pattern of the branch arteries paralleled the branching patterns of the anterior thalamo perforating arteries (branches of the posterior communicating artery), posterior thalamo perforating and thalamogeniculate arteries, medial lenticulostriate arteries (arising from the A1 segment of the anterior cerebral artery), lateral lenticulostriate arteries (arising from the pre-bifurcation (M1) segment of the middle cerebral artery[8].

Using a newborn cranial ultrasound, LSV was identified. The majority of researchers have predicated their LSV diagnoses with the existence of a linear or branched echogenicities in the thalamic or basal ganglia[9].

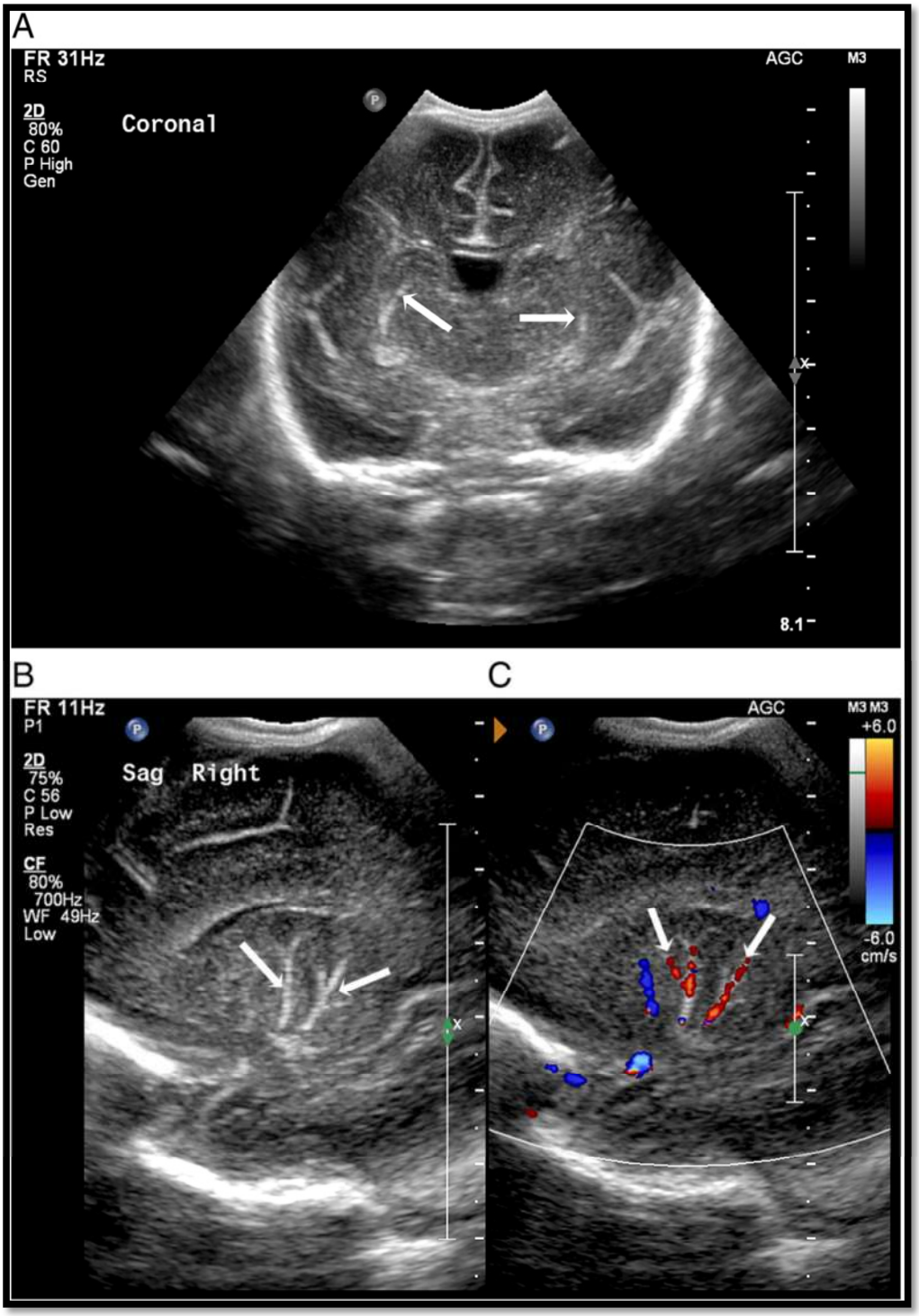
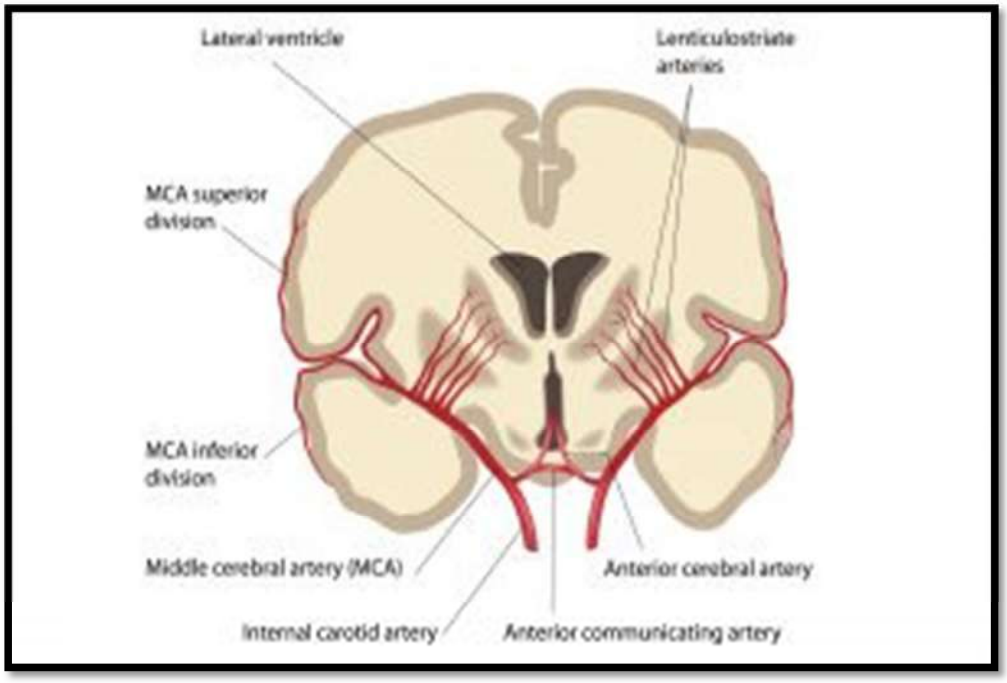



**Figure 1:** Newborn at 30 weeks gestation, coronal view. The echogenic, thick lenticulostriate vessels are present. Parasagittal grey scale (A) and coronal scale. B. The vessels are as thick and echogenic as the sulci, according to right parasagittal imaging.

Images display thick, linear echogenicities that are compatible with LSV (arrows). The arterial (red arrow) and venous (blue arrow) flow in parasagittal colour Doppler image (C) correlate to the LSV on A and B[10].

The lenticulostriate arteries arise from the M1 segment of the middle cerebral artery (MCA). They are tiny perforating arteries that feed blood to the posterior limb of the internal capsule and a portion of the basal ganglia at the anterior perforated substance on the underside of the brain. End arteries make up the lenticulostriate perforators. The lentiform nucleus and the striatum, two of the structures these arteries nourish, are where these arteries get their names[11].

**Figure 2: lenticulostriate arteries**



The basal ganglia are supplied by a group of tiny perforating arteries known as the lenticulostriate arteries, also called the anterolateral central arteries. They originate from the circle of Willis anteriorly. They are divided into:

1. lenticulostriate medial arteries
2. lenticulostriate lateral arteries

Most explain the medial and lateral arteries that emerge from the M1 section of the middle cerebral artery and the medial arteries that arise from the A1 portion of the anterior cerebral artery.

However, some writers further distinguish between those arising more proximally (medial) and those arising more distally (lateral) groups of the perforating arteries that emerge from the M1 segment[8].

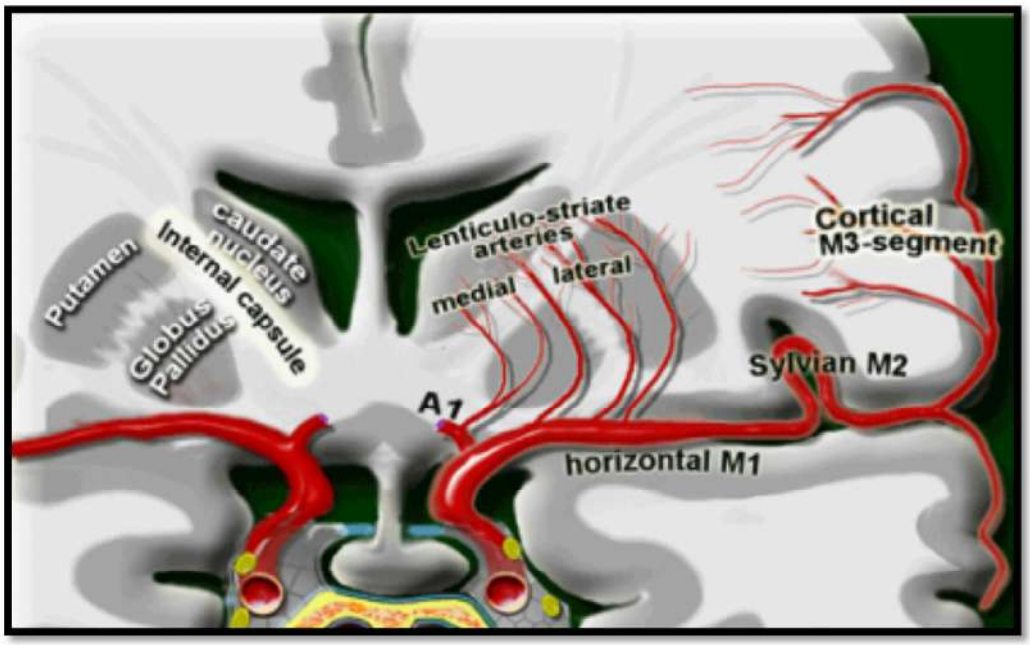



**Figure 3: perforating lenticulostriate arteries**

The medial lenticulostriate arteries nourish the putamen, and globus pallidus. They are widely thought to originate from the anterior cerebral artery's (ACA) A1 segment.

Compared to the lateral lenticulostriate arteries, which emerge farther down the M1 segment, they are thinner, shorter, and less numerous. There is still some debate about where they came from, although the majority say they came from the ACA A1 portion. However, some, such as[8], categorise the perforating arteries two groupings called medial (those arising closer together) and lateral (those arising farther apart), based on where they emerge from the middle cerebral artery (MCA) M1 section. Additionally, additional proximal branches, such as the fronto-orbital artery, can give rise to similar perforating arteries. Therefore, it is simpler to think of these arteries in terms of the tissue they serve rather than the artery from which they originate[13].

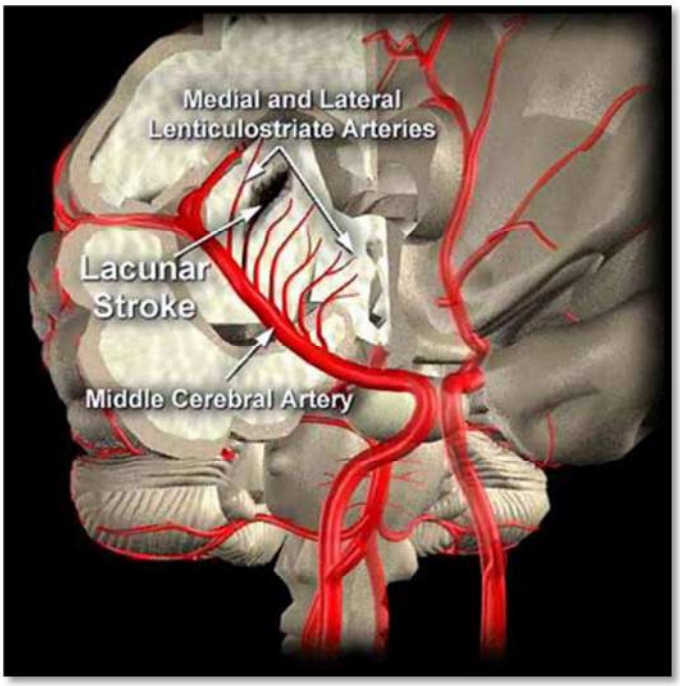



**Figure 4: Medial lentoculostriate arteries**

The postbifurcation or M2 segment is less frequently the origin of the lateral lenticulostriate arteries' proximal middle cerebral artery (MCA) origin. They supply the upper internal capsule as well as the lateral putamen and external capsule.

They arise more distantly from the A1 ACA segment and are longer (nearly twice the diameter) and more numerous than the medial lenticulostriate arteries.

The recurrent artery of Heubner, which arises from the ACA, supplies both the inferior head of the caudate and the lower anterior internal capsule[14].

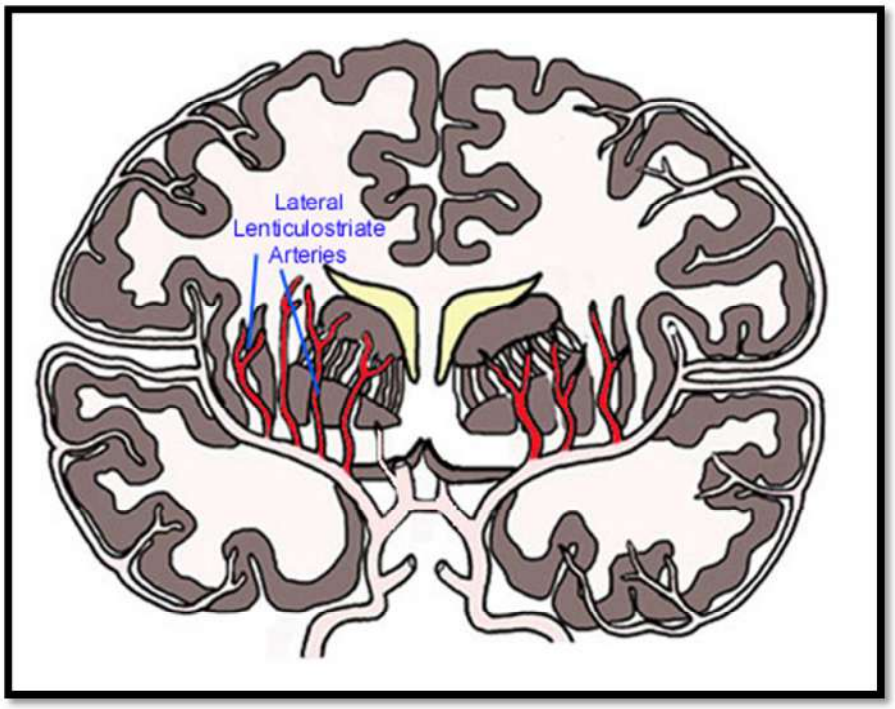



**Figure 5: lenticulostriate lateral arteries**

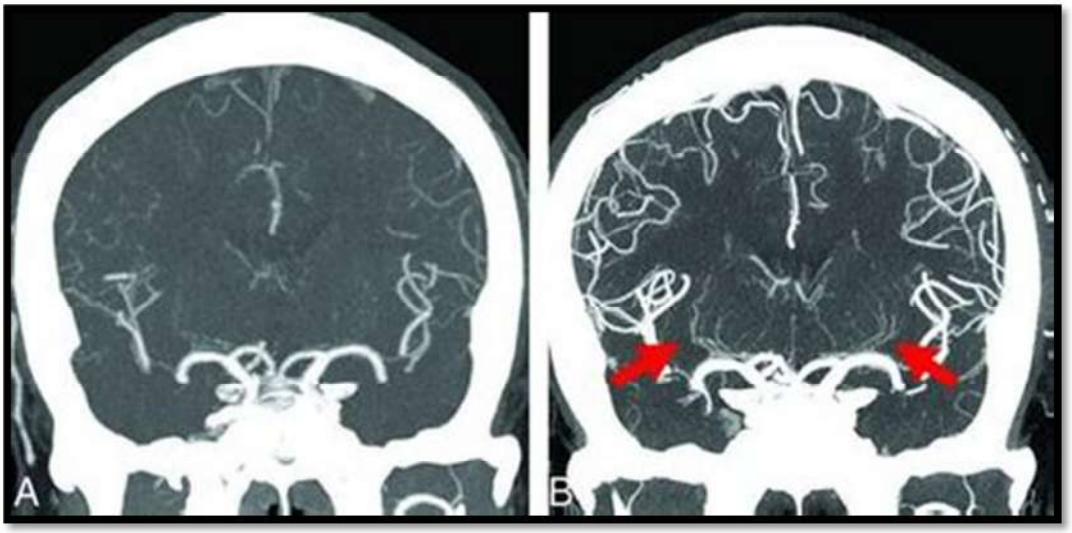



**Figure 6: Visualization of the lenticulostriate arteries on CT**

In the Hughes et al[15]. study, just one newborn with foetal alcohol syndrome, microcephaly, and ultimately infant sudden death syndrome exhibited calcifications in the basal ganglia, despite the fact that 4 of the 10 CT scans were deemed abnormal. Congenital CMV infections affected the other 3 patients, and in two of them, there were the recognisable coarse periventricular calcifications.

Three individuals with LSV, of all whom had congenital infections, developed calcifications on CT scans of their basal ganglia and thalami, as per Cabanas et al.[16] (toxoplasmosis, CMV and HIV).

In the Ben-Ami T et al. study shows that bacterial, viral, and spirochetal infections of the central nervous system can cause these echogenic arteries in the basal ganglia[5]

Numerous investigators have shown that modest head trauma can lead to acute ischemic strokes (basal ganglia infarcts)[17-20] .

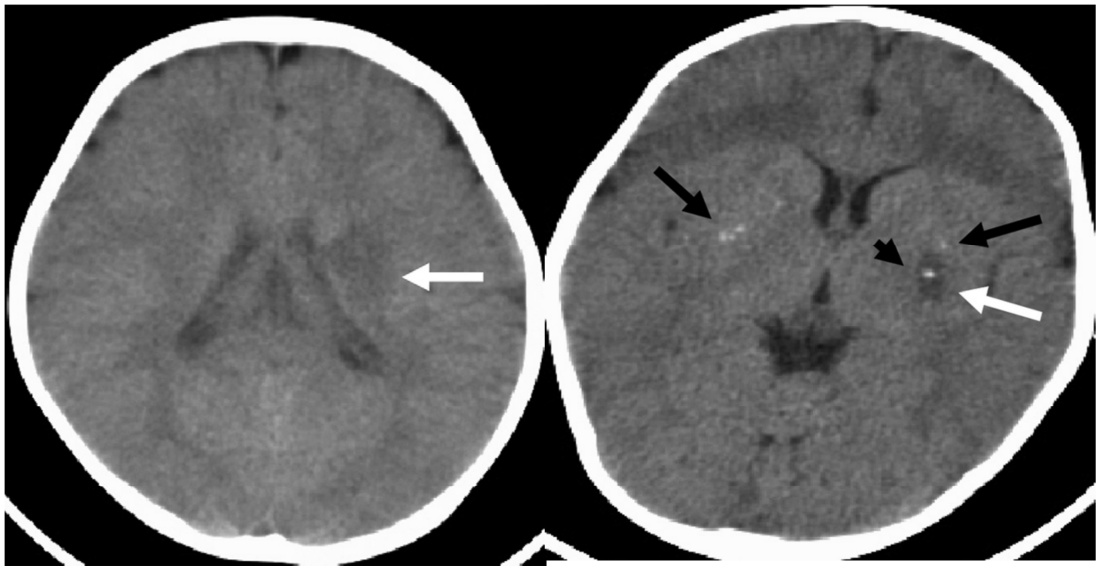


**Figure 7-** after a minor head trauma, infarction on a computed tomography scan. Twenty minutes following a stairfall, in 17-month-old child (patient 1) had a presentation of right hemiparesis. A low density lesion was discovered during a head CT scan in the left corona radiata (arrow)[20] .

- Post-trauma cerebral infarction computed CT evidence of basal ganglia calcification. Following a mild head injury, in 15-month-old kid, had presentation of right hemiparesis. A hyodense area in the left basal ganglia and bilateral hyperdense areas in the basal ganglia were seen on computed tomography (white arrow) [20] .

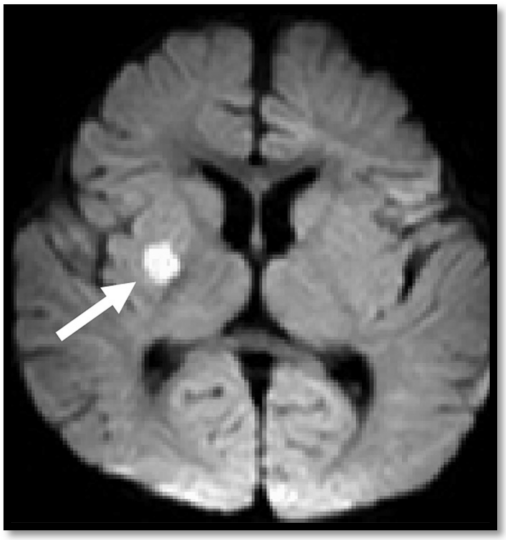


**Figure 8:** Following minor head trauma resulted in cerebral infarction in the basal ganglia. A 12-month-old boy experienced a seizure for 20 minutes after rolling out of bed. Acute infarction was discovered by diffusion-weighted images in the right basal ganglia (arrow)[20].

A distinct clinico-radiological stroke syndrome in children was described by Lingappa et al.[21]in 2014, which they called 'mineralizing angiopathy'. After minor head injury within minutes to hours, basal ganglia infarction, age at stroke greater than 28 days but younger than 18 months, linear calcifications along the lenticulostriate arteries, minimal risk of recurrence, and an overall favourable prognosis.

In 4 (1%) of the 400 CT scans, Lingappa and colleagues discovered accidental lenticulostriate arteries mineralization. On a CT scan done for unrelated reasons, calcific or mineralized lenticulostriate vasculopathy can occasionally be found.[21]

Pathological persistence of lenticulostriate vasculopathy on sonsography is likely to represent mineralizing angiopathy.[21]

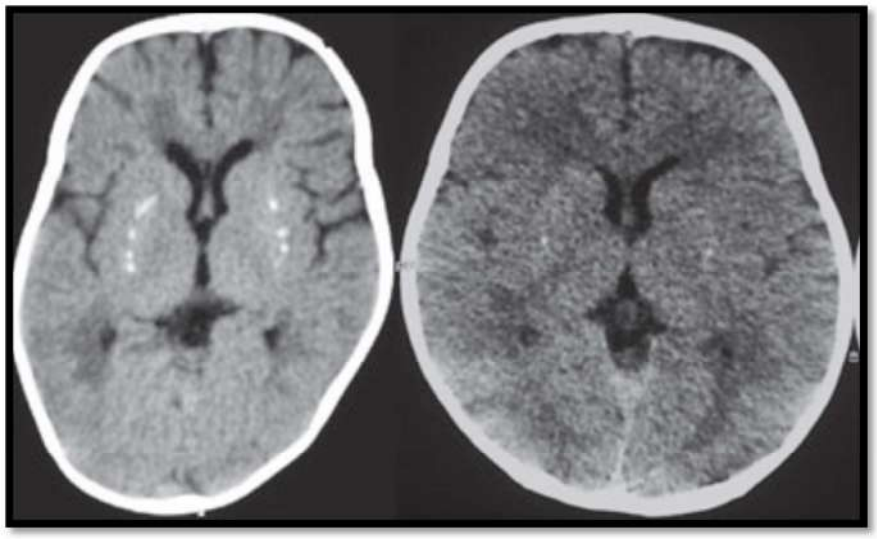


**Figure 9: Axial CT image showing punctate calcifications in the basal ganglia[21]**

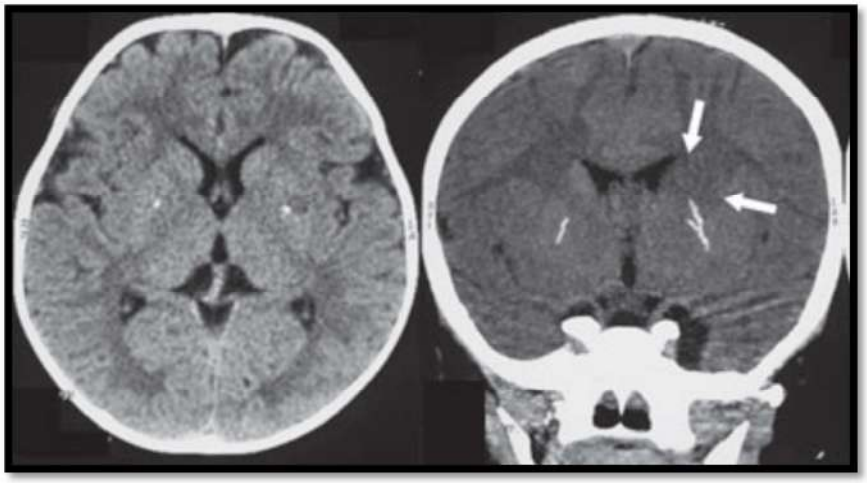


**Figure 10: Linear hyperdense lesions with linear margins that emerged vertically from the inferior region of the lentiform nucleus on coronal reconstructions[21]**

In every infant, a CT scan revealed typical linear hyperdense lesions with linear margins that emerged vertically from the inferior region of the lentiform nucleus. The distributions of these linear lesions on coronal and sagittal views matched those mentioned in literature anatomically about lenticulostriate arteries [21]

These hyperdense areas had attenuation values which represented mineralization between 58 and 90 HU. There were between one and five of these mineralized vessels on each side. No other areas of the brain, including the basal ganglia, had calcification foci.

With varying degrees of superior extension, the infarcts were situated at the mid-putaminal level and centred to the mineralized lenticulostriate arteries. Due to the use of multislice CT widespreadly, it is now possible to perform reconstructions of these hyperdense areas with multiplanar imaging and comprehend the linear orientation along the course of the lenticulostriate arteries[21]

The various interpretations of these hyperdense foci within the infarcts include thrombus or minute foci of haemorrhage[22]

It is still unclear what specifically caused the lenticulostriate vessels to mineralize. According to a study by Yang et al., serologically positive tests for CMV, Epstein-Barr virus, echovirus, and mycoplasma are associated with post-traumatic basal ganglia infarction.[20]

These linear zones of mineralization have a similar characteristic distribution to the sonographic lenticulostriate vasculopathy (SLV). There is evidence that between 1.9% and 5.8% of sick neonates and 0.4% of live-born neonates experience this.[7, 16, 23]

Bidirectional basal ganglia vessel mineralization has been thoroughly investigated in various species. Cynomolgus monkeys under the age of 3 to 4 years old had a higher incidence of severe lesions than older monkeys, according to a

detailed pathological investigation. It's possible that the type A mineralization seen in that study's arterioles and venules is similar to human mineralizing angiopathy.[24]

Calcium, iron, phosphorus, and chlorine peaks were visible in radiograph microanalysis, along with trace levels of zinc, potassium and aluminium. This concept definitely explains many aspects of basal ganglia vascular mineralization and its pathogenic substrate, but it is still unclear what causes some individuals' mineralization and why it does not occur in others.[24]

Lenticulostriate vasculopathy has hypercellular thick vessel walls with perivascular and intramural mineralization as its pathological hallmarks. The vascular alterations are typically not seen on CT or MRI, despite the fact that lenticulostriate vasculopathy is plainly visible on neurosonography.[15,23,25]

To distinguish it from SLV, we suggest calling this type of mineralization that can be seen on CT as "mineralizing angiopathy." To determine the reasons linked to the persistent lenticulostriate artery mineralization in these newborns, additional longitudinal investigations are necessary.[21]

In one patient from this series, the potential connection between mineralizing angiopathy and SLV was seen. When this baby was a month old, sonography revealed lenticulostriate vasculopathy and 7 months later, after suffering a post-traumatic infarct, a CT scan revealed lenticulostriate artery mineralization.[21]

An 8-month-old baby with a history of SLV was found to have post-traumatic cerebral infarction, according to Ivanov et al. They hypothesised that the newborn was susceptible to or that the vascular occlusion brought on by head injury was made worse by the underlying lenticulostriate vasculopathy.[26]

Prior reports of newborns with post-trauma strokes in basal ganglia may not have recognised the pathological entity (best seen on CT) because MRI was used as the predominant imaging modality for those cases.

The calcific foci on recalled gradient echo or vascular anomalies on MRA were not discernible when we retrospectively analysed the available MR images in our patients. Even when performing a conventional CT for head injury of 5mm axial slices, it may be difficult to understand the true nature of the vascular mineralization.[21]

They proposed that the infant was predisposed to, or that the vascular occlusion brought on by, the head injury was made worse by, the underlying lenticulostriate vasculopathy.

According to our theory, small trauma may cause strains across the mineralized lenticulostriate arteries that may put people at risk for thrombosis and stroke. This seems a fairly age-specific tendency with distinct mineralization stages.

Despite persistent calcification, the vessels appear to be susceptible to occlusion following moderate trauma and to remain asymptomatic at older ages (beyond 2-3 years).[21]

It seems to be interesting that an idiopathic or isolated variant of LSV might strike apparently healthy newborns even in the lack of identified risk factors.[27-30] Isolated lenticulostriate vasculopathy was not accompanied by any symptoms and is typically regarded as benign with no long-term negative effects on neurodevelopment. It has been discovered that isolated lenticulostriate vasculopathy affects 3.6% to 7.4% of healthy newborn newborns having regular cranial ultrasonography.[27,28,30]

A study conducted by the Neurology division of Pediatrics department ,from Ludhiana Punjab proposed that, although the Although the exact reason of basal ganglia calcification (or lenticulostriate artery calcification) is unknown, the fact that it can be detected on CT in infants as young as six months age indicates the pathophysiological mechanisms causing lenticulostriate artery calcification are likely to have prenatal or early postnatal onset. There haven't been any blatantly detrimental prenatal or perinatal occurrences associated with these infants, though. Affected neonates undergo typical physical and neurodevelopment prior to the onset of stroke. According to clinical or laboratory findings, basal ganglia calcification in children is typically not associated with many etiologies (such as disorders of calcium/phosphorus metabolism, intrauterine infections, neonatal asphyxia or inborn errors of metabolism).[31]

Children with mineralizing angiopathy had a higher rate of perinatal infections than kids without focal cerebral arteriopathy or stroke, according to a study done at KLE Hospital. Forty non-stroke patients were included, including sixteen kids with mineralizing angiopathy, fourteen kids with focal cerebral arteriopathy, and sixteen other kids. To check for prenatal infection, a thorough parent interview was undertaken. There were no children with focal cerebral arteriopathy, however there were 8 individuals (68.2%) with mineralizing angiopathy who had infection in the perinatal period (PROM in 8 and confirmed sepsis neonatal period in 2. Among non-stroke patients, only 3 (7.5%) had a history of PROM.[32]

Understanding the cause aids in management, prevention, and parent counselling. Genetic causation is unlikely in the majority of instances of MA due to

the absence of case clustering in families and the non-progressive nature of the disease.[32]

Chorioamnionitis is one of the more severe complication[33] of PROM. In cases of preterm rupture of the membranes without problems, neonatal infection increased by ten times. The inflammatory response to low grade infection associated with PROM may result in the calcification of lenticulostriate arteries, whereas severe infection during the perinatal period might cause newborn sepsis and its accompanying complications.[33, 34]

Because of the orientation being oblique and shorter length in children under the age of five, these arteries, after a minor trauma, undergo dissection via shearing force or become vasospastic, resulting in an ischemic stroke[35]

The lenticulostriate arteries later develop into linear arteries with growth, and their occurrence decreases as people get older. Therefore, children under the age of five have a significant incidence of mineralizing angiopathy (MA).[32]

It's probable that prenatal infections and mineralizing angiopathy are related. A percentage of children with perinatal infections develop mineralizing angiopathy, and if they experience moderate trauma in the first five years, they may get basal ganglia stroke.[32]

Mineralizing angiopathy is a distinctive clinical and radiological entity. It is believed to be as a consequence of head trauma, clinically they will present as basal ganglia stroke with good prognosis on follow up. MA is also found in asymptomatic children with no clinical symptoms. Further research is required to understand its mechanisms of etiology and associations.

# MATERIALS AND METHODS

**7.1 Source of data:** Pediatric Patients of age between 1 month to 12 years of age referred to the Department of Radio-Diagnosis at the KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi to undergo Computed tomography study of the brain irrespective of any indication.

**7.2** Method of collection of data:

**Study design:** Cross sectional study

**Study duration:** January 2021- December 2021

**Sampling method:** Random sampling

**Sample size:** Study comprises of 45 patients.

**Inclusion Criteria:** Children aged 6 month to 12 years of age, who are undergoing a computed topographic evaluation of the brain for any clinical indication.

**Exclusion Criteria:** Nil

**Sample size formula:** The minimum sample size formula based on prevalence rate is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where P is the prevalence percentage and d is the likelihood of the prevalence difference in percentage.

$z\alpha$ is linked with the level of significance. For 5% level of the significance $z\alpha = 1.96$.

Ref: A study conducted at KLE hospital discovered that

68.2% of children with mineralizing angiopathy had perinatal infections. [32]

With P = 68.2% and d = 20% of P = 13.64%, the sample size is 45.

**Statistical Analysis:** The study's main objective is to compare the two groups. We will compute the mean and standard deviation for the continuous quantitative data. Utilizing appropriate statistical methods like the unpaired student's t test, the continuous variables between groups will be compared. The student's paired t test will be utilised to compare two quantitative variables within a group.

Rates, ratios, and percentages will be used to express the categorical data. Using the Chi-square test or Fisher's exact test, the relationship between the result, clinical, and demographic factors will be examined.

Discrete variables will be represented by median.

Nonparametric tests will be used for comparing discrete variables.

Suitable graphs will be used to depict the comparison.

For all the tests the value of p less than 5% (0.05) will be considered significant.

**Methodology:** After taking consent from parents data will be collected from Children aged from 6 months to 12 years referred for a computed tomography study of the brain irrespective of clinical indication to the Department of Radio-Diagnosis at KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi. Detailed parenteral interview will be conducted to look for perinatal infections and clinical findings will be noted.

**Method collection tools:** Patients will undergo 128 slice Computed tomography scan to find out the prevalence of mineralizing angiopathy.

Standard scan protocol will be followed for all the patients undergoing Computed Tomography. Once the Computed tomography is done, findings will be noted and analyzed.

Patients who show positive findings of mineralizing angiopathy, will be interviewed to find out the history perinatal infections (premature rupture of membranes and neonatal sepsis).

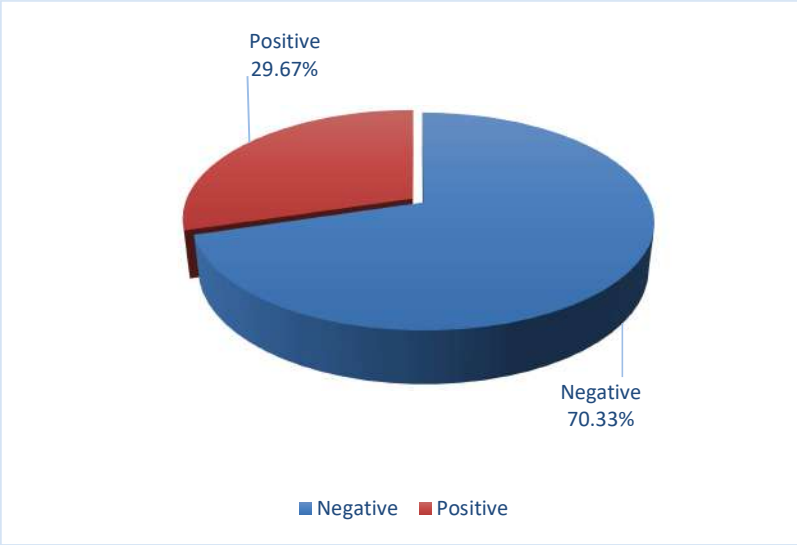
# RESULTS

The study included a total of 180 children between the ages of 6 months and 12 years referred to the Department of Radio-Diagnosis at KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi irrespective of the clinical indication, for computed tomography study of the brain for a period from January 2021- December 2021.

**Table 1: Prevalence of mineralizing angiopathy**

| Prevalence of mineralizing angiopathy | No of patients | % of patients |
|---|---|---|
| Study subjects without calcification | 126 | 70.33 |
| Study subjects with calcification (MA) | 54 | 29.67 |
| Total no of study subjects | 180 | 100.00 |

**Graph 1: Pie chart - prevalence of mineralizing angiopathy**




The prevalence of Mineralizing angiopathy among the study population was 29.67% Among the study population of a total of 182 patients that were enrolled 54 patients had MA.

**Table 2: Age wise distribution in the study group**

| Age groups | No of patients | % of patients |
|---|---|---|
| 0.06-1yr | 20 | 11.11 |
| 2-5yrs | 69 | 38.33 |
| 6-10yrs | 71 | 39.44 |
| 11-12yrs | 20 | 11.11 |
| Total | 180 | 100.00 |
| Mean age | 5.68 | |
| SD age | 3.58 | |

**Graph 2: Age wise distribution of the study group**

Among the study population the most common subjects belonged to the age group of 6 to 10 years (39.4%) followed by 2 to 5 years (38.3%)

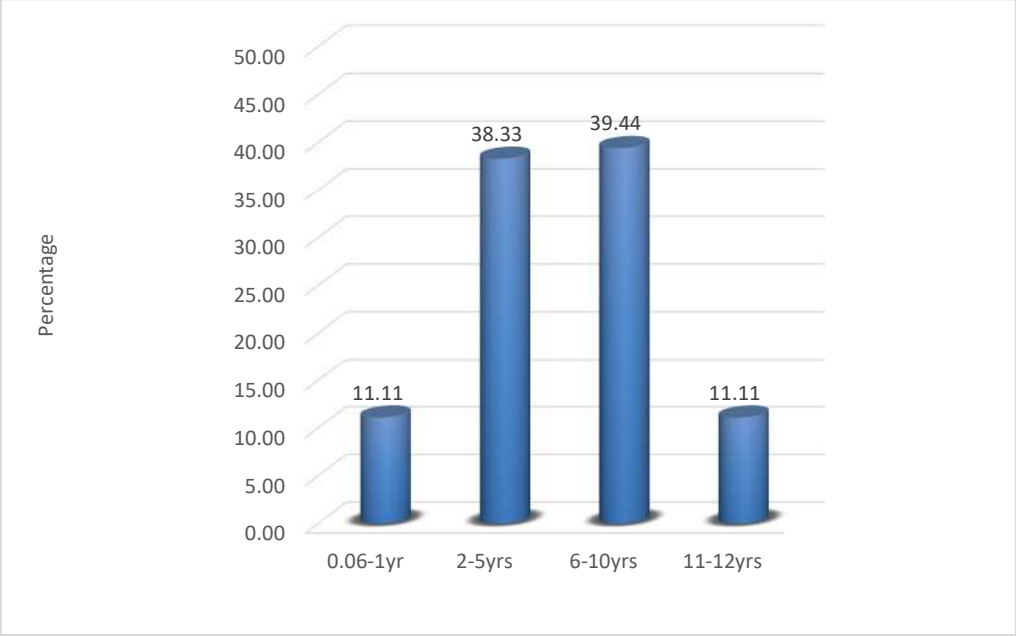
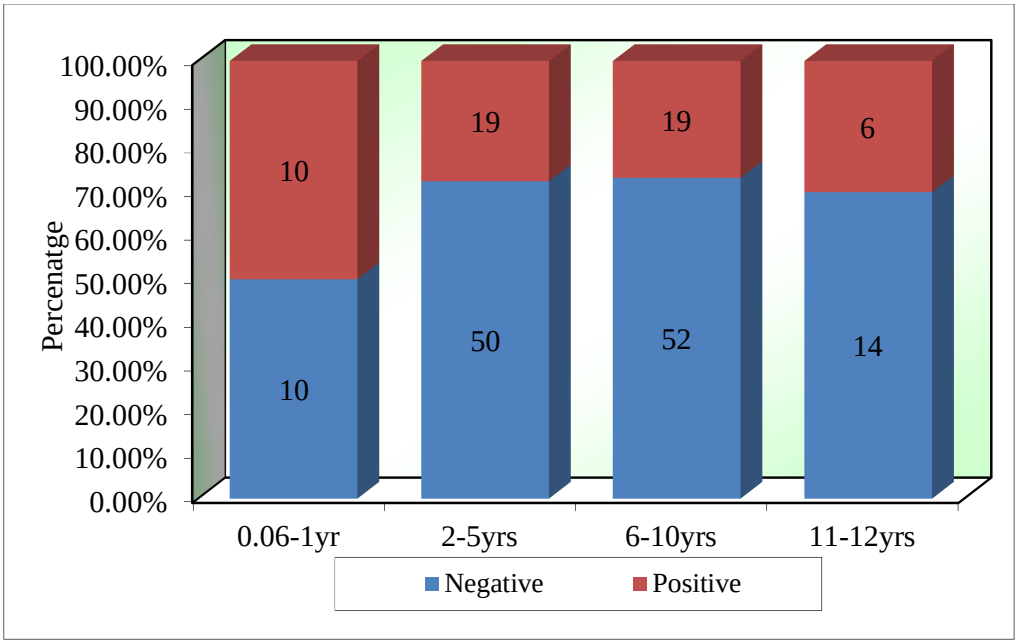


**Table 3: Association between age groups of patients with prevalence of mineralizing angiopathy**

| Age groups | Negative | % | Positive | % | Total | % |
|---|---|---|---|---|---|---|
| 0.06-1yr | 10 | 50.00 | 10 | 50.00 | 20 | 11.11 |
| 2-5yrs | 50 | 72.46 | 19 | 27.54 | 69 | 38.33 |
| 6-10yrs | 52 | 73.24 | 19 | 26.76 | 71 | 39.44 |
| 11-12yrs | 14 | 70.00 | 6 | 30.00 | 20 | 11.11 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Mean age | 5.84 | | 5.32 | | 5.68 | |
| SD age | 3.43 | | 3.93 | | 3.58 | |
| Chi-square=4.3640, p=0.2250 | | | | | | |

**Graph 3: Association between age groups of patients with Prevalence of mineralizing angiopathy**

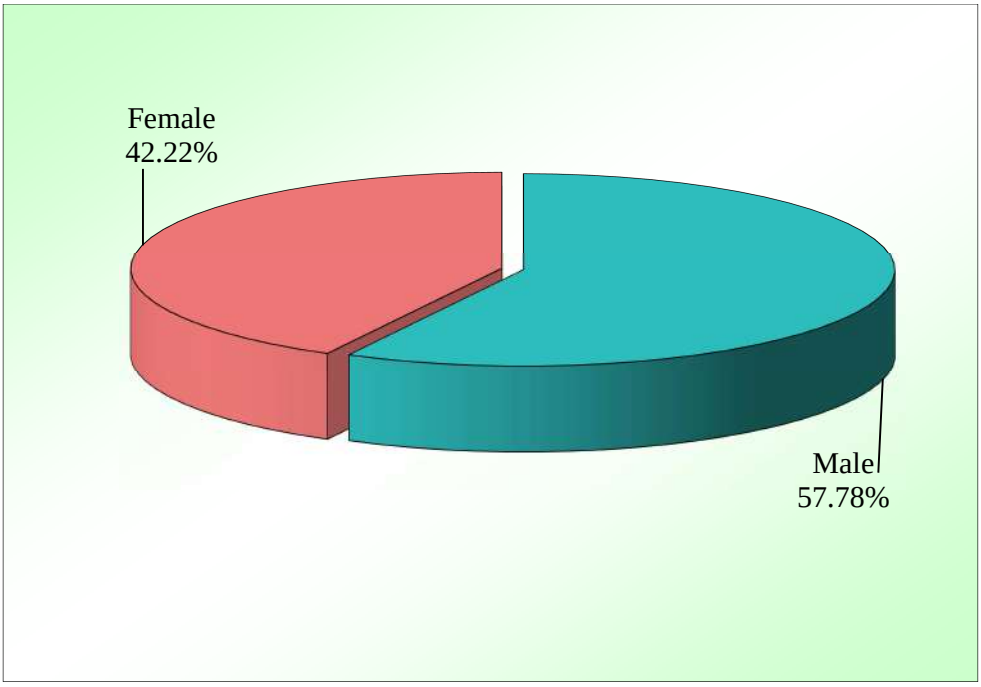



Among the cases of MA majority of the cases belonged to the age group of 2-5 years (38.2 %) followed by 6-10 years (36.2%) with both the age groups having 19 cases each. The mean age of MA was 5.32 with a standard deviation of 3.93.

**Table 4: Gender wise distribution of children in the study group**

| Gender | No of patients | % of patients |
|---|---|---|
| Male | 104 | 57.78 |
| Female | 76 | 42.22 |
| Total | 180 | 100.00 |

**Graph 4: Pie chart of gender wise distribution of children**

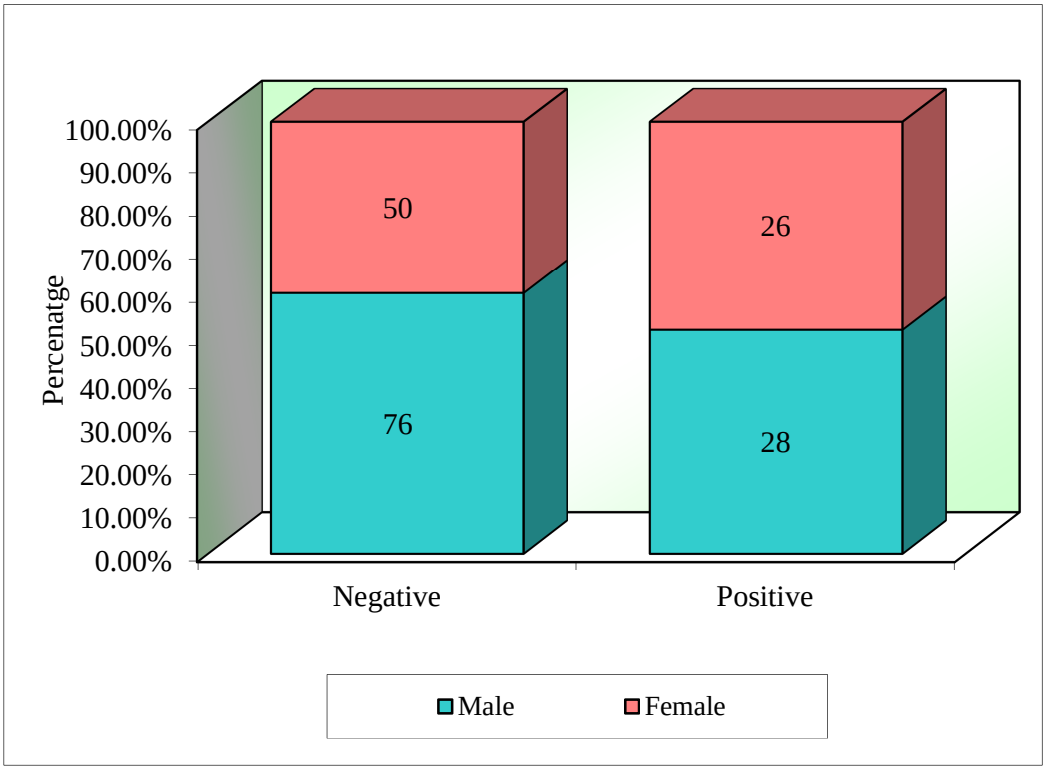



The male to female ratio in the study population was 1.3: 1, with a little male predominance.

**Table 5: Comparison between age groups of children with mineralizing angiopathy**

| Gender | Non- MA | % | MA | % | Total | % |
|---|---|---|---|---|---|---|
| Male | 76 | 73.08 | 28 | 26.92 | 104 | 57.78 |
| Female | 50 | 65.79 | 26 | 34.21 | 76 | 42.22 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Chi-square=1.1100, p=0.2920 | | | | | | |

**Graph 5: Association between age groups of patients with Prevalence of mineralizing angiopathy**

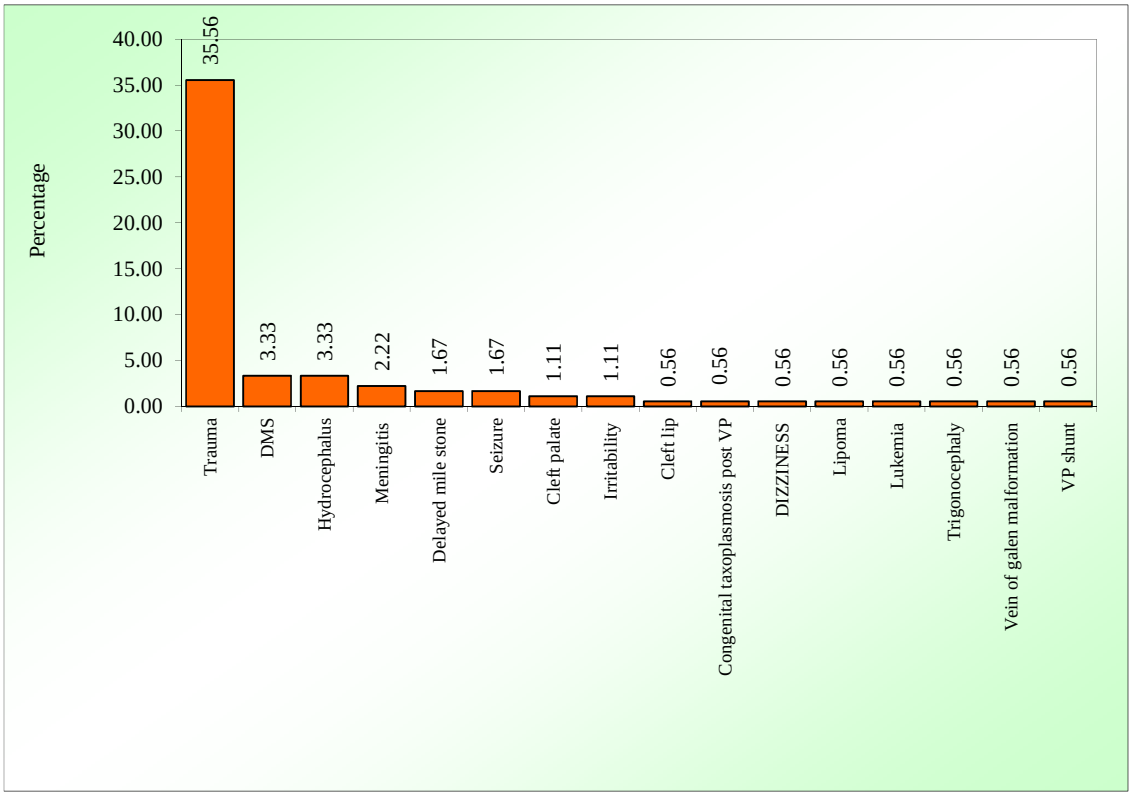



MA was found in 28 boys (26.92%) and 26 girls (34.2%) with a ratio of 1.07: 1 and a p value of 0.29 which states that occurrence of MA is independent of gender.

**Table 6: Indications for computed tomography scan of the head among the study population**

| Indications for the scan | Number | % |
|---|---|---|
| Trauma | 64 | 35.556 |
| Delayed mile stones | 9 | 4.990 |
| Hydrocephalus | 6 | 3.333 |
| Meningitis | 4 | 2.222 |
| Seizure | 3 | 1.667 |
| Cleft palate | 2 | 1.111 |
| Irritability | 2 | 1.111 |
| Cleft lip | 1 | 0.556 |
| Congenital taxoplasmosis post VP shunt | 1 | 0.556 |
| Dizziness | 1 | 0.556 |
| Lipoma | 1 | 0.556 |
| Lukemia | 1 | 0.556 |
| Trigonocephaly | 1 | 0.556 |
| Vein of galen malformation | 1 | 0.556 |
| VP shunt | 1 | 0.556 |

**Graph 6: Indications for the scan**




The most common indication for the scan among children were head trauma followed by delayed mile stones and hydrocephalus, followed altered sensorium.

The indication for the san was done for unrelated conditions.

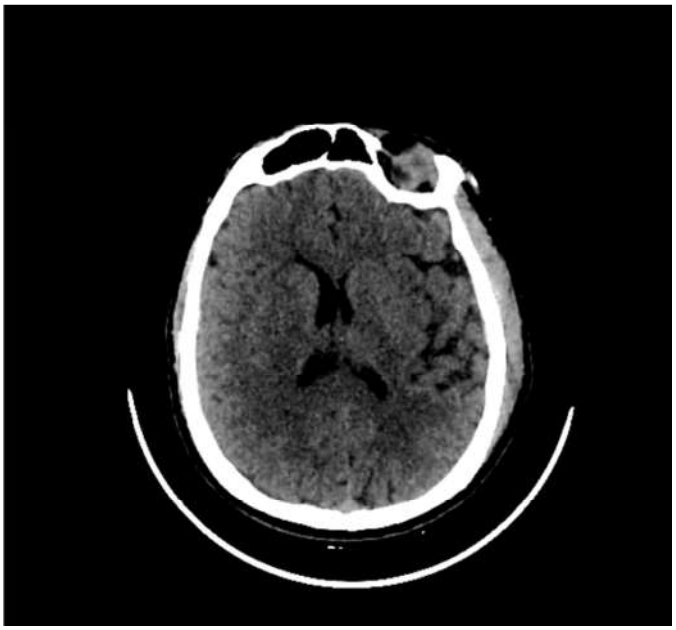


**Figure 11: A 10 year old child who has presented with a history of trauma with sub-galeal hematoma in the left fronto-parietal region.**

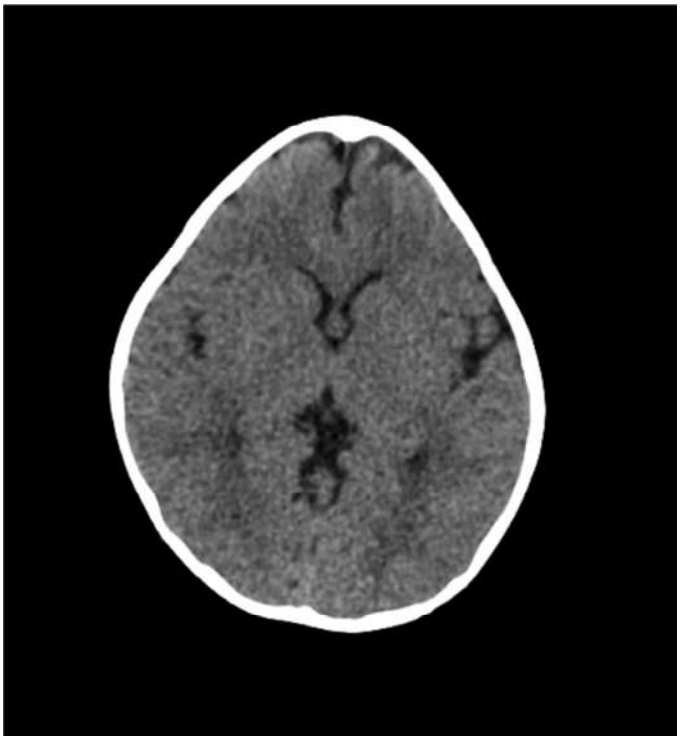


**Figure 12: 10 months old child with trigonocephaly**

After computed tomography scan of the brain was performed in patients

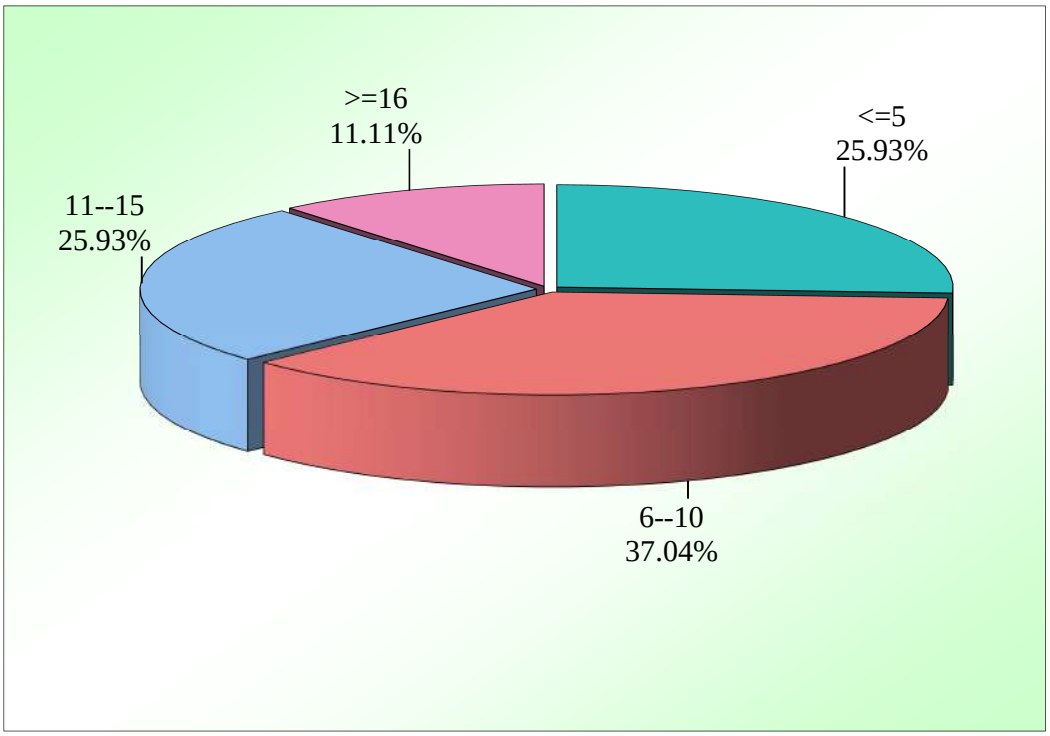
We analysed all the scans for the presence of calcifications.

In the cases of mineralizing angiopathy the number of calcifications was counted at the level of the basal ganglia using for 8 to 10 slices at a slice interval of 0.63 mm.

**Table 7: Number of calcifications found in cases of MA.**

| Number of calcifications | No of patients | % of patients |
|---|---|---|
| <=5 | 14 | 25.93 |
| 6—10 | 20 | 37.04 |
| 11—15 | 14 | 25.93 |
| >=16 | 6 | 11.11 |
| Total | 54 | 100.00 |
| Mean | 9.43 | |
| Median | 9.00 | |
| SD | 4.53 | |

**Graph 7: Pie chart: Number of calcifications**

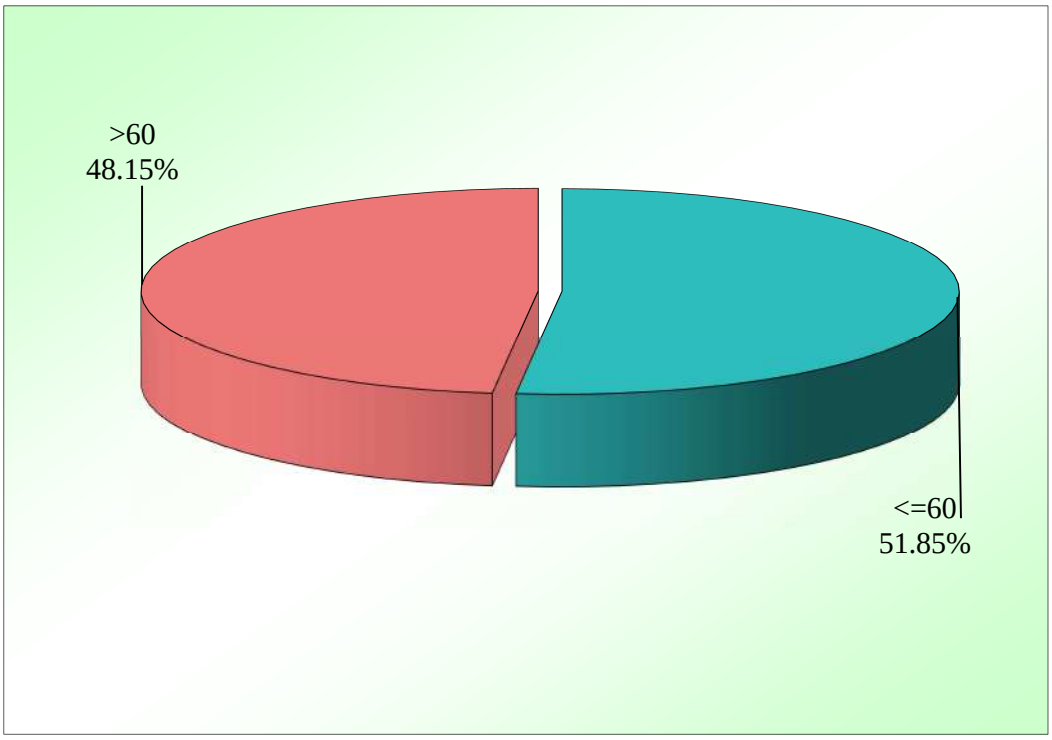



Majority of cases of MA had 6-10 calcific foci in 37% of the cases and the mean foci of calcification in cases were 9.43 with a standard deviation of 4.53.

**Table 8: Average HU of calcification among cases of MA**

| Average HU | No of patients | % of patients |
|---|---|---|
| <=60 | 28 | 51.85 |
| >60 | 26 | 48.15 |
| Total | 54 | 100.00 |
| Mean | 60.41 | |
| Median | 61.00 | |
| SD | 4.95 | |

**Graph 8: Average HU among cases of MA**




The average HU (housefield unit) of the calcific foci was +60.47 with a standard deviation of 4.95.

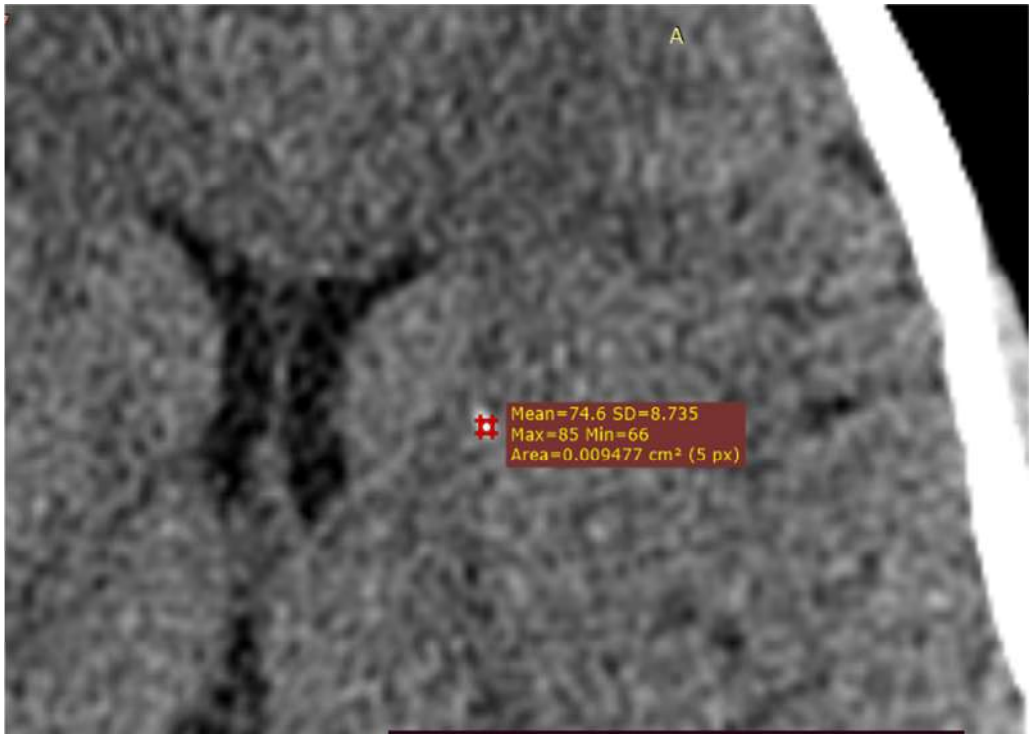



**Figure 13: HU of calcification was taken by placing ROI (region of interest) on the calcification and taking the mean value**.

**Table 9: Symmetry of calcifications among the study population**

| Symmetrical or asymmetrical calcifications | No of patients | % of patients |
|---|---|---|
| Bilateral basal ganglia | 13 | 24.07 |
| Lt basal ganglia | 23 | 42.59 |
| Rt basal ganglia | 18 | 33.33 |
| Total | 54 | 100.00 |

The calcific foci were symmetrical in 13 cases (24.0%) and were involving the left basal ganglia in 23 cases (42.5%) and the right basal ganglia in 18 cases (33.3 %).

**Graph 9: Pie chart Symmetrical or asymmetrical calcifications among the study population.**

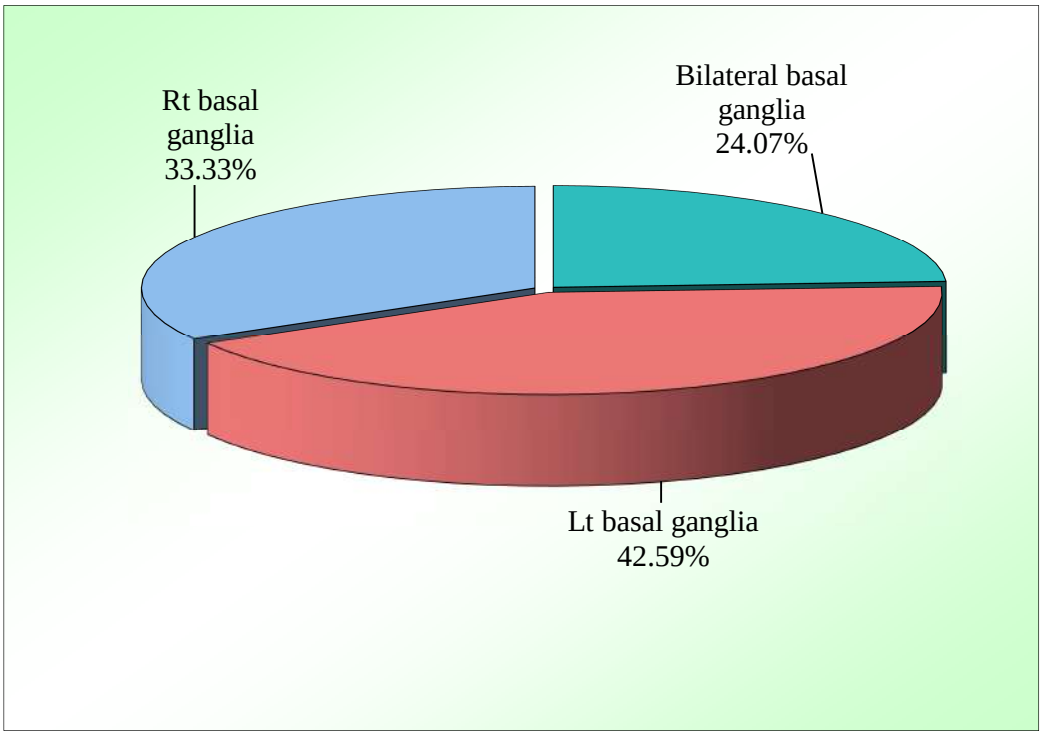
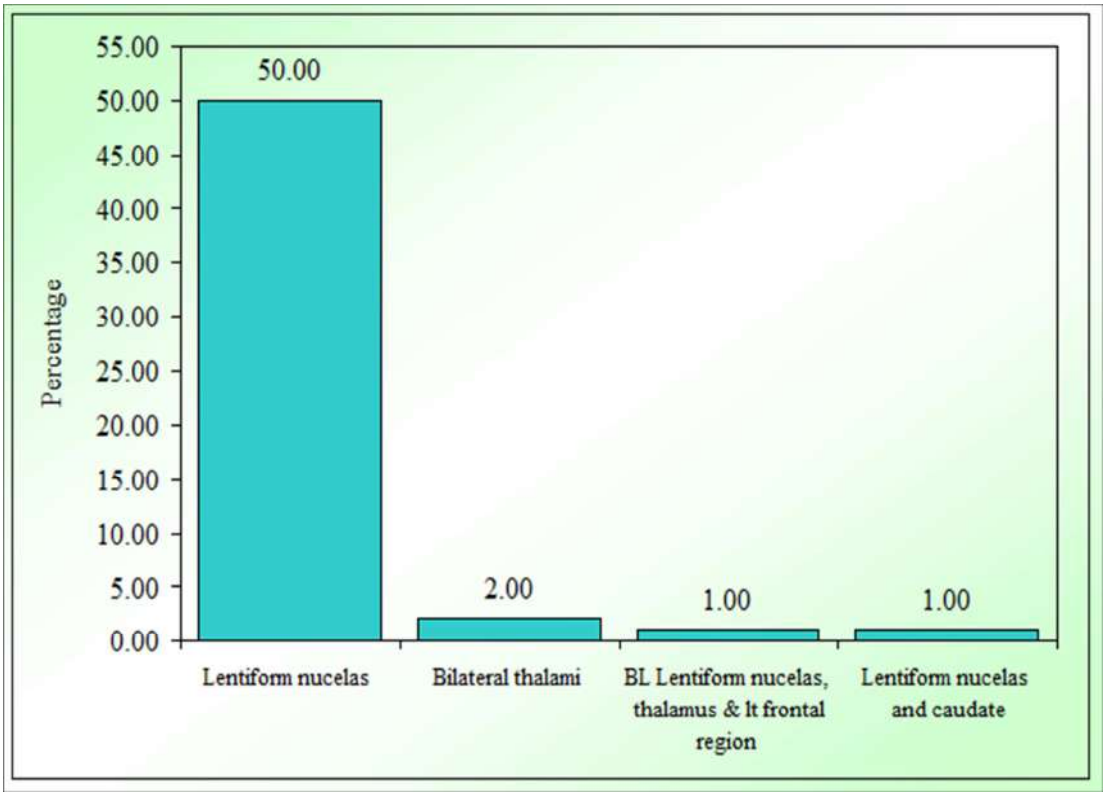


**Table 10: Location of the calcifications among the study population**

| Location of the calcifications | No of patients | % of patients |
|---|---|---|
| Lentiform nucleus alone | 50 | 92.59 |
| Bilateral thalami | 2 | 3.70 |
| Bilateral lentiform nucleus, thalamus & lt frontal region | 1 | 1.85 |
| Lentiform nucleus and caudate nucleus | 1 | 1.85 |
| Total | 54 | 100.00 |

**Graph 10: Pie chart: location of the calcifications among the study population**



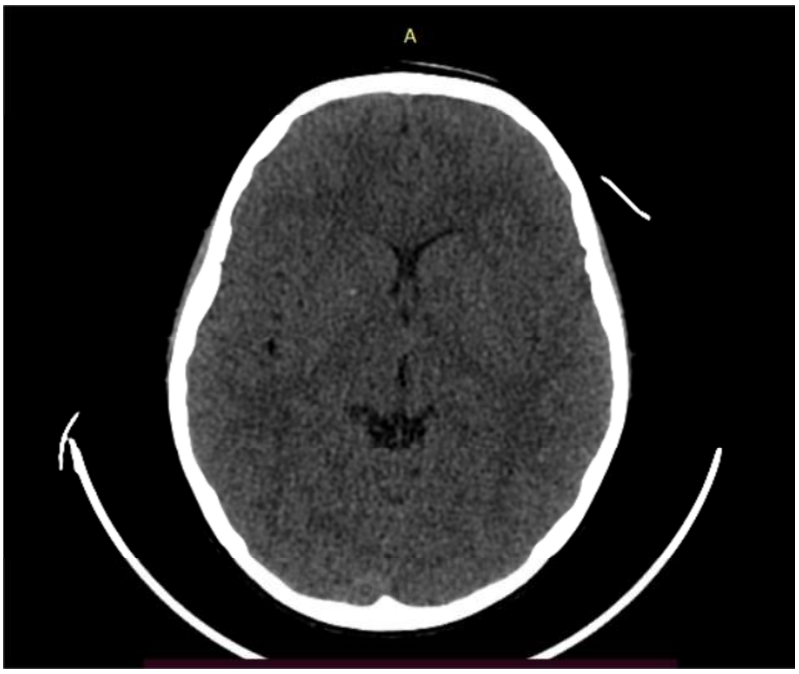



**Figure 14: Axial CT image showing calcification in the right lentiform nucleus in a 9 year old male child with mineralizing angiopathy**

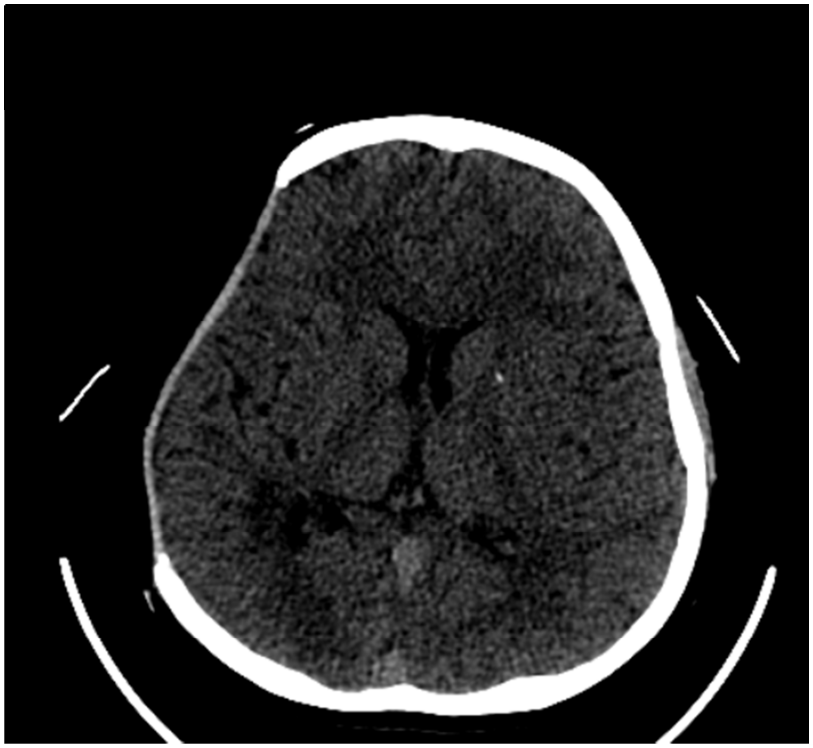


**Figure 15: Axial CT image of a 12 year old male child with mineralizing angiopathy showing calcification in the left lentiform nucleus, the child underwent decompressive criniectomy for evacuation of bleed post trauma**

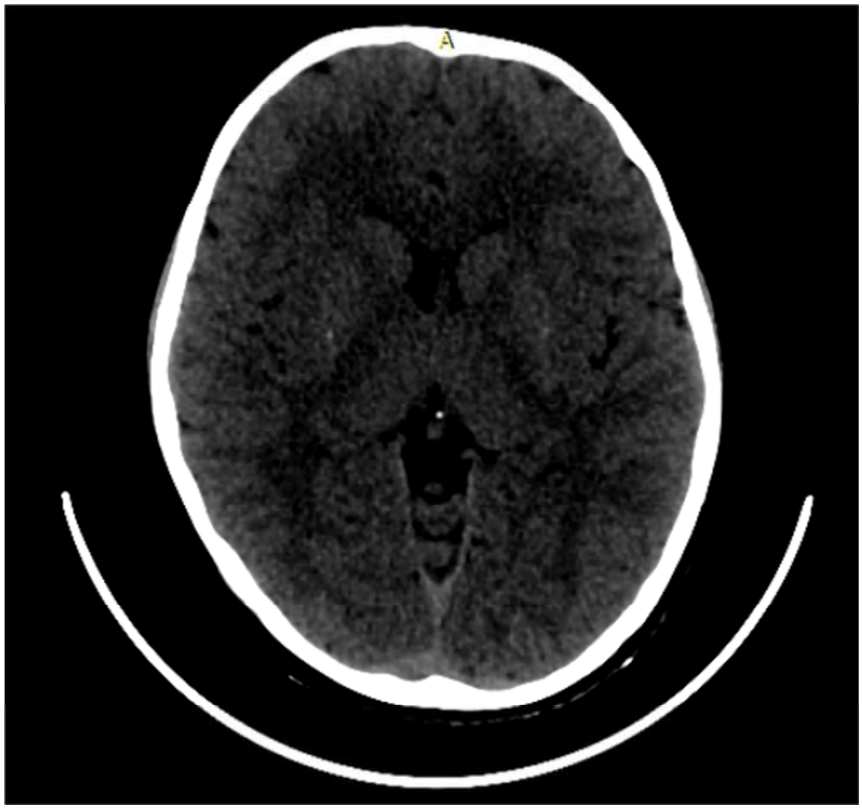


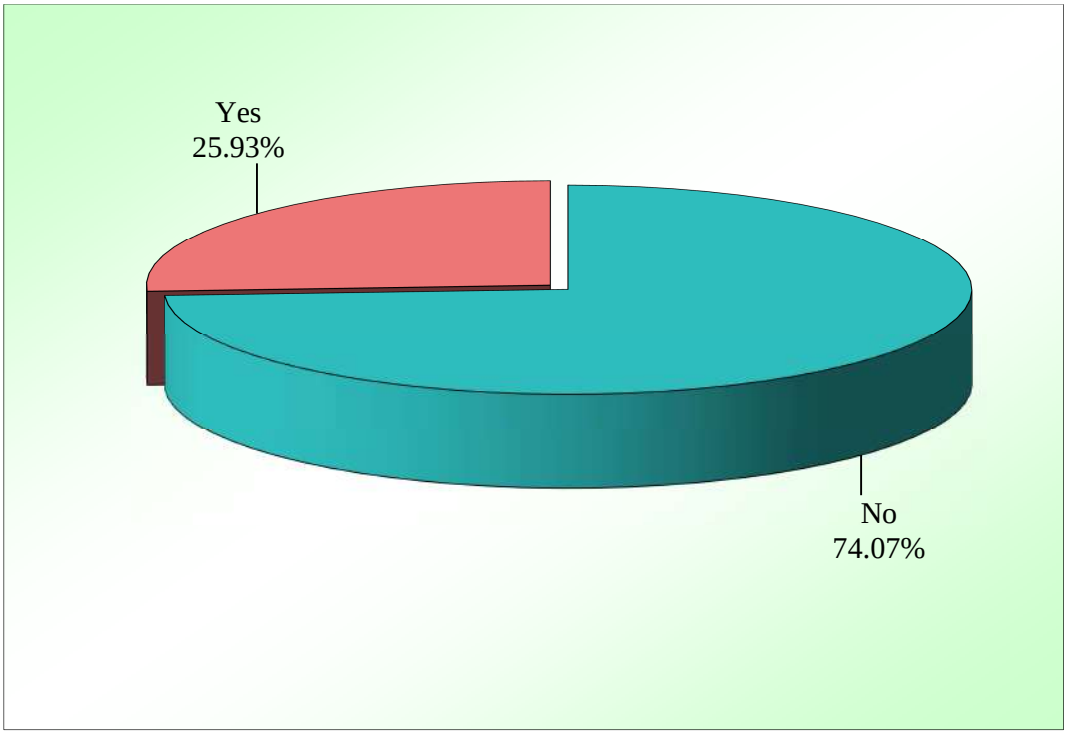

**Figure 16: Axial CT image showing calcifications in the bilateral lentiform nucleus in a 12 year old male child with MA**

Among the cases of MA by using multiplanar reconstruction the visualization of the linear foci of calcification along the lenticulostriate vessels was demonstrated

**Table 11: Linear foci of calcification on coronal reconstructions among the cases of MA**

| Calcifications on coronal reconstructions | No of patients | % of patients |
|---|---|---|
| No | 40 | 74.07 |
| Yes | 14 | 25.93 |
| Total | 54 | 100.00 |

**Graph 11: Pie chart: linear foci of calcification on coronal reconstructions**




In 25.93% (14) of the cases of MA, the reconstruction of the linear foci of calcification along the lentiulostriate arteries was possible.

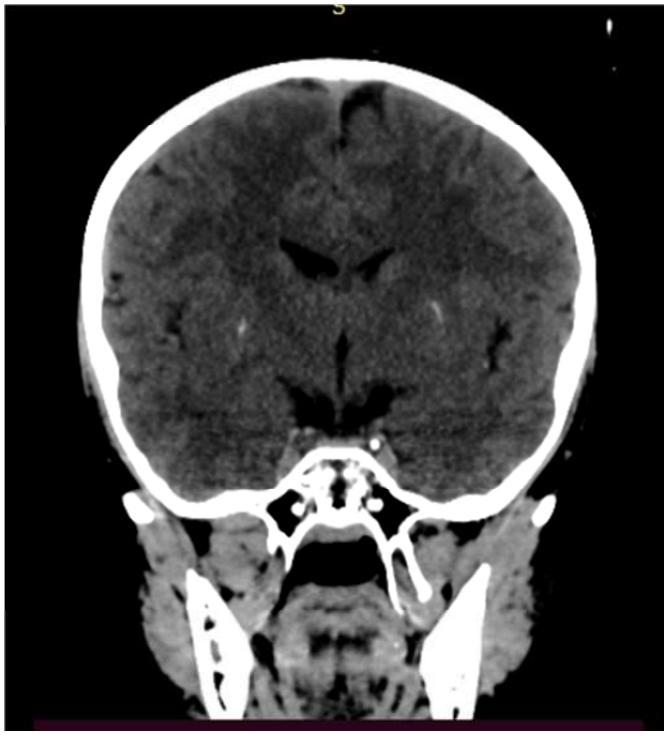


**Figure 17: Visualization of the linear foci of calcification on coronal reconstruction in a 12 year old boy**

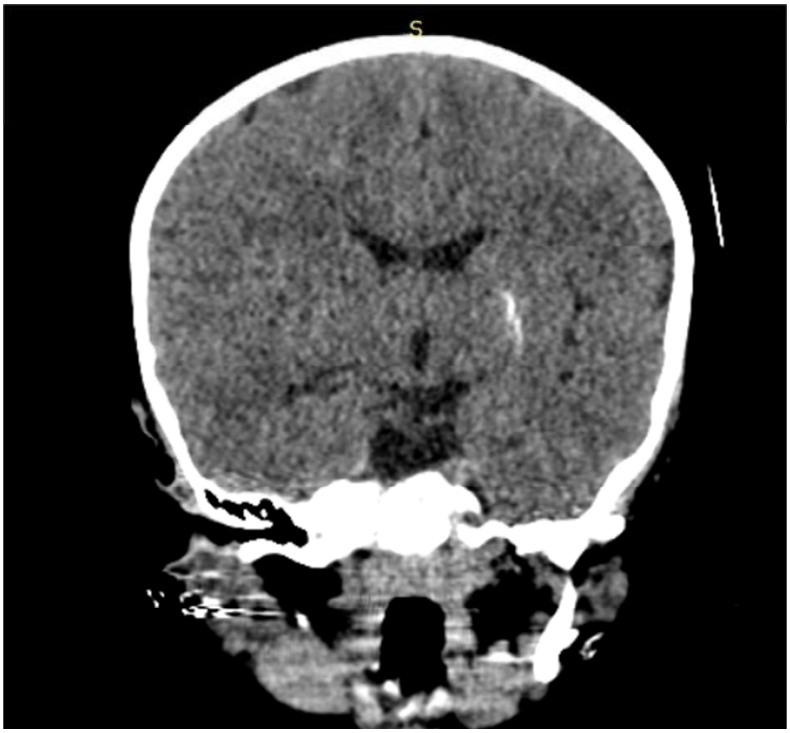
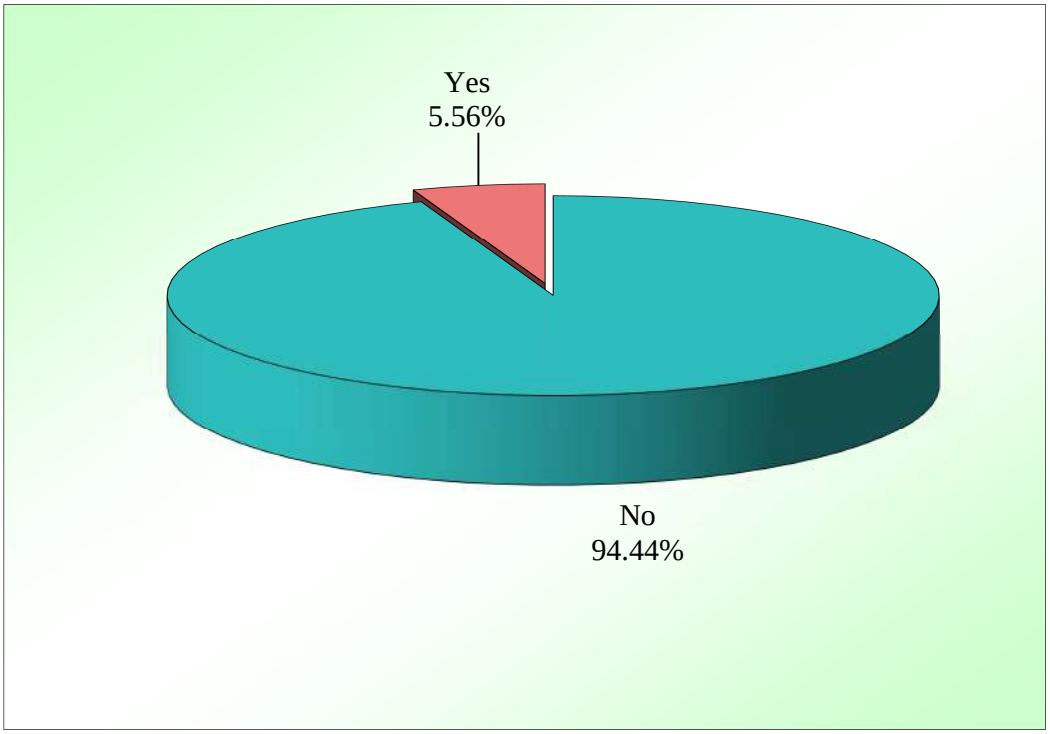


**Figure 18: Visualization of the linear foci of calcification on coronal reconstruction in a 7 year old girl with MA.**

**Table 12: Incidental calcifications found outside basal ganglia**

| Calcifications found outside basal ganglia | No of patients | % of patients |
|---|---|---|
| No | 51 | 94.44 |
| Yes | 3 | 5.56 |
| Total | 54 | 100.00 |

**Graph 12: Calcifications outside the basal ganglia was found in 5.5% cases of MA**

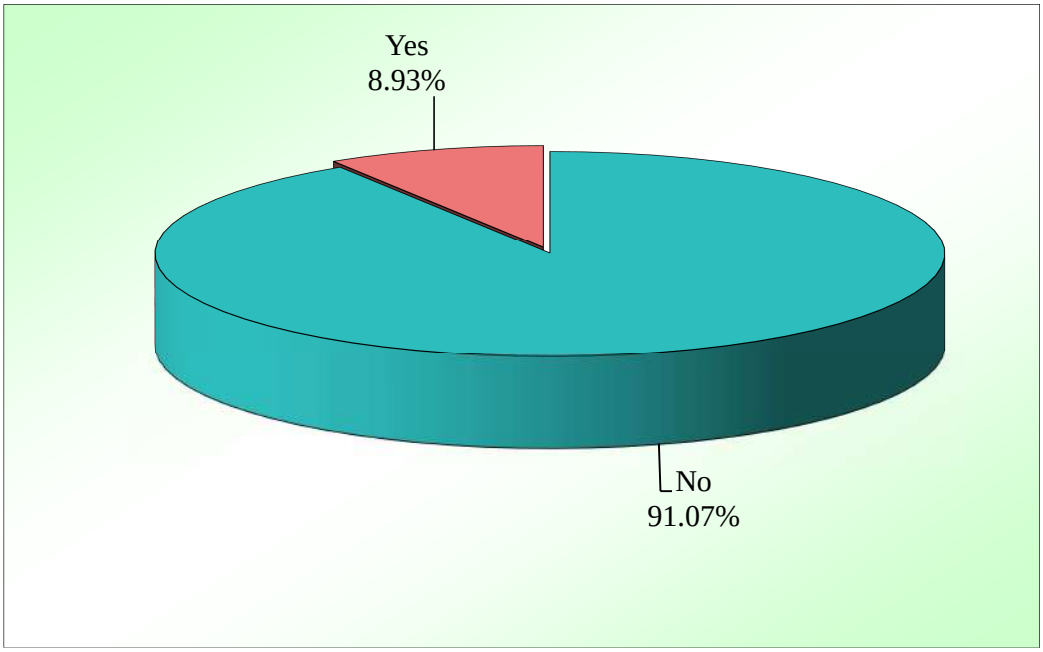



Among the 3 cases, one was a congenital toxoplasmosis, one was a case of tubercular meningitis and one was a case of vein of galen malformation.

**Table 13: No of cases of MA who stroke**

| Infarct | No of patients | % of patients |
|---|---|---|
| No | 51 | 91.07 |
| Yes | 5 | 8.93 |
| Total | 54 | 100.00 |

**Graph 13: Pie chart Stroke among cases of MA**




Among the study population 5(8.9 %) of the cases of MA (54cases) had presented with stroke.

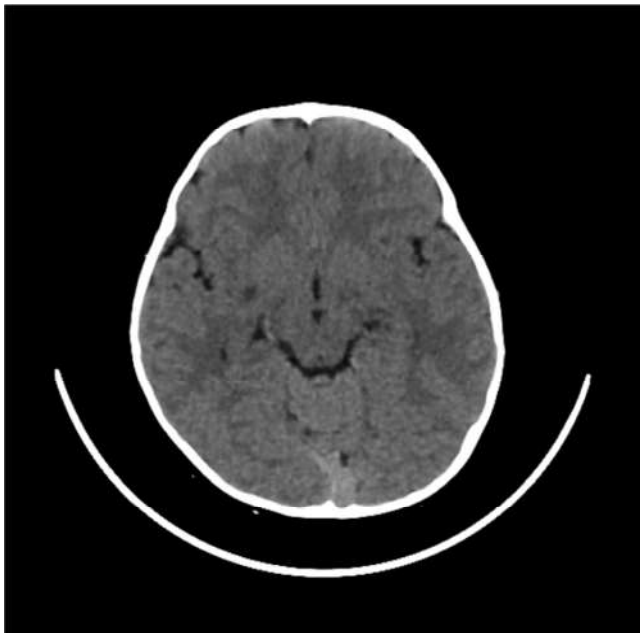
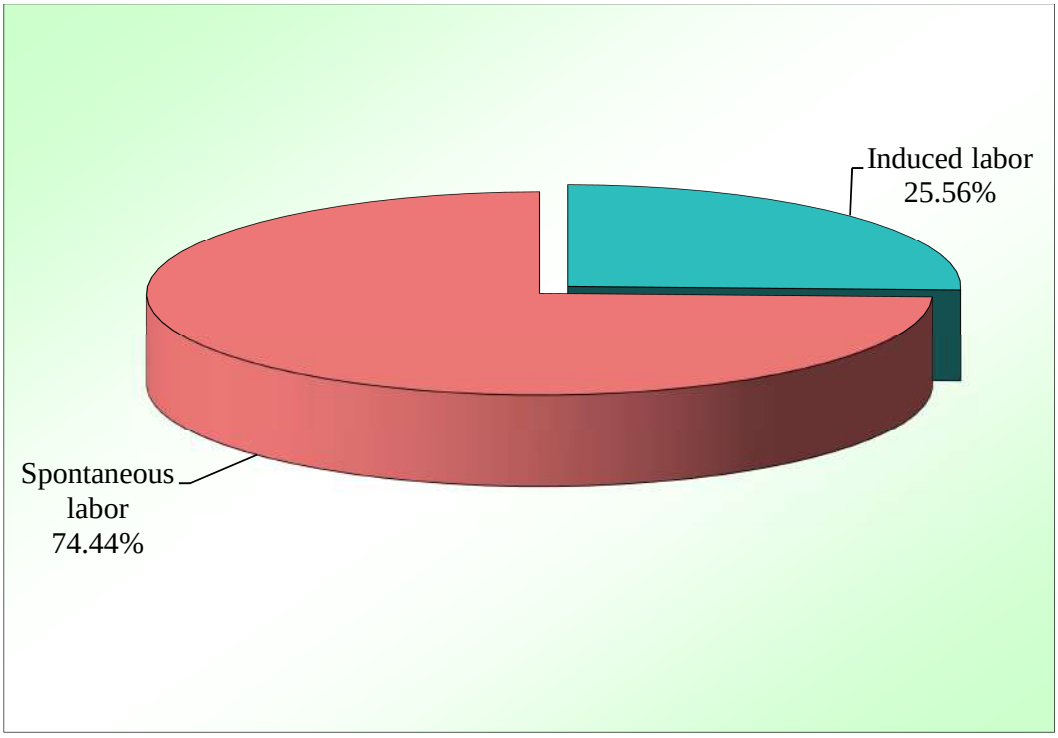


**Figure 19: Axial CT image showing a subacute infarct in the posterior aspect of the lentiform nucleus in a 2 year old girl who had history of fall followed by weakness of the left lower limb**

**Table 14: Spontaneous vs induced labour among the study population**

| Spontaneous or induced labour | No of patients | % of patients |
|---|---|---|
| Induced labour | 46 | 25.56 |
| Spontaneous | 134 | 74.44 |
| Total | 180 | 100.00 |

**Figure 14: Pie chart - Spontaneous vs induced labour among the study population**

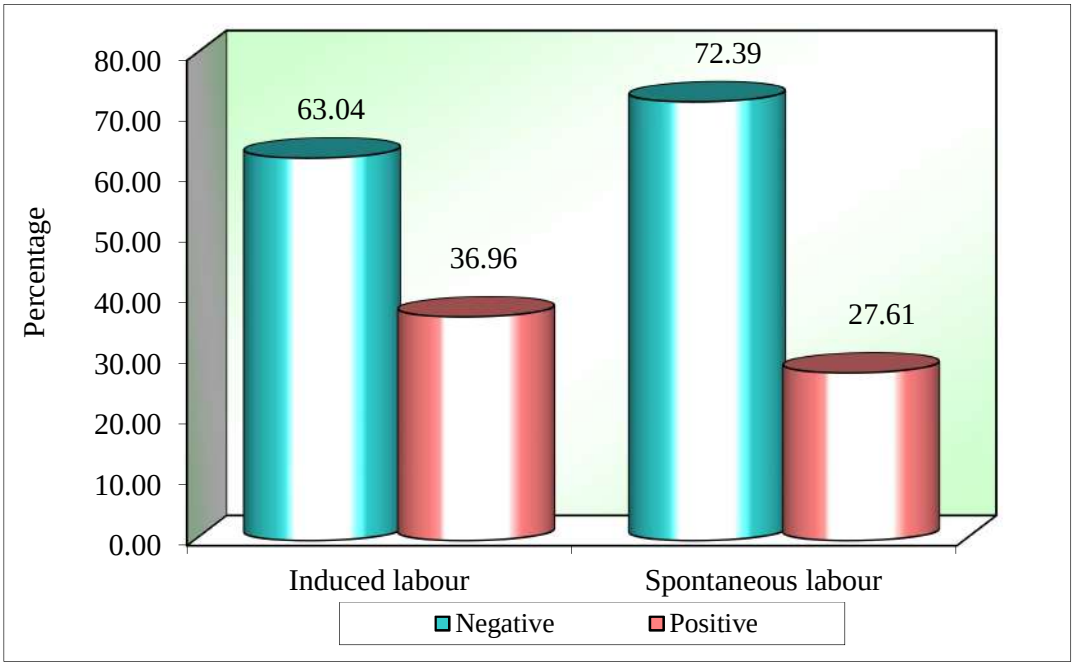



Among the study population 74.4% of the cases has spontaneous labor and 25.6% had induced labor.

**Table 15: Association between spontaneous or induced labour with Prevalence of mineralizing angiopathy**

| Spontaneous or induced labour | Negative | % | Positive | % | Total | % |
|---|---|---|---|---|---|---|
| Induced labour | 29 | 63.04 | 17 | 36.96 | 46 | 25.56 |
| Spontaneous labour | 97 | 72.39 | 37 | 27.61 | 134 | 74.44 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Chi-square=1.4240, p=0.2330 | | | | | | |

**Graph 15: Association between spontaneous or induced labour with Prevalence of mineralizing angiopathy**



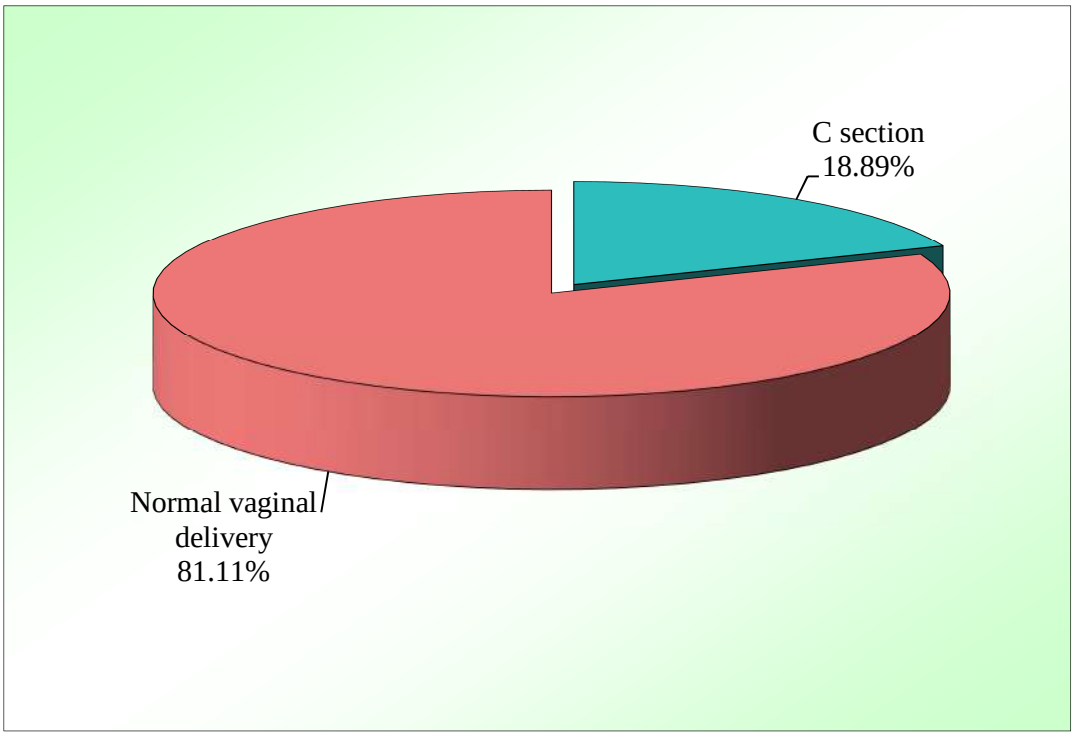

In our study, 17 cases (31.0%) of MA had induced labor and 37 cases (68%) of the cases had spontaneous labor with a p value of 0.230, the difference was not statistically significant.

Cases of MA were common in patients who had spontaneous labor and less common in less common in patients who had induced labor.

**Table 16: Mode of delivery among the study population**

| Mode of delivery | No of patients | % of patients |
|---|---|---|
| Caesarean section | 34 | 18.89 |
| Vaginal delivery | 146 | 81.11 |
| Total | 180 | 100.00 |

**Graph 16: Mode of delivery among the study population**

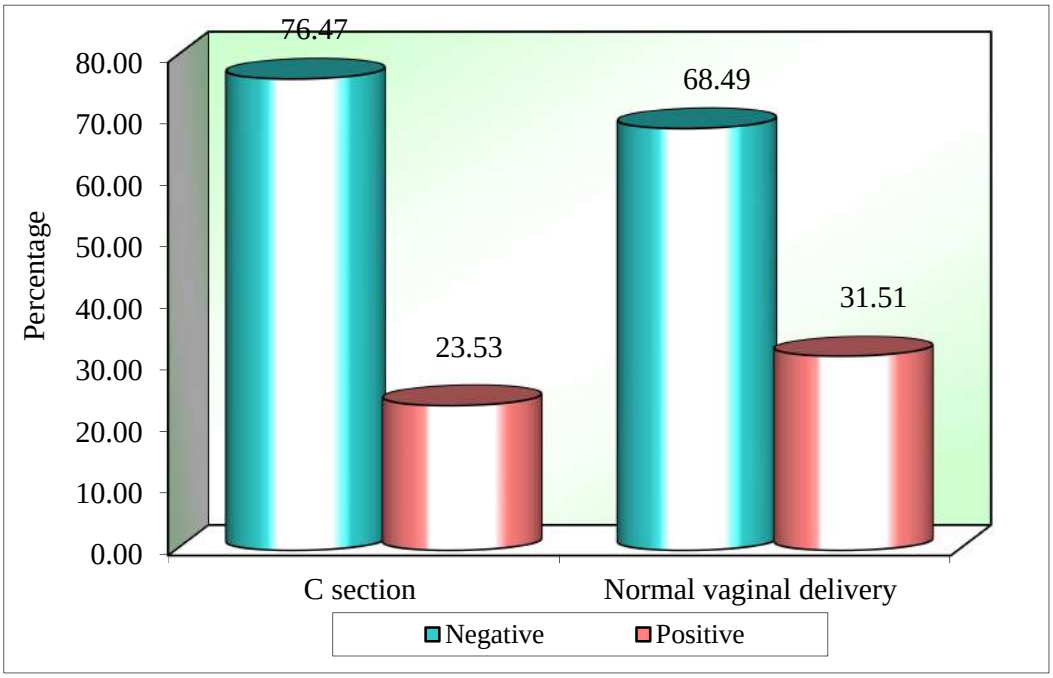



Among the study population 81.1% (146 cases) of the cases had normal vaginal delivery and 18.8% (34cases) had C-section.

**Table 17: Association between mode of delivery with Prevalence of mineralizing angiopathy**

| Mode of delivery | Negative | % | Positive | % | Total | % |
|---|---|---|---|---|---|---|
| Caesarean section | 26 | 76.47 | 8 | 23.53 | 34 | 18.89 |
| Vaginal delivery | 100 | 68.49 | 46 | 31.51 | 146 | 81.11 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Chi-square=0.8360, p=0.3610 | | | | | | |

**Graph 17: Association between mode of delivery with Prevalence of mineralizing angiopathy**



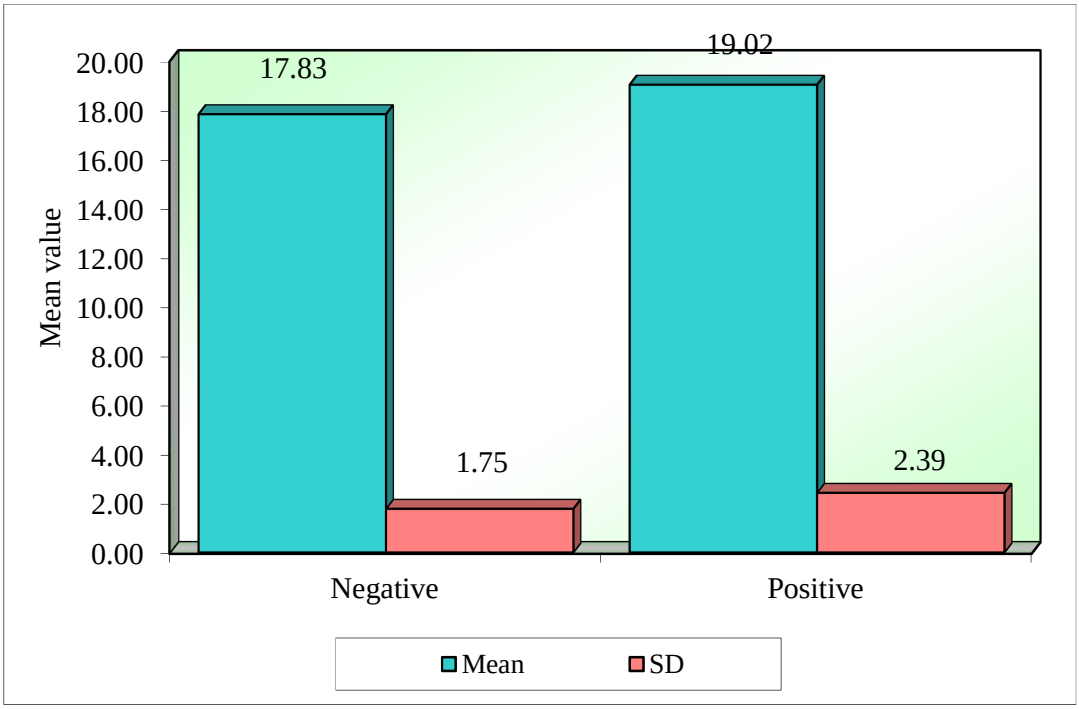

MA was present in 46(31.5%) cases of normal vaginal delivery and 8(23.5%) cases of C-section, with a a p value of 0.8 which indicates that MA was more in cases of vaginal delivery but no significant difference was made between MA group and non-MA group.

**Table 18: Comparison of status of Prevalence of mineralizing angiopathy with mean Duration of labour (Hr) by independent t test**

| Prevalence of mineralizing | n | Mean | SD | SE | t-value | P-value |
|---|---|---|---|---|---|---|
| Negative | 126 | 17.83 | 1.75 | 0.16 | -3.7159 | 0.0003* |
| Positive | 54 | 19.02 | 2.39 | 0.33 | | |

*p<0.05

**Graph 18: Comparison of status of Prevalence of mineralizing angiopathy with mean Duration of labour (Hr) based on history**



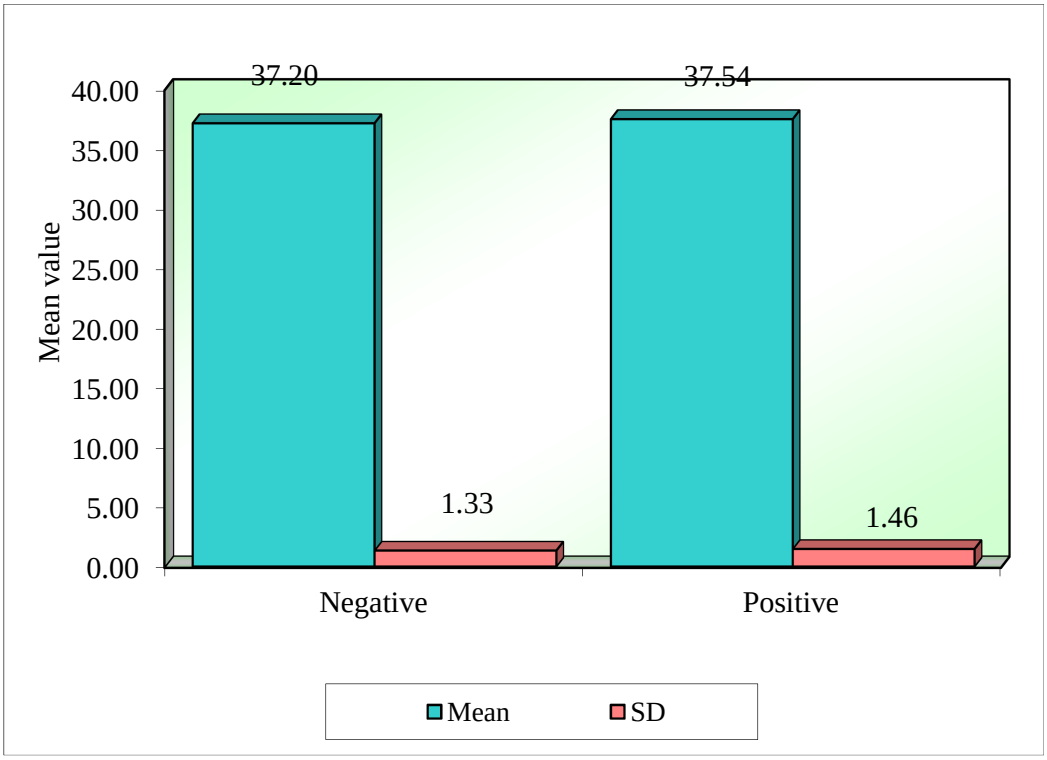

In our study detailed history of mode, duration of labour and gestational age of the children were obtained from the mother.

Cases of MA had mean duration labor around 19.02 hr with a standard deviation of 2.39 as compared to the Non-MA group which had a mean of 17.83 hr with a standard deviation of 1.75 hr. with a p value of 0.003 which indicates that the difference was statistically significant, But this was based on history

**Table 19: Comparison of status of Prevalence of mineralizing angiopathy with mean Gestational age (weeks) by independent t test**

| Prevalence of mineralizing | n | Mean | SD | SE | t-value | P-value |
|---|---|---|---|---|---|---|
| Negative | 126 | 37.20 | 1.33 | 0.12 | -1.5211 | 0.1300 |
| Positive | 54 | 37.54 | 1.46 | 0.20 | | |

**Graph 19: Comparison of status of Prevalence of mineralizing angiopathy with mean Gestational age based on history**



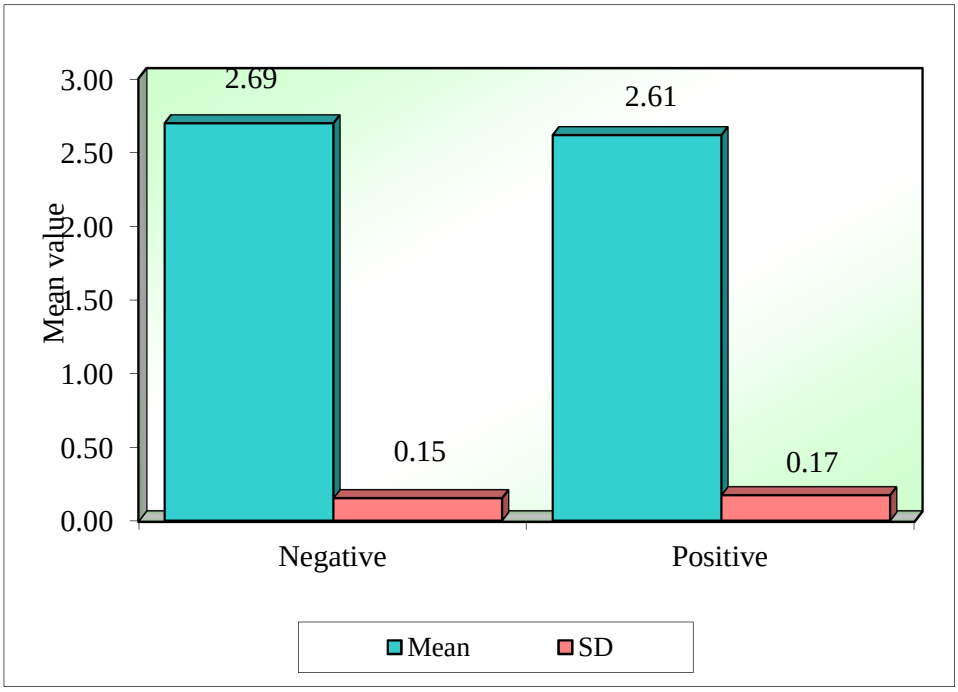

The mean gestational age among the MA group was 37.54 weeks with a standard deviation of 1.46 weeks and among the non-ma group was 37.2 with a standard deviation of 1.33 weeks. The association between the MA group and non-MA group was not significant.

**Table 20: Comparison of status of Prevalence of mineralizing angiopathy with mean Birth weight by independent t test**

| Prevalence of mineralizing | n | Mean | SD | SE | t-value | P-value |
|---|---|---|---|---|---|---|
| Negative | 126 | 2.69 | 0.15 | 0.01 | 3.3193 | 0.0011* |
| Positive | 54 | 2.61 | 0.17 | 0.02 | | |

*p<0.05

**Graph 20: Comparison of status of Prevalence of mineralizing angiopathy with mean Birth weight based on history**

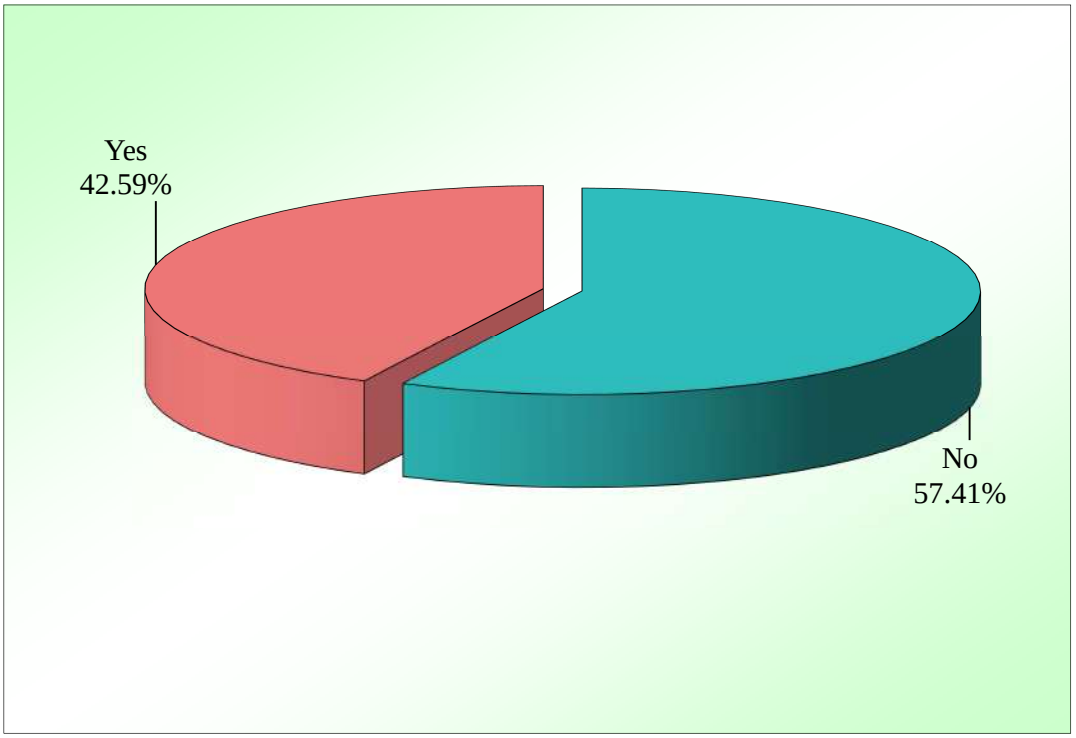



The mean birth weight among the MA group was 2.61kg with a standard deviation of 0.17 kg and among the non-ma group was 2.69 with a standard deviation of 0.15kgs. The association between the MA group and non-MA group was significant. Cases of MA had a statistically significant difference with birth weight.

**Table 21: History of PROM (premature rupture of membranes) among MA group**

| PROM (premature rupture of membranes) | No of patients | % of patients |
|---|---|---|
| No | 31 | 57.41 |
| Yes | 23 | 42.59 |
| Total | 54 | 100.00 |

**Graph 21: pie chart -History of PROM (premature rupture of membranes) among MA group**

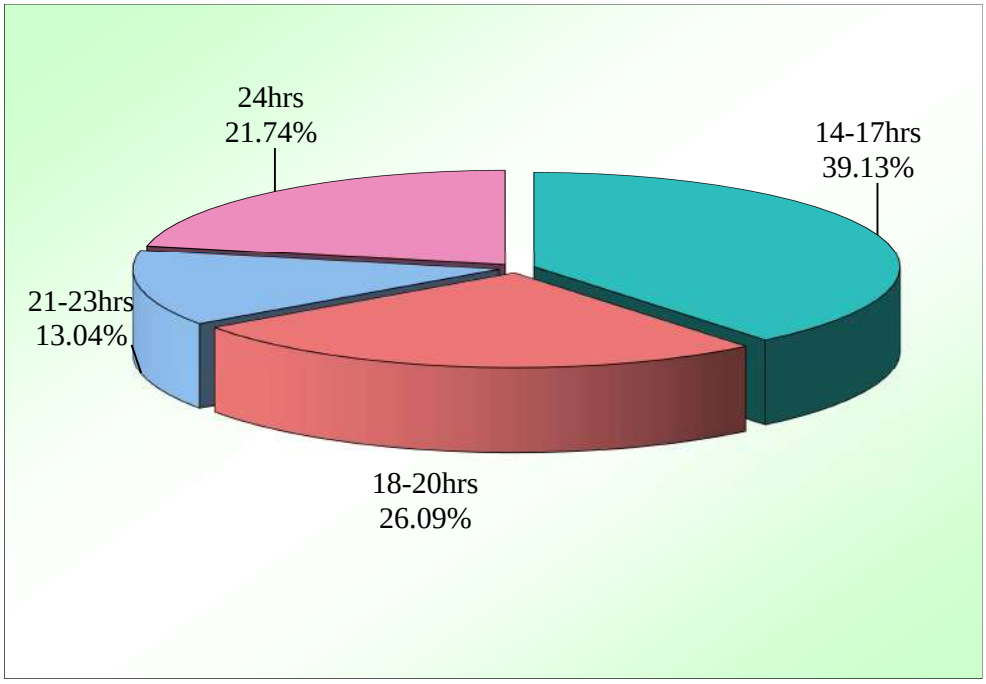



23 (42.59%) patients in the MA group had history of PROM and 31 patients of MA did not have history of PROM.

**Table 22: Duration of PROM (premature rupture of membranes) among MA group**

| Duration of PROM | No of patients | % of patients |
|---|---|---|
| 14-17hrs | 9 | 39.13 |
| 18-20hrs | 6 | 26.09 |
| 21-23hrs | 3 | 13.04 |
| 24hrs | 5 | 21.74 |
| Total | 23 | 100.00 |
| Mean | 18.74 | |
| SD | 3.63 | |

**Graph 22: Pie chart: Duration of PROM (premature rupture of membranes) among the MA group by history**




The duration of mean duration of PROM was obtained from history in the MA group was 18.74hr with a standard devition of 3.63 hr. The majority of the cases of MA 9 (39.3%) were in the group of 14-17 hours.

**Table 23: comparison of the duration of PROM with no of calcifications and HU of the calcifications.**

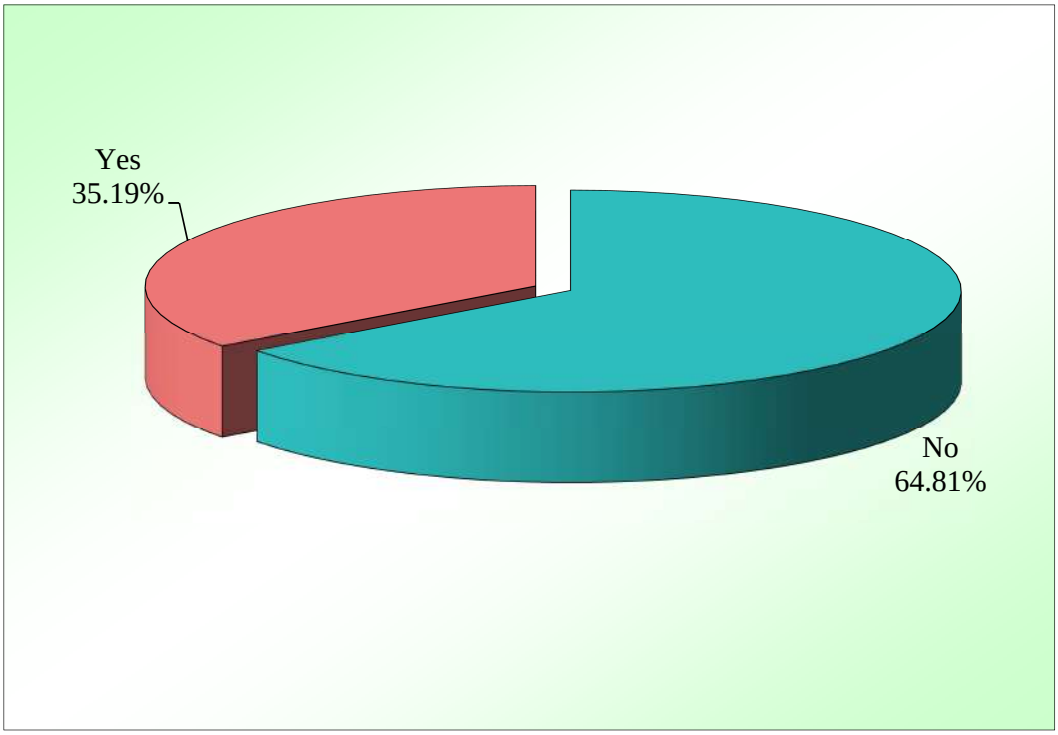
| Duration of PROM | No of patients | Percentage of patients | Average number of calcifications | | Average HU of calcifications | |
|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD |
| >6 hrs | 3 | 13.04 | 3.7 | 0.8 | 58 | 4.8 |
| 06-24 hrs | 6 | 26.09 | 9.55 | 3.7 | 61.2 | 4.3 |
| >24hrs | 5 | 21.74 | 6.6 | 2.3 | 59.1 | 2.7 |

The average number of calcifications and average HU of the calcifications was more when the increased duration of PROM was in the range of 6 to 24 hours and more than 24 hours

**Table 24: History of NICU (neonatal intensive care unit) admissions among the MA group**

| NICU admissions | No of patients | % of patients |
|---|---|---|
| No | 35 | 64.81 |
| Yes | 19 | 35.19 |
| Total | 54 | 64.81 |

**Graph 23: History of NICU admissions among the MA group**

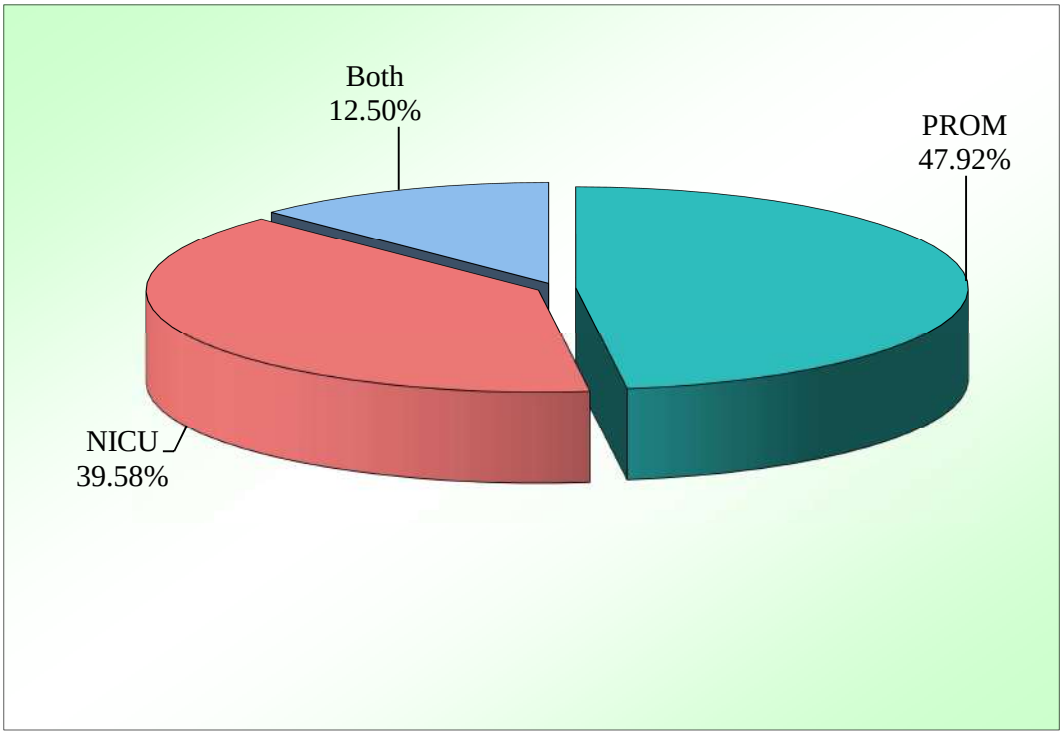



Among the MA group, 19 patients (35.19%) had history of NICU admissions post-delivery and 35 patients (64.18%) did not have history of NICU admissions.

**Table 25: Distribution of patients with PROM, NICU and both**

| | No of patients | % of patients |
|---|---|---|
| PROM | 23 | 42.59 |
| NICU | 19 | 35.19 |
| Both | 6 | 11.11 |
| Total | 54 | 64.81 |

**Graph 24: Distribution of patients with PROM, NICU and both**




Among the MA group 6 patients had history of both NICU admission and PROM history.

**Table 26: Children with history of PROM or NICU or both among the MA group.**

| | No of patients | % of patients |
|---|---|---|
| PROM | 23 | 42.59 |
| NICU | 19 | 35.19 |
| PROM+ NICU | 41 | 77.78 |
| Both | 6 | 11.11 |

**Graph 25: Distribution of patients with PROM, NICU and both**

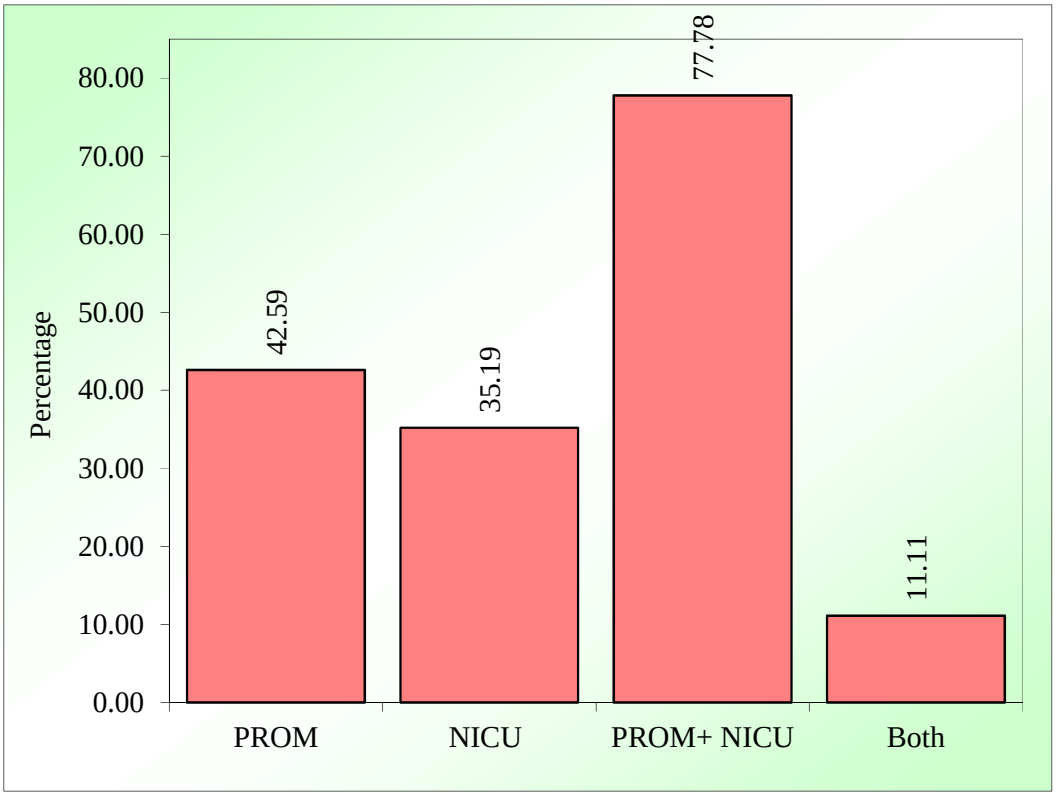
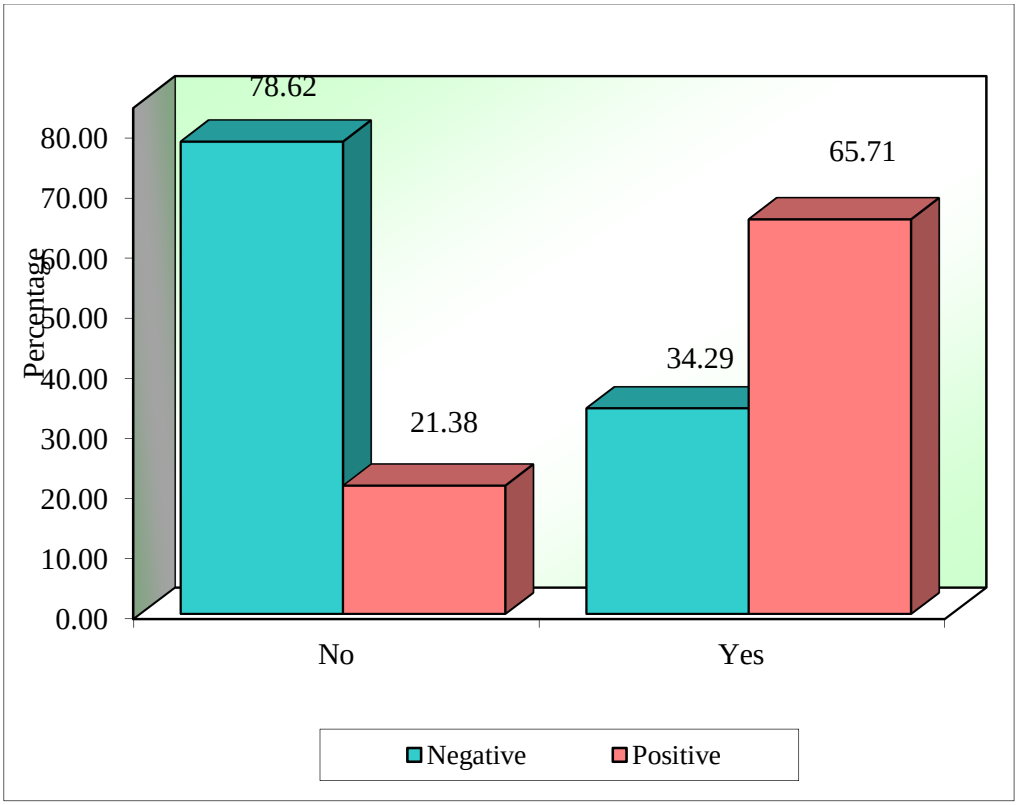



**Table 27: Association between H/O PROM with Prevalence of mineralizing angiopathy**

| H/O PROM | Negative | % | Positive | % | Total | % |
|---|---|---|---|---|---|---|
| No | 114 | 78.62 | 31 | 21.38 | 145 | 80.56 |
| Yes | 12 | 34.29 | 23 | 65.71 | 35 | 19.44 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Chi-square=26.3900, p=0.0001* | | | | | | |

*p<0.05

**Graph 26: Association between H/O PROM with Prevalence of mineralizing angiopathy**



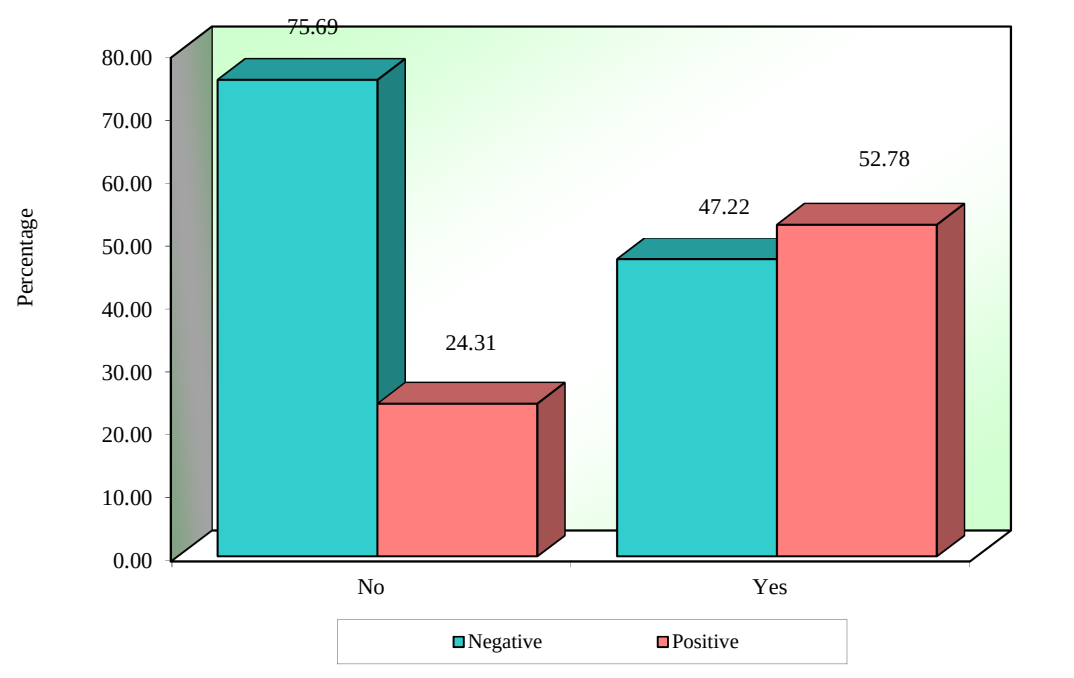

Among the study group 35 patients had history of prom out of which 25(65.71) cases belonged to the MA group and 12(34.29%) belonged to the non MA group. On statistical analysis we got a p value of 0.0001. there is a statistically significant relation of history of PROM and MA.

**Table 28: Association between H/O NICU admissions with Prevalence of mineralizing angiopathy**

| H/O NICU admissions | Negative | % | Positive | % | Total | % |
|---|---|---|---|---|---|---|
| No | 109 | 75.69 | 35 | 24.31 | 144 | 80.00 |
| Yes | 17 | 47.22 | 19 | 52.78 | 36 | 20.00 |
| Total | 126 | 70.00 | 54 | 30.00 | 180 | 100.00 |
| Chi-square=11.1180, p=0.0010* | | | | | | |

*p<0.05

**Graph 27: Association between H/O NICU admissions with Prevalence of mineralizing angiopathy**




Among the study group 36 (20%) patients had history of NICU admission out of which 19(52.78%) cases belonged to the MA group and 17(47.22%) belonged to the non MA group. On statistical analysis we got a p value of 0.0010. This gives us a statistically significant correlation of history of NICU and MA.

# DISCUSSION

The present hospital based observational study was done on 180 children aged between 6 months and 12 years who had underwent a brain computed tomography study for various indications, referred to the department of radio-diagnosis in a tertiary care institute. CT of the brain was carried out with a 128 slice CT machine.

The present study lays the groundwork for a distinct clinico-radiological entity described by Lingappa et al.[21] in 2014, which they called 'mineralizing angiopathy who suffered a minor injury followed by a basal ganglia stroke.

After taking consent from parents detailed parenteral interview will be conducted to look for perinatal infections and clinical findings will be noted, among the MA and non-MA group.

Prevalence of mineralizing angiopathy in the study population (children aged between 6 months to 12 years) was 29.67%.

In the current study, mean age of the children was 5.68 years. Most of the children belonged to the age group of 6 to 10 years (39.4%) followed by 2 to 5 years (38.3%).

Among the cases of MA majority of the cases belonged to the age group of 2-5 years (38.2 %) followed by 6-10 years (36.2%) with both the age groups having 19 cases each. The mean age of children with MA was 5.32 years in the present study. Majority of the cases of MA belonged to 1 to 5 years.

In the present study the male to female ratio in the study population was 1.3: 1, with a little male predominance. MA was found in 28 boys (26.92%) and 26 girls

(34.2%) with a ratio of 1.07: 1 and a p-value of 0.29 which states that occurrence of MA is independent of gender.

The most common indication for CT head among the study population was trauma followed by altered sensorium, delayed mile stones and hydrocephalus, the indication for the scan was for unrelated conditions.

In the present study among the cases of MA Majority of them had 6-10 calcific foci in 37% of the cases and the mean foci of calcification in cases were 9.43 with a standard deviation of 4.53. The number of calcific foci in a study done by Lingapppa was one to five on each side.[21] Five of the patients of MA had also underwent MRI examination which did not show evidence calcifications or blooming on SWI sequence. MRI is not sensitive for detection of MA.

The average HU of the calcific foci was +60.47 with a standard deviation of 4.95 is in accordance with the study done by Lingappa and others[21] were the attenuation values ranged from 58 to 90 HU.

Calcifications outside the basal ganglia were found in 3 cases. Among the 3 cases, one was a case of congenital toxoplasmosis, one was a case of tubercular meningitis and one was a case of vein of galen malformation, these cases also had calcifications in the basal ganglia.

Visualization of the linear foci of calcification along the lenticulostriate vessels was demonstrated in coronal reconstruction in 25.93% (14 of the cases of MA). Similar linear foci was described in studies.[14,20] This can be attributed to that the calcifications were not dense in all the cases owing to its difficult reconstruction on coronal images.

Most the cases of MA either did not have symptoms or were found incidentally on brain CT and unrelated indications.

Only 5 cases (8.9 %) of the cases of MA (54cases) had presented with stroke after minor trauma/fall. The occurrence of stroke among the children in the present study was just 8.9%, this means to tell us that not all cases of MA will present with stroke after minor fall, but they are a risk factor for paediatric stroke after minor fall. This was also brought forward by Kamate and others.[32]

Few cases of Lenticulostriate vasculopathy can show calcifications on CT. Many children with LSV who fall may have stroke. Lenticulostriate vasculopathy on USG mimics calcification. LSV has a mean risk age group of 1 to 5 years.[30]

Stroke occurs in these kids because lenticulostriate arteries are very fragile thi intima media thickness may be thin in children.

Also kids in the age group of 1 of 5 years have a large head and haven't had developed good balance.

In our study, 17 cases (31.0%) of MA had induced labour and 37 cases (68%) of the cases had spontaneous labour the difference was not statistically significant.

Cases of MA were common in patients who had spontaneous labour and less common in less common in patients who had induced labour.

MA was present in 46 (31.5%) cases of normal vaginal delivery and 8(23.5%) cases of C-section, MA was more common in cases of vaginal delivery.

The mean duration of labour was longer in cases of MA with a mean of 19.02 hr with a standard deviation of 2.39 as compared to the Non-MA group which had a mean of 17.83 hr with a standard deviation of 1.75 hr. this data was not clinically significant or important.

The mean gestational age among the MA group was 37.54 weeks with a standard deviation of 1.46 weeks and among the non-ma group was 37.2 with a standard deviation of 1.33 weeks. This was not significant.

The mean birth weight among the MA group was 2.61kg and among the non-ma group was 2.69. The association between the MA group and non-MA group was significant. But this was not of clinical importance.

Among the study group 35 patients had history of prom out of which 25(65.71) cases belonged to the MA group and 12(34.29%) belonged to the non-MA group, there is a statistically significant relation of history of PROM and MA. The same significance was obtained from s similar study done at KLE hospital

The duration of mean duration of PROM was obtained from history in the MA group was 18.74hr with a standard deviation of 3.63 hr. The majority of the cases of MA 9 (39.3%) were in the group of 14-17 hours.

The average number of calcifications and average HU of the calcifications was more when the duration of PROM was in the range of 6 to 24 hours. Increased duration of PROM predispose to increased chances of perinatal infections, hence the average HU of the calcifications was more when the increased duration of PROM.

Among the study group 36 (20%) patients had history of NICU admission out of which 19(52.78%) cases belonged to the MA group and 17(47.22%) belonged to the non-MA group. this gives us a statistically significant correlation of history of NICU.

In our present study we have found the history PROM and NICU admission and duration of labour had a statistically significant relationship with MA, which states that there is a possible association between perinatal infections and mineralizing angiopathy. Any site of infection in the brain during the intranatal and peripartum period can predispose to MA.

**Strategies to prevent MA:**

Maintaining good hygiene during the peripartum period Minimizing infections during delivery by advising moms to drink more water to increase frequency of micturition decreases the possibility of infections.

Patients who come with PROM are predisposed to urinary tract infections and chorioamnionitis, emergency C section in patients who come with PROM can help to prevent MA.

Avoiding recurrent pervaginal examinations can minimize the spread of ascending infections during peripartum period.

**Protective factors**

Counselling to avoid trauma/ fall to the parents of children with MA

Identify the high risk age group – children of 1 to 5 years and target them

**Limitations of the study**

- The fact that we relied on parental reporting of the history of PROM/NICU admissions and the duration of labour rather than on documented data is one of the study's shortcomings.

- The study is based on CT in cases coming to the hospital, MRI being a safer option and a better option for various disease pickup but not sensitive for MA

**Strengths of the study**

- The present study was a prospective study.

- Detailed history was taken from the parents directly.

- All the scans were done with the 128 slice CT machine, calcifications are detected with higher accuracy in a 128 slice CT as compared to 64 slice CT.

- Use of 3d reconstruction with coronal images for better visualisation of linear streak of calcifications.

# CONCLUSION

- This study aimed to evaluate the presence of mineralizing angiopathy and its correlation with PROM/NICU admission (perinatal infections) in department of radio-diagnosis of tertiary care hospital.
- The diagnostic utility of imaging modalities like MRI is limited in children with MA, but CT has higher utility in picking up foci of calcification and demonstration of linear calcifications in coronal reconstruction
- The prevalence of mineralizing angiopathy in our study was 29.67%.
- Majority of the cases of MA belonged to the age group of one to 5 years
- Mineralizing angiopathy has got association with perinatal infections (history of PROM and NICU admissions) during delivery.
- Minimal number of the cases of MA present with stroke when they have a fall in the vulnerable period of 1 to 5 years.

# SUMMARY


- A prospective study was conducted in the department of Radio-diagnosis presence of mineralizing angiopathy and its correlation with PROM/NICU admission
- This study highlighted the role of CT in guiding the clinician to diagnose MA and its possible association with perinatal infections
- In our present study we have found the history PROM and NICU admission and duration of labour had a statistically significant relationship with mineralizing angiopathy
- Mineralizing angiopathy has got association with perinatal infections (history of PROM and NICU admissions) during delivery
- Strategies to prevent MA include maintaining good hygiene during the peripartum period, minimizing infections during delivery by frequent micturation.
- Only few of the cases of MA will present with stroke after trauma
- Counselling of parents to prevent fall in children with MA in the high risk age group could prevent the incidence of stroke in children with MA

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# ANNEXURE I – CONSENT FORM

## KAHER

## J. N. MEDICAL COLLEGE, BELAGAVI.

## DEPARTMENT OF RADIODIAGNOSIS

## INFORMED CONSENT

**"PREVALENCE OF MINERALISING ANGIOPATHY IN YOUNG CHILDREN: AN OBSERVATIONAL STUDY".**

**PRINCIPAL INVESTIGATOR:** **Dr.**
Post Graduate student
Department of Radiodiagnosis

**CO-INVESTIGATOR:** **DR.**
Professor, Department of Radiodiagnosis

**INTRODUCTION AND PURPOSE:** The purpose of this study is to determine the prevalence of mineralizing angiopathy on computed tomography in children aged 1 month to 12 years of age and arbitrate on an etiology based on history. Multidetector Computed tomography provides accurate parameters and information in detection of mineralizing angiopathy. This study aids in improving the quality of treatment towards patients suffering from it.

**PROCEDURE:** I request you to kindly allow the participation of your child/ ward in the study titled study titled study **"MINERALISING ANGIOPATHY IN YOUNG CHILDREN: AN OBSERVATIONAL STUDY".** at Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi is being conducted by

**Dr. _____,** Post graduate in Radio diagnosis at J.N. Medical College, Belagavi, Karnataka, under the guidance of **Dr. _____,** Professor, Dept. of Radio-diagnosis, J. N. Medical College, Belagavi.

We request you to kindly allow the participation of your child/ wardin this study as you are eligible to be included. During the study, you will be asked questions regarding your present and past medical history, your childs medical history and you will be required to answer to the best of your knowledge. You will also be clinically examined as per the protocol drawn.

If you agree to participate in the study, please furnish the details pertaining to the study.

**BENEFITS:** No use of surgical equipment/risk associated with it.

**COMPLICATIONS:** Minimal exposure to radiation. A single exposure to radiation during CT scan usually does not cause any adverse effects. Some rare events that can occur are skin reddening, induction of cancer.

**ALTERNATIVES:** If the parents are not willing for their child to take part in the study, the child's treatment or any other further investigations that he/she wants to undergo, in future, in KLE will not be affected by their decision.

**VOLUNTARY PARTICIPATION/WITHDRAWAL:** Taking part in this study is voluntary. I may choose for my child not to take part in this study, or if I decide for him/her to take part I can later change my mind and withdraw my child from the study. My decision will not change the present or future health care or other services that my child receives. The study doctor or the sponsor may stop my child's participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. If I choose for my child to not to take part in the study I will receive the standard treatment for patients with my child's condition.

**COSTS:** Nil (The study is to be conducted on the participants who are advised COMPUTED TOMOGRAPHY as an investigation by the referring consultant and the participants will bear the charges for it.)

**Payment for Participation:** No incentive will be paid to you for participating in this study.

**COMPENSATION:** In the event that my child becomes injured as a result of taking part in this study, treatment whatever available at KLE charitable hospital, Belagavi, will be offered to him/her. No reimbursement, compensation or free medical care is given.

**CONFIDENTIALITY:** All information collected about my child during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify him/her in this research record. Information from this study may be published but his/her identity will be confidential in any publication.

**QUESTION:**

If you have any enquiries in the future or in case of research related injury illness, you may contact following persons:

Name: Dr. Kondabattula Rakesh

Mobile No: 9482723971

Email ID: rakk1996@gmail.com

| | | |
|---|---|---|
| | | **DR. HARSHA HEGDE** |
| | | CHAIRPERSON JNMC IEC<br>&<br>SCIENTIST D, ICMR, NATIONAL INSITUTE OF TRADITIONAL MEDICINE, BELAGAVI |
| | | Ph. No: 9480422500 |

**KAHER**

**J. N. MEDICAL COLLEGE, BELAGAVI.**

**DEPARTMENT OF RADIODIAGNOSIS**

**INFORMED SIGNED CONSENT:**

1. I understand that my child will be participating in the study, which includes computed tomography imaging.

2. I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of my child taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent my child to participate in the trial outlined above.

3. I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw my child from the study at any point of time.

4. I consent to the photographing or recording of the procedure to be performed including appropriate portions of my child's body, for medical, scientific or educational purposes provided his/her identity is not revealed in the pictures or by the descriptive texts accompanying them.

5. I understand that there is no significant risk involved in the test that would be done in this study.

6. No guarantee or assurance has given by anyone as to the results that may be obtained.

7. My signature on this form signifies that I have willingly decided for my child's participation after understanding the above information.

Participant's Name/ legally authorized_____representative

Signature _____

Name and signature of witness _____

Name and signature of interviewer _____

Date: _____

Place_____

**KAHER**

**J. N. MEDICAL COLLEGE, BELAGAVI.**

**DEPARTMENT OF RADIODIAGNOSIS**

**VERBAL ASSENT FROM MINOR (7-12 years) ALONG WITH PARENTAL CONSENT**

I / my parent or legal guardian has read the previous page(s) of the consent form and the investigator has explained the details of the study. I/my parent or legal guardian understands that I am free to ask additional questions.

I/my parent or guardian understands that participation in this study entitled [Mineralising Angiopathy In Young Children: An Observational Study] is voluntary and I/my parent or legal guardian may refuse to participate or may discontinue participation at any time without penalty, loss of benefits, or prejudice to the quality of care which I will receive.

I/my parent or legal guardian, acknowledge that no guarantees have been made to meregarding the results of the treatment involved in this study, and I agree to participate in the study and have been given a copy of this form.

| | |
|---|---|
| Name of the Parent | Signature of the parent |
| Name of Investigator | Signature of investigator |
| Name of Witness | Signature of Witness |

# ANNEXURE II -PROFORMA

# DATA COLLECTION INSTRUMENT

## PROFORMA

**"MINERALISING ANGIOPATHY IN YOUNG CHILDREN: AN OBSERVATIONAL STUDY"**

**DATE OF INTERVIEW:**

**NAME OF THE PATIENT:**

| | | | |
|---|---|---|---|
| **SL.NO.** | | **DATE** | |
| **PATIENT NAME** | | **CT SCAN NO.** | |
| **AGE** | | **SEX** | |

**CHIEF COMPLAINTS:** **DURATION**

| | | |
|---|---|---|
| **1.** | | |
| **2.** | | |

**HISTORY OF PRESENTING ILLNESS:**

| | | |
|---|---|---|
| **1.** | | |
| **2.** | | |
| **3.** | | |

**HISTORY OF PROM / NICU ADMISSION**

| | | |
|---|---|---|
| **1.** | **DURATION OF LABOR** | |
| **2.** | **DURATION OF PROM** | |
| **3.** | **CESAREAN DELIVERY:**<br><br>**LOW BIRTH WEIGHT:** | |

**CT FINDINGS:**

| | | |
|---|---|---|
| **1.** | **NUMBER OF CALCIFICATIONS** | |
| **2.** | **LOCATION AND SYMMETRY OF CALCIFICATIONS** | |
| **3.** | **HU OF CALCIFICATIONS** | |
| **4.** | **CORONAL RECONSTRUCTION** | |

# ANNEXURE-III – PHOTOGRAPHS

## PHOTOGRAPH OF GE EVOLUTION 128 SLICE CT MACHINE AT KLES PRABHAKAR KORE HOSPITAL

# CASES

## CASE 1

**9 year old child who underwent CT scan for minor trauma, incidentally found calcification in the right lentiform nucleus, on detailed prenatal interview the mother had history of PROM of 22 hrs. The child was neurologically normal.**

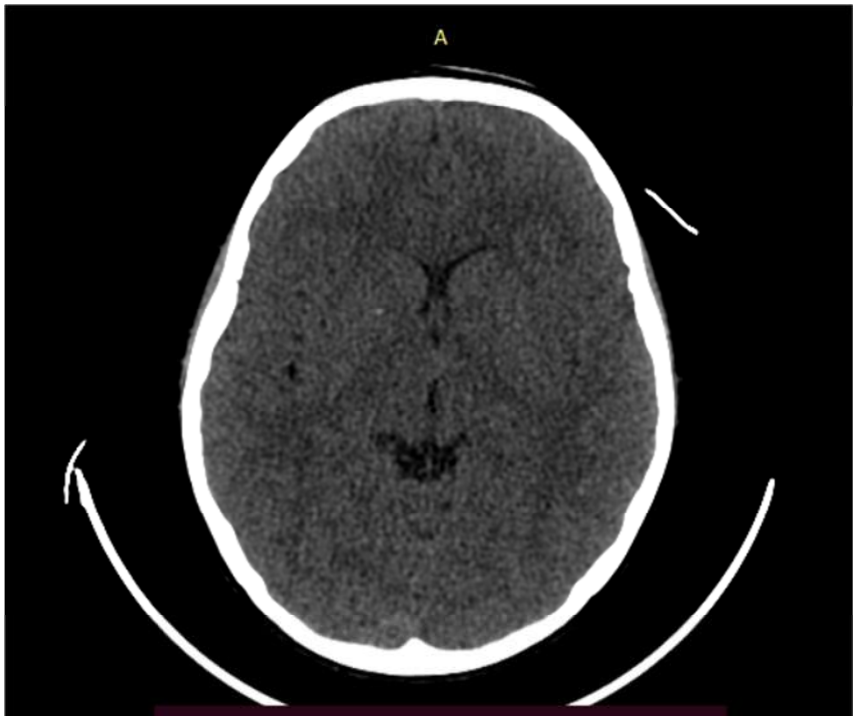



**Fig 1.1: Axial CT image showing calcification in the right lentiform nucleus in a 9 year old male child with mineralizing angiopathy**

**CASE 2:**

**A 12 year old male child with mineralizing angiopathy who had history of fall followed by basal ganglia infarction with cerebral edema, the child had underwent decompressed craniotomy for the same, on follow up scans showed liner foci of calcification along the lentiform nucleus on left side.**

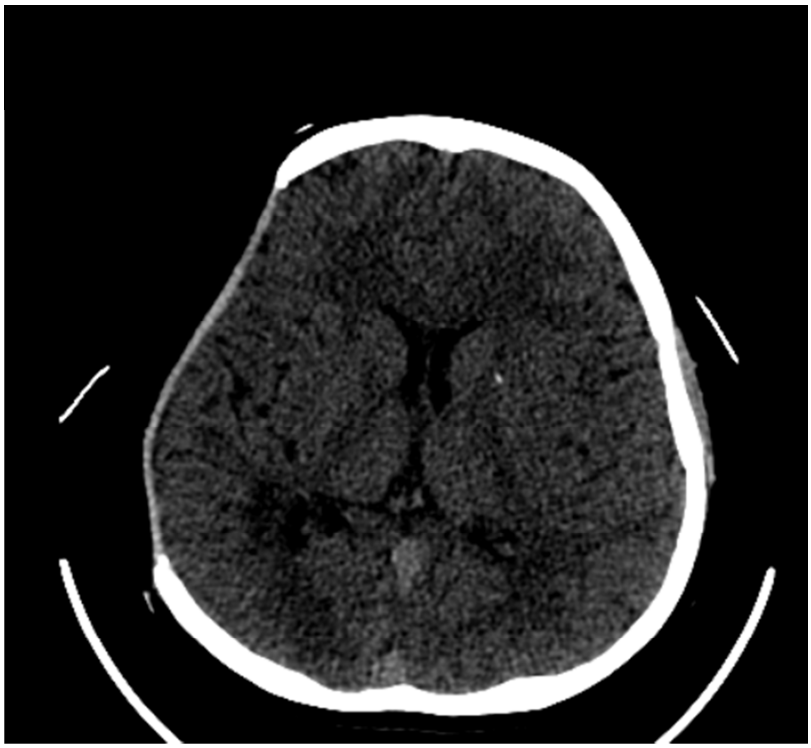


**Figure 2.1 Axial CT image of a 12 year old male child born via vaginal delivery who had history of PROM of 24 hours during peripartum period with mineralizing angiopathy showing linear streak of calcifications along the left lentiform nucleus, the child underwent decompressive criniectomy post basal ganglia infarction post trauma.**

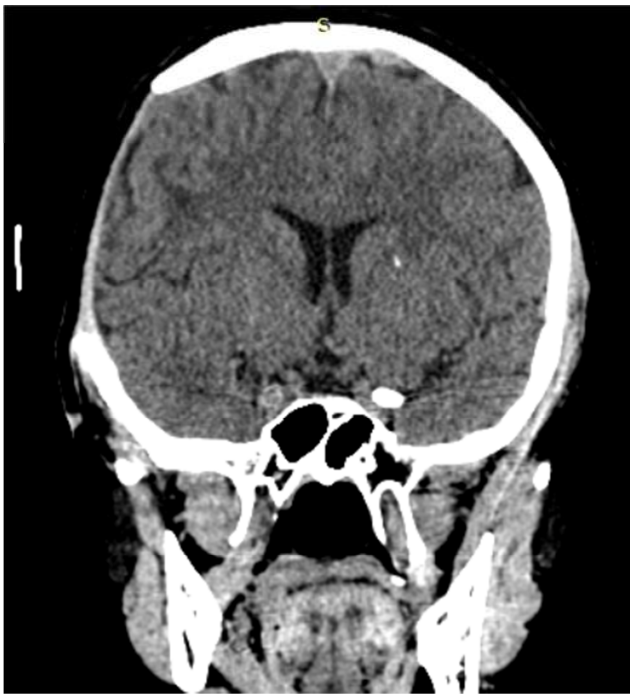
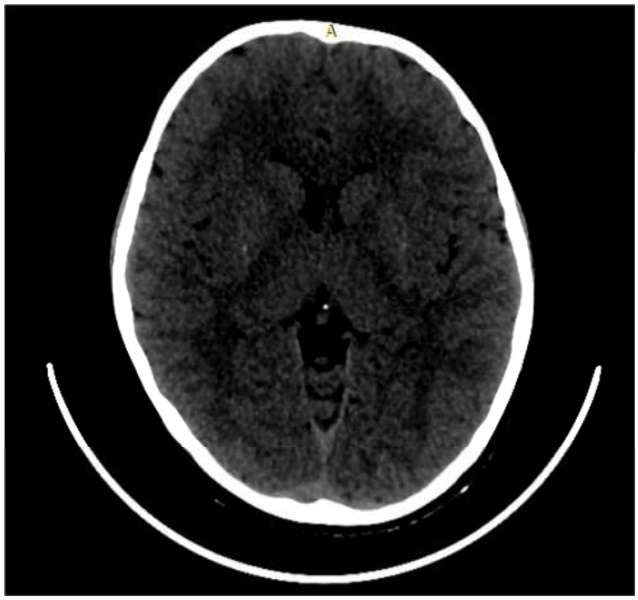


**Figure 2.2 Coronal CT image of a 12 year old male child born via vaginal delivery who had history of PROM of 24 hours during peripartum period with mineralizing angiopathy showing linear streak of calcifications along the left lentiform nucleus, the child underwent decompressive criniectomy post basal ganglia infarction post trauma.**

**CASE 3:**
**Figure 3.1**



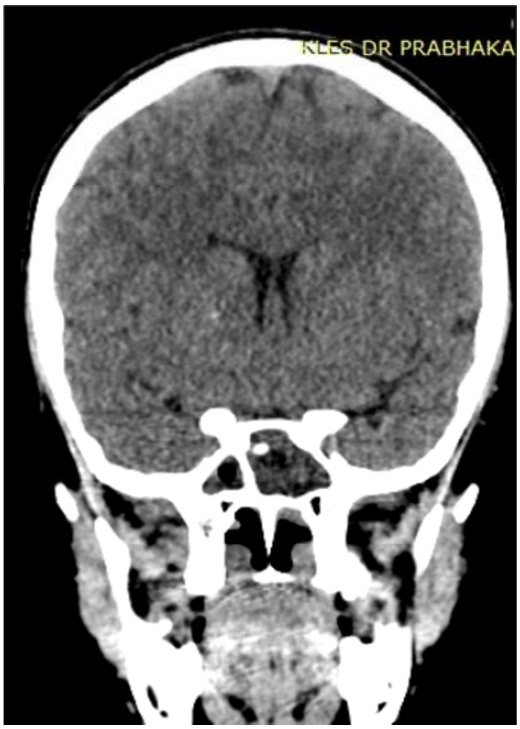




**Figure 3.2**

**Figure 3.1 and 3.2: Visualization of the linear foci of calcification on coronal reconstruction in a 1 year old girl child born via normal delivery who had delayed milestones who had history of NICU admission post-delivery.**

**CASE 4:**

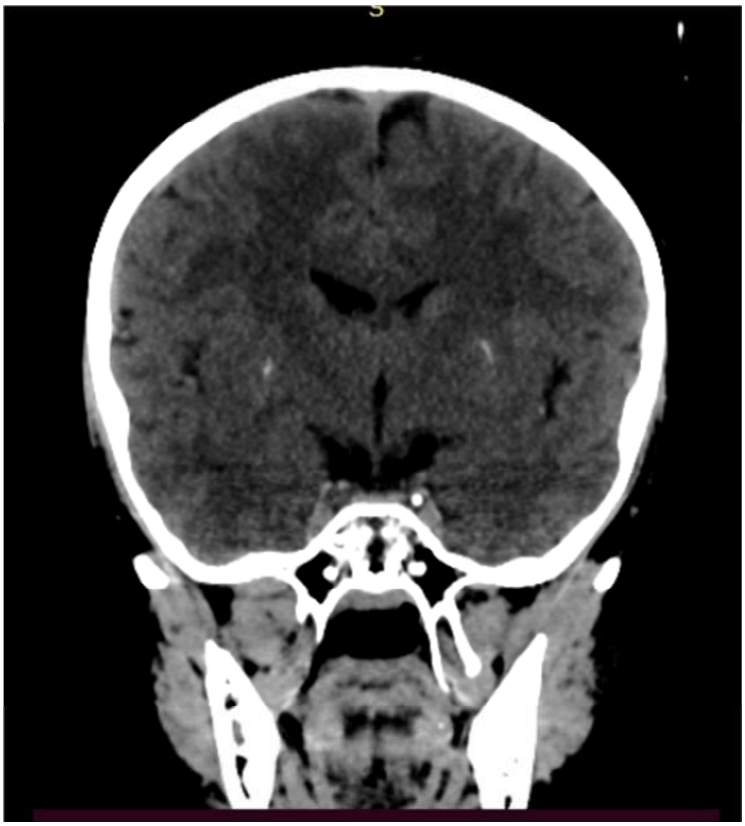


**Figure 4.1 : Visualization of the linear foci of calcification on coronal reconstruction in a 12 year old boy with MA.**

**The child came with an indication of minor trauma, the child was neurologically normal.**

**CASE 5:**

**2 year old girl who had history of fall followed by weakness of the left lower limb**

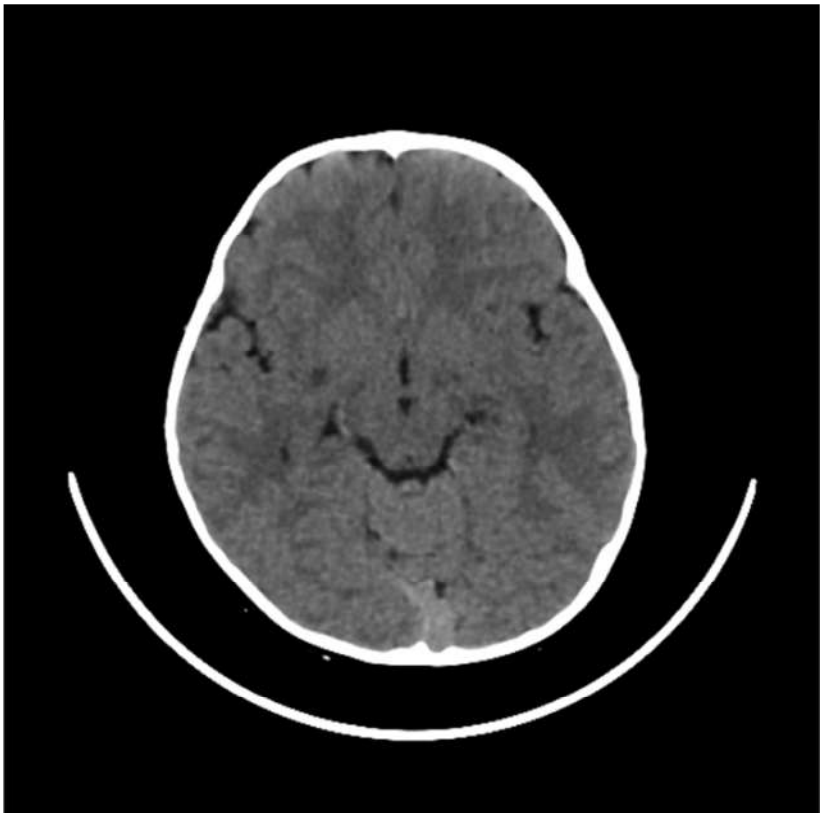


**Figure 5.1: Axial CT image showing a subacute infarct in the posterior aspect of the lentiform nucleus with small foci of hyperdensities within it likely to be calcific foci, in a 2 year old girl who had history of fall followed by weakness of the left lower limb. The child had history of PROM admission with prolonged PROM of 19 hours.**

# ANNEXURE IV - KEY TO MASTER CHART

| | | |
|---|---|---|
| S | - | Spontaneous labor |
| I | - | Induced labor |
| CN | - | Caudate nucleus |
| NVD | - | Normal vaginal delivery |
| CS | - | C section |
| Low birth weight | - | L low birth weight |
| N | - | Normal Weight |
| GDD | - | Global Developmental Delay |
| T | - | Term |
| Early preterm | - | EP |
| DMS | - | Delayed Mile Stones |

| Scan number | Date | Age | Sex | Mineralizing angiopathy/ calcifications | Number of calcifacations | Average HU | symmetrical or assemtrical calcifiations | location of intracranial calcifations | calcifications on coronal reccontructions | extracranial calcifications | infarct | Spontaneous or induced labour | Duration of labour (Hr) | Mode of delivery | Gestational age | Birth weight | H/O PROM | Duration of PROM (hr) | H/O NICU ADMISSIONS | other significant history/ PIH | indication for the scan | MRI if done | MK |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 23389 | 14-Oct-20 | 3Y | F | PRESENT | 5 | 53 | lt basal ganglia | lentiform nucleus | Y | N | N | S | 16 | NVD | 39 | 2.4 | NIL | | NIL | - | Delayed mile stone | - | |
| 24716 | 11-Nov-20 | 2Y | F | ABSENT | - | - | - | - | - | - | - | S | 14 | NVD | 37 | N | YES | 16 | YES | - | trauma | | |
| 29021 | 25-Jan-21 | 4Y | M | PRESENT | 13 | 65 | Bilateral basal ganglia | lentiform nucleas | Y | N | Y- lt CN, Lt IC and Lt LN | S | 16 | NVD | 38 | 2.5 | NIL | | YES | - | trauma | yes | |
| 29564 | | 1Y | F | ABSENT | - | - | - | - | - | - | - | I | 17 | NVD | 34 | N | YES | 18 | YES | - | | | |
| 20295 | 23-Dec-20 | 2Y | F | PRESENT | 6 | 66 | Bilateral basal ganglia | bilateral thalami | N | Y-lt fr & rt Pr | - | I | | CS | 39 | 2.5 | YES | 16 | YES | | DMS | | |
| 28765 | 20-Jan-21 | 2Y | M | ABSENT | - | - | - | - | - | - | - | S | 17 | NVD | 34 | N | NIL | | NIL | - | | | |
| 29489 | 03-Feb-21 | 6Y | M | ABSENT | - | - | - | - | - | - | - | S | | CS | 35 | N | NIL | | NIL | - | | | |
| 24704 | 11-Nov-20 | 8M | F | ABSENT | - | - | - | - | - | - | - | I | 18 | NVD | 36 | n | NIL | | YES | - | - | | |
| 25225 | 27-Jan-21 | 11Y | M | ABSENT | - | - | - | - | - | - | - | S | 20 | NVD | 36 | N | NIL | | NIL | - | - | | |
| 23846 | 24-Feb-21 | 10Y | M | PRESENT | 4 | 61 | Rt basal ganglia | lentiform nucleus and caudate | N | - | Y- mid brain and pons | S | 18 | NVD | 38 | 2.6 | NIL | | NIL | | trauma | yes | |
| 31103 | 08-Mar-21 | 10M | F | PRESENT | 11 | 68 | Lt basal ganglia | lentiform nucleas | Y | - | - | I | 12 | NVD | 35 | 2.7 | YES | 14 | NIL | | DMS | | MK |
| 30897 | 03-Mar-21 | 12Y | M | ABSENT | | | - | | | | | S | 17 | NVD | 37 | N | - | | yes | | - | | |
| 31910 | 25-Mar-21 | 11Y | M | ABSENT | | | - | | | | | S | 17 | NVD | 38 | N | NIL | | NIL | - | | | |
| 32266 | 04-02-2021 | 3Y | F | ABSENT | | | - | | | | | I | | CS | 35 | N | NIL | | NIL | - | | | |
| 34009 | 05-02-2021 | 2Y | M | ABSENT | | | - | | | | | S | 18 | NVD | 36 | n | NIL | | YES | - | - | | |
| 34736 | 05-11-2021 | 10Y | F | ABSENT | | | - | | | | | S | 20 | NVD | 37 | N | NIL | | NIL | - | - | | |
| 35377 | 18/05/2021 | 11Y | M | ABSENT | | | - | | | | | S | 17 | NVD | 37 | N | - | | - | | - | | |
| 35877 | 23/05/21 | 4Y | M | PRESENT | 6 | 55 | Rt basal ganglia | lentiform nucleas | N | 0 | 0 | S | 22 | NVD | 36 | 2.4 | no | | no | | seizure | | |
| 36721 | 28/05/21 | 8Y | M | PRESENT | 13 | 60 | Lt basal ganglia | lentiform nulceas | N | 0 | 0 | I | 22 | NVD | 38 | 2.9 | no | | yes | | trauma | | |
| 36317 | 28/05/21 | 10y | F | ABSENT | | | - | | | | | S | 17 | NVD | 38 | N | - | | - | | - | | |
| 35806 | 22/05/21 | 10y | F | ABSENT | | | - | | | | | S | 18 | NVD | 38 | n | NIL | | YES | - | - | | |
| 35437 | 18/05/21 | 2y | F | ABSENT | | | - | | | | | I | 20 | NVD | 38 | N | NIL | | NIL | - | - | | |
| 35913 | 24/05/21 | 6Y | F | ABSENT | | | - | | | | | S | 17 | NVD | 36 | N | yes | 16 | - | | - | | |
| 37717 | 25/06/2021 | 8Y | F | PRESENT | 14 | 55 | Rt basal ganglia | lentiform nucleas | Y | | | | 20 | NVD | 37 | 2.7 | no | | Yes | | trauma | | |
| 37746 | 25/06/2021 | 4Y | F | ABSENT | | | - | | | | | S | 17 | NVD | 37 | N | NIL | | NIL | - | | | |
| 37794 | 26-26-2021 | 1y | M | ABSENT | | | - | | | | | S | | CS | 36 | N | NIL | | NIL | - | | | |
| 37865 | 28-06-2021 | 11y | M | ABSENT | | | - | | | | | I | 17 | NVD | 35 | N | NIL | | NIL | - | | | |
| 37897 | 29-06-2021 | 10y | M | ABSENT | | | - | | | | | S | | CS | 36 | N | yes | | NIL | - | | | |
| 38181 | 07-05-2021 | 3Y | F | ABSENT | | | - | | | | | S | | CS | 36 | N | NIL | | NIL | - | | | |
| 38862 | 15/07/2021 | 2Y | M | PRESENT | 10 | 68 | Lt basal ganglia | lentiform nucleas | Y | | | I | 18 | NVD | 37 | 2.2 | no | | YES | | Delayed mile stone | | MK |
| 38660 | 15/07/2121 | 9Y | M | ABSENT | | | - | | | | | S | 17 | NVD | 38 | N | - | | - | | - | | |
| 38669 | 15/07/2021 | 5m | F | ABSENT | | | - | | | | | S | 17 | NVD | 38 | N | NIL | | NIL | - | | | MK |

| | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 38739 | 17-07-2021 | 6Y | M | ABSENT | | | - | | | | | I | | CS | 38 | N | NIL | | NIL | - | | | |
| 38789 | 18-07-2021 | 9y | M | ABSENT | | | - | | | | | I | 17 | NVD | 36 | N | NIL | | NIL | - | | | |
| 38818 | 19-07-2021 | 15m | F | PRESENT | 8 | 58 | Lt basal ganglia | lentiform nucleas | N | | | S | | CS | 35 | 2.7 | YES | 16 | no | PIH | trauma | | |
| 38964 | 21-07-2021 | 2y | F | PRESENT | 9 | 62 | Rt basal ganglia | lentiform nucleas | N | | | S | 14 | NVD | 36 | 2.8 | yes | 18 | no | | trauma | | |
| 40523 | 24-08-2021 | 2y | F | ABSENT | | | - | | | | | | | | 38 | | yes | 16 | - | | - | | |
| 40667 | 27-08-2021 | 12y | M | PRESENT | 10 | 65 | Rt basal ganglia | lentiform nucleas | N | | | S | 17 | NVD | 36 | 2.6 | NIL | | n | | DMS | | MK |
| 40740 | 29-08-2021 | 12y | M | ABSENT | | | - | | | | | I | 17 | NVD | 36 | N | NIL | | NIL | - | | | |
| 40739 | 30-08-2021 | 12y | M | ABSENT | | | - | | | | | I | 17 | NVD | 37 | N | NIL | | NIL | - | | | |
| 40789 | 30-08-2021 | 3y | M | PRESENT | 4 | 62 | Rt basal ganglia | lentiform nucleas | N | | | I | 16 | NVD | 36 | 2.7 | yes | 18 | no | | - | | MK |
| 40831 | 31-08-2021 | 12y | F | PRESENT | 5 | 63 | Lt basal ganglia | lentiform nucleas | | | | S | 17 | NVD | 37 | 2.8 | no | | no | | - | | |
| 40988 | 03-09-202 | 4y | M | ABSENT | | | - | | | | | I | 17 | NVD | 36 | N | NIL | | NIL | - | | | |
| 41145 | 09-07-2021 | 7y | M | ABSENT | | | - | | | | | S | | CS | 35 | N | NIL | | NIL | - | | | |
| 41266 | 09-09-2021 | 2y | M | ABSENT | | | - | | | | | S | | CS | 35 | N | NIL | | NIL | - | | | |
| 41692 | 19-09-2021 | 12y | F | PRESENT | 12 | 69 | Lt basal ganglia | lentiform nucleas | Y | | | S | 17 | NVD | 37 | 2.4 | no | | no | | irritability | | MK |
| 41635 | 17-09-2021 | 3M | M | ABSENT | | | - | | | | | S | | CS | 34 | N | yes | 18 | NIL | - | | | |
| 41980 | 24-09-2021 | 7Y | M | PRESENT | 14 | 62 | Rt basal ganglia | lentiform nucleas | Y | | | S | 16 | NVD | 38 | 2.9 | no | | no | | - | | |
| 41952 | 24-09-2021 | 12Y | M | ABSENT | | | - | | | | | S | | CS | 34 | N | NIL | | NIL | - | | | |
| 41995 | 25-09-2021 | 1yr | M | PRESENT | 14 | 58 | Lt basal ganglia | lentiform nucleas | Y | | | S | 20 | NVD | 38 | 2.7 | no | | no | | trauma | | |
| 41737 | 26-09-2021 | 1y | M | PRESENT | 10 | 55 | Rt basal ganglia | lentiform nucleas | N | | | S | | CS | 37 | 2.4 | yes | 22 | no | PIH | trauma | | |
| 42226 | 30-09-2021 | 7yr | F | ABSENT | | | - | | | | | S | 20 | NVD | 38 | 2.6 | NIL | | NIL | - | - | | |
| 42581 | 10-08-2021 | 12Y | F | ABSENT | | | - | | | | | S | 17 | NVD | 36 | N | - | | - | | - | | |
| 42668 | 10-10-2021 | 3y | M | PRESENT | 5 | 54 | Bilateral basal ganglia | lentiform nucleas | N | | | S | 20 | NVD | 39 | 2.6 | yes | 19 | no | | - | | * |
| 42746 | 10-12-2021 | 10y | M | ABSENT | | | - | | | | | S | 17 | NVD | 37 | N | - | | - | | - | | |
| 42845 | 14-10-2021 | 5y | M | ABSENT | | | - | | | | | S | 17 | NVD | 38 | N | NIL | | NIL | - | trauma | | |
| 42908 | 15-10-2021 | 12y | M | ABSENT | | | - | | | | | I | | CS | 35 | N | NIL | | NIL | - | trauma | | |
| 23024 | 19-10-2021 | 2y | M | ABSENT | | | - | | | | | S | 17 | NVD | 36 | N | - | | - | | - | | |
| 43244 | 23-10-2021 | 5y | F | PRESENT | 6 | 60 | Lt basal ganglia | lentiform nucleas | N | | | S | 18 | NVD | 36 | 2.7 | no | | no | | - | | * |
| 43917 | 11-09-2021 | 4y | F | ABSENT | | | - | | | | | S | 18 | NVD | 39 | n | NIL | | YES | - | - | | |
| 43624 | 11-02-2021 | 2m | M | ABSENT | | | - | | | | | I | 20 | NVD | 37 | N | yes | 18 | NIL | - | - | | |
| 43521 | 30-10-2021 | 2 yr | M | ABSENT | | | - | | | | | S | 18 | NVD | 36 | n | NIL | | YES | - | - | | * |
| 44327 | 27-11-2021 | 6m | M | ABSENT | | | - | | | | | I | 20 | NVD | 38 | N | yes | 17 | NIL | - | - | | |
| 44583 | 25-11-2021 | 1y | F | PRESENT | 5 | 62 | Rt basal ganglia | lentiform nucleas | N | | | S | 20 | NVD | 37 | 2.4 | Yes | 22 | no | | - | | * |
| 44748 | 28-11-2021 | 2y | F | PRESENT | 4 | 57 | lt basal ganglia | lentiform nucleas | N | | | S | 20 | NVD | 39 | 2.3 | yes | 24 | no | | - | | * |
| 45186 | 09-12-2021 | 3y | F | PRESENT | 3 | 55 | lt basal ganglia | lentiform nucleas | N | | | S | | CS | 40 | 2.5 | yes | 24 | Yes | | - | | |
| 42161 | 09-12-2021 | 10y | M | ABSENT | | | - | | | | | S | | CS | 35 | N | NIL | | NIL | - | | | |
| 45423 | 15-12-2021 | 9y | M | Present | 10 | 52 | Rt basal ganglia | lentiform nucleas | N | | | S | 19 | NVD | 37 | 2.5 | no | | no | | - | | |
| 45481 | 16-12-2021 | 12yr | M | PRESENT | 16 | 62 | Bilateral basal ganglia | lentiform nucleas | Y | | Yee- rt BG | S | 18 | NVD | 39 | 2.3 | no | | no | | trauma | yes | - |
| 45517 | 17-12-2021 | 9y | M | PRESENT | 12 | 68 | Bilateral basal ganglia | lentiform nucleas | N | | | S | 18 | NVD | 40 | 2.9 | no | | no | | trauma | | |
| 45548 | 17-12-2021 | 6m | M | ABSENT | | | - | | | | | S | 18 | NVD | 37 | n | NIL | | YES | - | - | | * |
| 45905 | 26-12-2021 | 5y | M | ABSENT | | | - | | | | | I | 20 | NVD | 37 | N | NIL | | NIL | - | - | | |
| | | | | | | | | | | | | | | | | | | | | | | | |
| 41452 | 01-09-2022 | 6y | M | PRESENT | 9 | 57 | lt basal ganlia | lentiform nucleas | N | | | S | 18 | NVD | 35 | 2.7 | NIL | | yes | - | trauma | | |
| 46627 | 01-10-2022 | 9y | M | ABSENT | | | | | | | | S | | CS | 39 | N | NIL | | YES | | hydrocephalus | | |
| 47488 | 20-01-2022 | 3y | M | ABSENT | | | | | | | | S | 17 | NVD | 37 | N | NIL | | n | | hydrocephalus | | |
| 42602 | 02-06-2022 | 11y | M | ABSENT | | | | | | | | S | 17 | NVD | 36 | N | NIL | | N | | trauma | | |
| 42890 | 02-07-2022 | 11y | F | ABSENT | | | | | | | | S | 20 | NVD | 38 | n | NIL | | N | | trauma | | |
| 43196 | 13-2-2022 | 5y | M | ABSENT | | | | | | | | I | 18 | NVD | 39 | n | NIL | | n | n | trauma | | |
| 48158 | 17-02-2022 | 6y | F | ABSENT | | | | | | | | S | | CS | 37 | n | NIL | | n | | hydrocephalus | | |
| 48199 | 1/6/12022 | 6Y | F | ABSENT | | | | | | | | S | 16 | NVD | 36 | n | NIL | | n | | trauma | | |
| 48366 | 22-02-2022 | 8 | M | ABSENT | | | | | | | | S | 18 | NVD | 38 | n | NIL | | n | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 48472 | 25-02-2022 | 3y | M | ABSENT | | | | | | | | S | 16 | NVD | 39 | n | NIL | | n | | trauma | | |
| 48572 | 28-02-2022 | 5y | F | ABSENT | | | | | | | | I | 19 | NVD | 37 | n | NIL | | n | | irritability | | |
| 48587 | 28-02-2022 | 5y | F | ABSENT | | | | | | | | S | | CS | 36 | N | NIL | | n | | Cleft palate | | |
| 48640 | 03-01-2022 | 4y | M | ABSENT | | | | | | | | S | 22 | NVD | 38 | n | NIL | | n | | trauma | | |
| 48741 | 03-02-2022 | 3y | M | ABSENT | | | | | | | | S | 20 | nvd | 39 | n | NIL | | n | | meningits? | | |
| 48800 | 03-05-2022 | 2y | M | ABSENT | | | | | | | | S | 18 | NVD | 37 | n | NIL | | n | | case of meningits | | |
| 49084 | 20-05-2034 | 2y | M | ABSENT | | | | | | | | I | 20 | NVD | 36 | n | yes | 19 | n | | | | |
| 40846 | 15-03-2022 | 12y | M | ABSENT | | | | | | | | S | 18 | NVD | 38 | n | NIL | | n | | | | |
| 49302 | 17-03-2022 | 2y | M | ABSENT | | | | | | | | S | 17 | NVD | 38 | n | NIL | | n | | trauma | | |
| 49323 | 17-03-2022 | 4y | M | ABSENT | | | | | | | | S | 18 | NVD | 39 | n | NIL | | n | | trauma | | |
| 49522 | 23-03-2022 | 9y | F | ABSENT | | | | | | | | S | 20 | NVD | 37 | n | NIL | | yes | | trauma | | |
| 49834 | 21-03-2021 | 1y | F | PRESENT | 5 | 67 | lt basal ganlia | lentiform nucleas | N | | | I | 17 | NVD | 38 | 2.7 | - | | - | | - | | |
| 49683 | 27-03-2022 | 10y | F | ABSENT | | | | | | | | S | 18 | NVD | 39 | N | NIL | | N | | hydrocephalus | | |
| 49384 | 30-03-2022 | 5y | m | ABSENT | | | | | | | | S | | CS | 37 | n | NIL | | n | | hydrocephalus | | |
| 50458 | 04-08-2022 | 6y | F | PRESENT | 5 | 60 | lt basal ganglia | lentiform nucleas | N | N | N | S | 19 | NVD | 37 | 2.7 | NIL | | yes | | DMS | | mk |
| 50277 | 04-10-2022 | 6y | F | ABSENT | | | | | | | | I | 20 | NVD | 36 | n | NIL | | n | | trauma | | |
| 50394 | 13-04-2022 | 8y | F | ABSENT | | | | | | | | S | | CS | 38 | n | NIL | | n | | | | |
| 50471 | 04-05-2022 | 8y | F | ABSENT | | | | | | | | S | 18 | NVD | 39 | n | NIL | | n | n | | | |
| 11580737 | 24-04-2022 | 7y | F | ABSENT | | | | | | | | S | 18 | NVD | 37 | n | NIL | | YES | - | - | | * |
| 11577747 | 27-04-2022 | 7y | M | PRESENT | 7 | 58 | rt basal ganglia | lentiform nucleas | N | N | N | I | 16 | NVD | 39 | 2.6 | no | | n | | trauma | | |
| 11595124 | 27-04-2022 | 1Y | M | PRESENT | 10 | 60 | lt basal ganglia | lentiform nucleas q | N | N | N | I | 22 | NVD | 36 | 2.8 | yes | 17 | Yes | | trauma | | |
| 11580737 | 29-04-2022 | 6Y | F | ABSENT | | | | | | | | I | 18 | NVD | 36 | N | yes | 14 | n | | trauma | | |
| 11603908 | 30-04-2022 | 3y | F | ABSENT | | | | | | | | S | 18 | NVD | 35 | LBW | no | | yes | | trauma | | |
| 11606185 | 05-01-2022 | 3y | M | ABSENT | | | | | | | | S | 18 | NVD | 39 | n | n | | n | | | | |
| 175196 | 05-03-2022 | 9y | M | ABSENT | | | | | | | | S | | CS | 37 | n | n | | n | | trauma | | |
| 11622566 | 05-03-2022 | 4y | M | PRESENT | 9 | 60 | Bilateral basal ganglia | lentiform nucleas | N | N | N | I | 18 | NVD | 39 | 2.7 | yes | 14 | Yes | | DMS | | |
| 175353 | 05-04-2022 | 5y | F | ABSENT | | | | | | | | I | 18 | NVD | 36 | N | n | | n | | trauma | | |
| 11640519 | 05-05-2022 | 5Y | F | ABSENT | | | | | | | | S | | CS | 38 | n | NIL | | n | | Seizures | | |
| 11652227 | 05-06-2022 | 9Y | F | PRESENT | 13 | 62 | Lt basal ganglia | lentform nucleas | N | N | N | S | 20 | NVD | 39 | 2.7 | n | | Yes | | | | |
| 11663976 | 05-09-2022 | 1Y | F | PRESENT | 16 | 65 | Bilateral basal ganglia | Lentiform nucelas | Y | n | N | S | 22 | NVD | 38 | 2.6 | yes | 16 | yes | | trauma | | |
| 1111027 | 05-09-2022 | 5y | F | ABSENT | | | | | | | | S | 18 | NVD | 37 | n | yes | 19 | n | | trauma | | |
| 11673065 | 05-09-2022 | 4y | M | ABSENT | | | | | | | | I | 19 | NVD | 38 | n | NIL | | n | | lipoma | | |
| 176500 | 05-12-2022 | 7y | M | ABSENT | | | | | | | | S | | CS | 37 | N | NIL | | n | | cleft palate | | |
| 11766780 | 20-05-2022 | 4y | M | ABSENT | | | | | | | | S | 22 | NVD | 37 | n | NIL | | n | | trauma | | |
| 1778862 | 23-05-2022 | 2y | M | PRESENT | 12 | 62 | rt basal ganglia | Lentiform nucleas | N | n | n | S | 17 | NVD | 35 | 2.4 | Yes | 14 | n | | | | |
| 11788694 | 23-05-20222 | 6y | M | PRESENT | 16 | 65 | Bilateral basal ganglia | Lentiform nucleas | N | n | n | I | 20 | NVD | 37 | 2.5 | no | | n | | | | |
| 177940 | 24-05-2022 | 10Y | M | ABSENT | | | | | | | | S | 18 | NVD | 37 | LBW | no | | yes | | lukemia | | |
| 117921413 | 24-05-2022 | 12y | F | ABSENT | | | | | | | | S | 18 | NVD | 38 | n | n | | n | | trauma | | |
| 12339138 | 06-03-2022 | 5Y | F | ABSENT | | | | | | bl thalami | n | I | | CS | 37 | n | no | | yes | | Vein of galen malformation | | |
| 1892304 | 06-05-2022 | 3Y | M | ABSENT | | | | | | | | I | 18 | NVD | 37 | n | NIL | | n | | trauma | | |
| 179642 | 06-04-2022 | 10y | M | ABSENT | | | | | | | | S | | CS | 37 | n | NIL | | n | | | | |
| 11911100 | 06-06-2022 | 6y | F | ABSENT | | | | | | | | S | 16 | NVD | 38 | n | yes | 18 | n | | trauma | | |
| 179942 | 06-06-2022 | 6y | M | ABSENT | | | | | | | | S | 18 | NVD | 37 | n | NIL | | n | | trauma | | |
| 11953320 | 06-01-2022 | 12y | F | PRESENT | 14 | 62 | Lt basal ganglia | lentiform nucleas | Yes | n | n | I | 22 | NVD | 37 | 2.6 | n | | Yes | | | | |
| 11966219 | 06-12-2022 | 2y | F | PRESENT | 10 | 58 | Bilateral basal ganglia | lentiform nucleas | No | n | n | S | 22 | NVD | 38 | 2.4 | | 18 | n | | trauma | | |
| 11982403 | 14-06-2022 | 1Y | F | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | trauma | | |
| 181255 | 15-06-2022 | 6y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | VP shunt | | |
| 119988501 | 15-06-2022 | 8y | M | ABSENT | | | | | | | | I | 18 | NVD | T | n | NIL | | n | | trauma | | |
| 180818 | 16-06-2022 | 10y | F | ABSENT | | | | | | | | S | | CS | T | n | NIL | | n | | trauma | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12007100 | 16-06-2022 | 7y | M | PRESENT | 16 | 60 | Bilateral basal ganglia | lentiform nucleas | Y | n | n | S | 20 | NVD | 39 | 2.7 | n | | yes | | trauma | | |
| 181472 | 16-06-2022 | 8y | m | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | n | | | | |
| 1219300 | 17-06-2022 | 5y | M | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | | | |
| 12032545 | 19-06-2022 | 2y | m | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | TRAUMA | | |
| 181935 | 19-06-2022 | 6Y | F | ABSENT | | | | | | | | I | 18 | NVD | T | n | NIL | | n | | TRAUMA | | |
| 2067565 | 22-06-2022 | 8y | M | Present | 13 | 62 | lt basal ganglia | lentiform nuleas | N | n | n | S | 22 | NVS | 35 | 2.7 | no | 12 | n | | TRAUMA | | |
| 182755 | 23-06-2022 | 8y | f | ABSENT | | | | | | | | S | | CS | T | N | NIL | | YES | | TRAUMA | | |
| 1215753 | 27-06-2022 | 2y | m | Present | 5 | 65 | rt basal ganglia | lentiform nucleas | N | n | n | S | 22 | CS | 35 | 2.4 | yes | 24 | n | | DMS | | MK |
| 12113054 | 27-06-2022 | 8y | F | Present | 6 | 66 | lt Basal ganglia | lentiform nucleas | N | n | n | S | 24 | NVD | 39 | 2.5 | yes | 21 | n | | trauma | | |
| 184539 | 07-05-2022 | 10y | M | Present | 8 | 61 | rt basal ganglia | lentiform nucleas | N | n | n | S | 22 | NVD | 40 | 2.6 | no | | n | | trauma | | |
| 184676 | 07-05-2022 | 9y | M | ABSENT | | | | | | | | S | 22 | NVD | T | n | n | | n | | | | |
| 184684 | 07-05-2022 | 10y | M | ABSENT | | | | | | | | S | | CS | T | N | n | | n | | | | |
| 1126701 | 14-07-2022 | 5y | F | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | DIZZINESS | | |
| 1125622 | 16-07-2022 | 3y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | trauma | | |
| 186397 | 16-07-2022 | 8y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 186413 | 16-07-2022 | 2y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 1124569 | 17-07-2022 | 7y | M | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | trauma | | |
| 188548 | 28-07-2022 | 2y | F | PRESENT | 11 | 57 | Lt basal ganglia | lentiform nucleas | N | n | yes- left putamen | I | 18 | NVD | 38 | 2.7 | Yes | 14 | n | | trauma | | |
| 188557 | 28-07-2022 | 4y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 112977 | 29-07-2022 | 6y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | SEIZURES | | |
| 188657 | 29-07-2022 | 6y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | TRAUMA | | |
| 188779 | 29-07-222 | 6y | M | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | TRAUMA | | |
| 189117 | 08-01-2022 | 7y | F | ABSENT | 12 | 54 | rt lentiform nucleas | lentiform nucleas | | | yes right putamen | S | 20 | NVD | T | n | NIL | | N | | TRAUMA | | |
| 1132160 | 08-09-2022 | 5Y | M | ABSENT | | | | | | | | S | 18 | NVD | PT | LBW | no | | yes | | trauma | | |
| 12598150 | 08-09-2022 | 12yr | M | PRESENT | 22 | 72 | Bilaterla basal ganglia | lentiform nucleas | Y | n | n | S | 20 | NVD | 39 | 2.6 | n | | yes | | trauma | | |
| 1133341 | 13-08-2022 | 5y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 12671084 | 17-08-222 | 2y | F | PRESENT | 6 | 52 | lt basal ganglia | lentiform nucleas | Y | n | n | I | 20 | NVD | 39 | 2.7 | yes | 18 | N | | trauma | | |
| 191940 | 08-07-2022 | 2y | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | trauma | | |
| 12887663 | 18-08-202 | 10yr | M | PRESENT | 9 | 59 | rt basal ganglia | lentiform nucleas | n | n | n | S | 20 | NVD | 38 | 2.7 | n | | yes | | trigonocephaly | | |
| 192530 | 20-08-2022 | 2y | M | ABSENT | | | | | | | | S | 17 | NVD | T | | NIL | | N | | TRAUMA | | |
| 192771 | 22-08-2022 | 9Y | m | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 192872 | 22-08-2022 | 6y | M | ABSENT | | | | | | | | I | 18 | NVD | T | n | NIL | | n | | cleft lip | | |
| 1135225 | 23-08-2022 | 6y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 12771804 | 25-08-2022 | 10 yr | M | Present | 7 | 55 | lt basal ganlglia | lentiform nucleas | N | n | n | I | 20 | NVD | 39 | 2.7 | N | | Yes | | hydrocehpalus post-VP | | |
| 1138549 | 09-09-2022 | 2y | F | ABSENT | | | | | n | n | n | S | 16 | NVD | | 2.8 | nil | | no | | Meningits | | |
| 4050692 | 09-12-2022 | 10y | F | Present | 12 | 1200 | bl basal ganglia | bl lentiofrm nucleas, thalamus & lt frontal region | | | N | I | | CS | 35 | 2.9 | yes | 24 | yes | | TB meningits POSr VP | | |
| 197778 | 17-09-2022 | 6Y | M | ABSENT | | | | | | | | S | 17 | NVD | T | N | NIL | | N | | TRAUMA | | |
| 198597 | 22-09-2022 | 7y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | TRAUMA | | |
| 1141718 | 23-09-2022 | 7y | M | ABSENT | | | | | | | | I | 18 | NVD | T | n | yes | 16 | n | | TRAUMA | | |
| 1142164 | 24-09-2022 | 8y | F | Present | 21 | | bilateral thalami | thalami and | n | subcortical white matter | N | I | | CS | 38 | 2.5 | m | | YES | | Congenital taxoplasmosis post VP | | |
| 1143944 | 30-09-2022 | 7Y | M | ABSENT | | | | | | | | I | 18 | NVD | T | n | NIL | | n | | Trauma | | |
| 200991 | 06-06-2022 | 2Y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 5503493 | 10-07-2022 | 7m | F | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 201859 | 10-11-2022 | 3Y | F | PRESENT | 3 | 55 | rt basal ganglia | lentiform nucleas | N | n | n | S | | CS | 39 | 2.4 | yes | 24 | no | | trauma | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 843185207 | 16-10-202 | 7m | M | PRESENT | 3 | 55 | rt basal ganglia | lentiform nucleas | n | n | n | S | 22 | NVD | 38 | 2.7 | yes | | no | | delayed mile stones | yes | MK |
| 2015485 | 17-10-2022 | 2y | M | ABSENT | | | | | | | | S | 20 | NVD | T | n | NIL | | N | | | | |
| 6780379 | 25-10-2022 | 1y | F | PRESENT | 4 | 58 | rt basal ganglia | lentiform nucleas | N | n | n | S | 22 | NVD | 37 | 2.7 | yes | 18 | n | | trauma | yes | MK |
| 6781317 | 28-10-2022 | 1y | F | PRESENT | | | Lt basal ganglia | lentiform nucelas | n | n | n | S | 24 | NVD | 37 | 2.7 | yes | 18 | n | | | | |