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**ORIGINAL ARTICLE**

# COMPUTED TOMOGRAPHY EVALUATION OF SPONTANEOUS INTRACRANIAL HAEMORRHAGE

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**ABSTRACT**

**AIM:** To assess the site, type and volume of Intracranial Hemorrhage using Computed Tomography. **METHODOLOGY:** Patients with clinical symptoms suggestive of Intracranial Hemorrhage were evaluated by Computed Tomography. **RESULTS:** Assessment of Non- traumatic Intracranial Hemorrhage in patients with clinical symptoms is made using Computed Tomography. Hypertension was assessed as the main causative factor for spontaneous Intracranial Hemorrhage. **CONCLUSION**: computed tomography has got higher sensitivity for detection of intra cranial hemorrhage. CT imaging provides detailed information regarding site, type, volume and etiology of intra cranial hemorrhage.

**Keywords:** Computed tomography, spontaneous intra cranial hemorrhage, sub arachanoid hemorrhage, hypertension, incidence.

## 1.INDRODUCTION

Intracranial haemorrhage is a common outcome of acute cerebrovascular accidents which may lead to neurological deterioration and is a frequent indication for the urgent neuro imaging. The aim of imaging is to identify the type of haemorrhage and thus select the patient who requires either surgical or medical management. Craniocerebral trauma and hypertension are the two most important causes of intracranial haemorrhage.It is very important to detect intracranial haemorrhage as early as possible, for proper medical management. Before the advent of the computed tomography (CT) the main stay of diagnosis was mainly based on clinical expertise, plain skull roentgenograms, cerebral angiography and isotope brain scans. But with the advent of computed tomography in 1972 accurate and early diagnosis of intracranial haemorrhage has been achieved. CT has got high sensitivity of detection of intracranial haemorrhage. Within a short time CT scan image soft tissues as well as cranial bones with significant degree of accuracy. Hence it is possible to study the spectrum of intracranial haemorrhage in relation to the etiology and clinical presentation. Since the natural history of the diseases is not well known, the determination of the prognostic factors would contribute in selecting the therapeutic options. In the present study the CT features of fifty patients with intracranial haemorrhage have been analysed.

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## 2.MATERIALS AND METHODS

A total of 50 patients of whom males and females were studied. Age group varied from 20-80 years. Patients were selected from in patients department with clinical symptoms suggestive of intracranial hemaorhage.All the examination were performed on a PHILIPS MX 800 computed tomographic scanner.

Serial Axial CT Sections of brain is scanned and patients is positioned supine. Sections were obtained starting from orbitomeatel line at 5 mm interval in posterior fossa to Top of head and 10 mm interval thereafter. Scan time is reduced upto 1 second in uncooperative patients. Sedation with I.V. Diazepam (5 mg. To 20 mg.) was given patients who were restless.

The volume of the intracranial haemorrhage was measured by a rapid simplified method.

The formula used (A*B*C) / 2 is an approximation for the volume of an ellipsoid where A is the greatest haemorrhage diameter on axial CT scans, B is the largest diameter 90 to A and C is the number of CT slices with haemorrhage multiplied by the slice thickness.

The volume of the intracranial haemorrhage was further categorized in the following:

1. 0 – 29 cc.
2. 30 – 59 cc.
3. > 60 cc.

## 3.RESULTS

In This study comprising of 50 patients with nontraumatic spontaneous intracranial haemorrhage, 93% of the patients had an association with hypertension and 7% of the patients had no association with hypertension. There was male preponderance with age incidence was maximum around 60 years. Intraparenchymal haemorrhage was the commonest variety of the bleed(39) (78%) followed by subarachnoid haemorrhage (10) (20%) and intraventricular haemorrhage (1) (2%).In total 39 cases of intraparenchymal haemorrhage, the locations of bleed were lobar 10 (25.6%), basal ganglia 17 (43.6%), thalamic 4 (10.2%), thalamoganglia 3 (7.6%), cerebellar 3 (7.6%) and brain stem 2 (5.1%).

## 4.DISCUSSION

**SPONTANEOUS INTRACRANIAL HAEMORRHAGE**

Spontaneous intracranial haemorrhage is haemorrhage into the brain due to causes other than trauma. Approximately 10% of the strokes are intracerebral haemorrhages[(1)]. In the analysis of spontaneous intracranial haemorrhage, the location of haematoma is important. It is termed intraparenchymal when bleeding is into the brain parenchyma, subarachnoid when haemorrhage is into subarachnoid space around brain and intraventricular when bleeding has occurred purely into the ventricles of Brain[(2)].

**Types of spontaneous intracranial hemorrhage:** 1.primary intracerebral haemorrhage (small vessel damaged by chronic hypertension or amyloid angiopathy), 2.secondary intracerebral hemorrhage (association with vascular abnormalities, tumors, impaired coagulation), 3.hypertensive hematoma, 4.putaminal hemorrhage, 5. Lobar hemorrhage (begins at grey white junction and dissects linear along white matter tracts), 6.thalamic hemorrhage, 7. Cerebellar hemorrhage, 8.pontine hemorrhage,9.intraventricular hemorrhage.10.sub arachanoid hemorrhage(rupture aneurysm is major etiology).

**COMPUTEDTOMOGRAPHY INTRAPARENCHYMAL HAEMORRHAGE:**

The computed Tomography features of intraparenchymal haemorhage has a linear relationship between Computed Tomography value and haemorrhage. A non enhanced Computed Tomography demonstrates a well marginated hyperdense mass due to the high protein content of intact red blood cells. The attenuation value of fresh blood to be approximately 56 HU[(3)].

A fresh haematoma on NCCT appears as a homogenously dense (55-90HU) well defined lesion with a rounded tooval configuration. A thin well defined low density zone surrounding the haematoma can be observed as early as a few hours after the haemorrhage. This rim is caused by clotting of liquid haemorrhage with extrusion of low-density plasma at the periphery of haematoma[(4)].

After 3-4 days additional low density on Computed Tomography appears around haematoma spreading peripherally in the white matter.

Haematoma produce ventricular compression and when large considerable midline shift with brain herniation[(4,5)].

From 3rd to 7th day mass effect may increase due to development of edema. A haematoma if non homogeneousuly dense should be due to haemorrhage occurring within a tumour, inflammation, contusion or arterial or venous infarct. CECT will reveal abnormal enhancement within the haemorrhage and helps in differentiation. In anaemic patient acute haemorrhage can be isodense to brain[(4)].

Haematoma of 2 cm or less reach isodensity on or before 19th day, while large haematomas took 4-6 weeks to become isodense.

The decreasing density is due to lysis of RBC. Haematoma lose their density from periphery inward and therefore show a progressive decrease in apparent size. Although visualized portion of haematoma becomes smaller on computed Tomography, the actual size of clot does not change significantly. It merely becomes isodense. Mass effect may be prominent for as long as 4 weeks with a large haematoma. Dolkinkasi et al noted that the earliest visualized decrease in mass effect for haematoma of all sizes average 16.7 days after bleeding[(6)].

Over next 2-3 months on Computed Tomography, density of a haematoma is completely lost. After passing through isodense stage, the haematoma becomes hypodense. At its end stage which may vary between 3 and 6 months depending on initial size a well-defined low-density region which may be considerably smaller than original lesion is present at the site of original haematoma. With small haematomas a slit like cystic area may be the residual change. Atrophic dilatation of the adjacent portion of the ventricle occurs as well as sulcal enlargement. Rarely calcification occurs at haematoma site.

After 7-9 days, contrast enhancement develops around the periphery of haematoma. Pathologically there is ingrowth of capillaries, neovascularity at the margin of haematoma by the end of first week. This newly formed capillaries have an abnormal blood brain barrier, resulting in enhancement with contrast around haematoma (Ring enhancement).

At the time when the haematoma is passing through its isodence stage NCCT may show little abnormality except a slight residual mass effect. However, the ring contrast enhancement on CECT persists through the isodense period and into the first few months of the hypodense state. The surrounding edema is clearing during the third to fourth week and the ring enhancement then appears to surround an isodense or hypodense core with normal surrounding brain tissue or no mass effect[(4)].

The enhancing capsule becomes more intense and thicker over the next 4-6 weeks before beginning to fade. Pathologically at this stage there is well developed glial vascular capsule. The diameter of the enhancing ring decreases during the final hypondense stage as the gliotic capsule constricts around the absorbed haematoma. The Computed Tomography appearance of this enhancing ring may be confused with that of a tumour or abscess([4,7)].

The lack of enhancement of the central region when the haematoma is isodense and the lack of surrounding edema and mass effect, particularly during the hypondense phase, tends to strongly favour a diagnosis of resolving haematoma.

If the bleeding is very rapid or re-bleeding is occurring during the time of the CT examination, the blood that did not have enough time to retract is seen as isodense in comparison to the brain parenchyma. In conditions where this fresh blood accumulates in formerly retracted clot, is seen as a hypodense area in a hyperdense haematoma which is called ‘swirl’ sign[(8)].

**COMPUTED TOMOGRAPHY EVALUATION OF SUBARACHNOID HAEMORRHAGE:**

The CT scan usually provides confirmation by demonstrating subarachnoid blood as well as providing information on the location of the aneurysm that ruptured. In order to visualize the high absorptive CT appearance of subarachnoid blood on the non-contrast scan, a sufficient volume of haemorrage has to be present within the subarachnoid space to provide adequate spatial and contrast resolution. The length of time the high absorptive appearance of the blood remains is dependent on the volume of the haemorrhage in a given region. This may be as short as 24 hours but usually persists for as long as one week. As the clot is degraded with time, the high absorption becomes isodense, similar to nearby brain. At this time the isodense blood may obliterate the basal cisterns and the subarachnoid space. After one or two weeks, the cisternal spaces resume their low absorption values as the cerebrospinal fluid replaces the blood. Since the initial high absorption value of blood is due to the protein of the hemoglobin if the patient were anemic with a hemoglobin of approximately 5 gm per 100ml, the haemorrhage would be relatively more isodense on CT scan[(4)]. If only a small subarachnoid haemorrhage occurs, blood may not be evident on the original scan. The amount of blood present in the subarachnoid space has been evaluated in an attempt to determine whether serious vasospasm of the nearby vessels will occur[(9)].

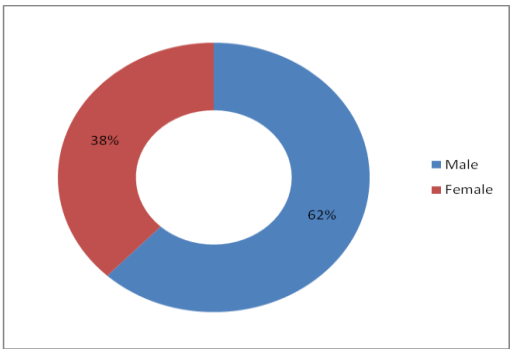
McCormack et al have described the 'flip-flop sign' whereby an aneurysm could be identified within a haematoma on non contrast computed tomography as a small area of relative low density which on following IV contrast infusion becomes higher in density than surrounding haematoma.

In order to make such an evaluation, the CT scan must be performed in the first 24 to 48 hours from onset of bleeding[(10)]. One of the most challenging problems in the treatment of aneurysms is the precise identification of the lesion and of its neck.

Cerebral angiography remains the gold standard in the exploration of the brain's vessels; however, it is an invasive procedure. CTA and MRA are less invasive procedures and their sensitivities appear to be nearly equivalent to that of angiography. Both 3D-CTA and MRA may fail to identify small aneurysms, the former especially if they are close to bony structures and the latter due to its flow artifacts. Virtual endovascular methods have already been proposed to display the inner structure of intracranial vascular ectasias. Although providing some information on the diameter and location of the aneurysms and of their neck, they cannot be considered as genuine vascular endoscopy methodologies as they provide only an outside view of the lumen of the vessels through a window opened on its walls.

**RESULTS AND OBSERVATION SEX DISTRIBUTION OF NTSICH**

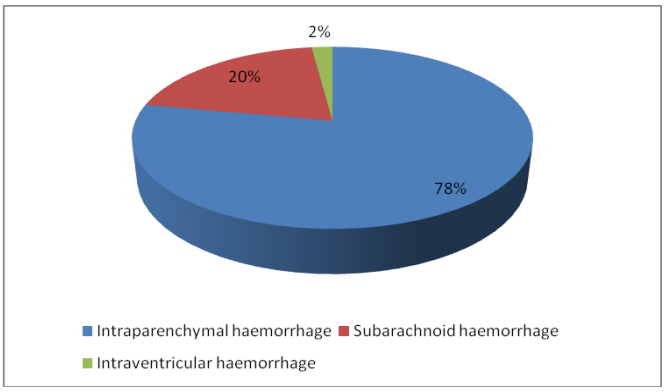
| Sex | No. of Patients | Percentage |
|---|---|---|
| Male | 31 | 62% |
| Female | 19 | 38% |
| Total | 50 | 100% |




**SEX INCIDENCE OF NTSICH:** In our study 62% patients were male and remaining 38% were females.

**SITE OF SPONTANEOUS INTRACRANIAL HAEMORRHAGE**

| Site | No. of Patients | Percentage |
|---|---|---|
| Intraparenchymal haemorrhage | 39 | 78% |
| Subarachnoid haemorrhage | 10 | 20% |
| Intraventricular haemorrhage | 1 | 2% |
| Total | 50 | 100% |




In our study 78% patients had intraparenchymal haemorrhage,20% patients had primary subarachnoid haemorrhage and remaining 2% had primary intraventricular haemorrhage.

**AGE DISTRIBUTION OF NTSICH**

| Age | Non-Traumatic Spontaneous Intracranial Haemorrhage | | |
|---|---|---|---|
| | IPH | SAH | IVH |
| 20-29 | 1 | 1 | 0 |
| 30-39 | 2 | 2 | 0 |
| 40-49 | 4 | 3 | 0 |
| 50-59 | 7 | 2 | 0 |
| 60-69 | 15 | 1 | 0 |
| > 70 | 10 | 1 | 1 |
| Total | 39 | 10 | 1 |

**AGE Distribution NTSICH**

Age distribution for IPH is maximum between 60-69 age group. Age distribution for SAH is maximum between 50-59 age group. And only one case of primary IVH which was seen in above 70 yearsage group.

Mean age for intraparenchymal haemorrhage was 67 years.

**INCIDENCE RELATIONSHIP BETWEEN INTRA PARENCHYMAL HAEMORRHAGE AND SAH AMONG 50**

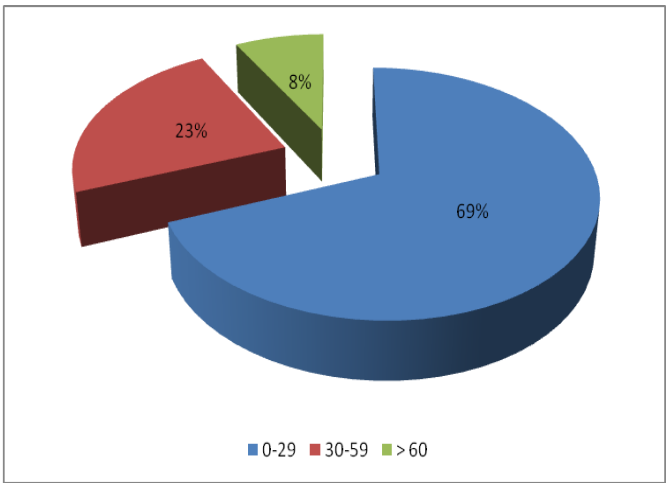
**PATIENT OF NON-TRAUMATIC SPONTANEOUS INTRACRANIAL HAEMORRHAGE:**

In our study of 50 patients of non-traumatic spontaneous intracranial haemorrhage.We have observed that number of intraparenchymal haemorrhage was more than twice the number of subarachnoid haemorrhage.

Number of patients with intraparenchymal haemorrhage 39.
Number of patients with subarachnoid haemorrhage 10.

**VOLUME OF BLEED IN 39 PATIENTS OF INTRAPARENCHYMAL HAEMORRHAGE:**

| Volume (ml) | No. of Patients | Percentage |
|---|---|---|
| 0-29 | 27 | 69% |
| 30-59 | 9 | 23% |
| > 60 | 3 | 8% |
| Total | 39 | 100% |



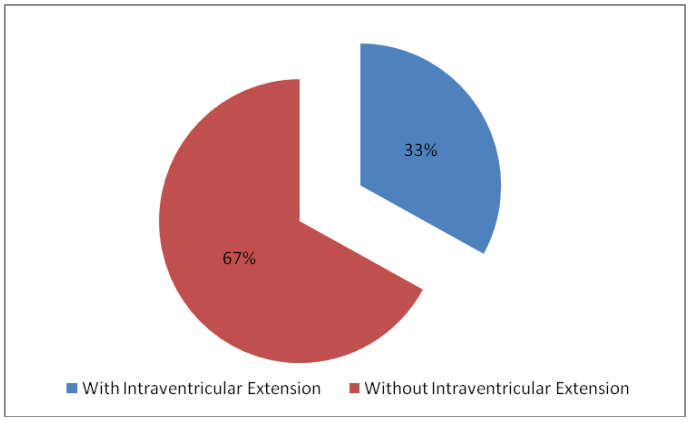

**Volume of Haematoma**: 27 out of 39 patients of intraparenchymal haemorrhage had volume of bleed in the range of 0-29ml (71%).

9 out of 39 patients of intraparenchymal haemorrhage had volume of bleed in the range of 30-59ml (24%).

4 out of 39 patients of intraparenchymal haemorrhage had volume of bleed in the range of above 60ml (5%).

**INTRAVENTRICULAR EXTENSION OF BLEED IN 78 PATIENTS OF INTRAPARENCHYMAL HAEMORRHAGE"**

| | No. of Patients | Percentage |
|---|---|---|
| With Intraventricular Extension | 13 | 33% |
| Without Intraventricular Extension | 26 | 67% |
| Total | 39 | 100% |

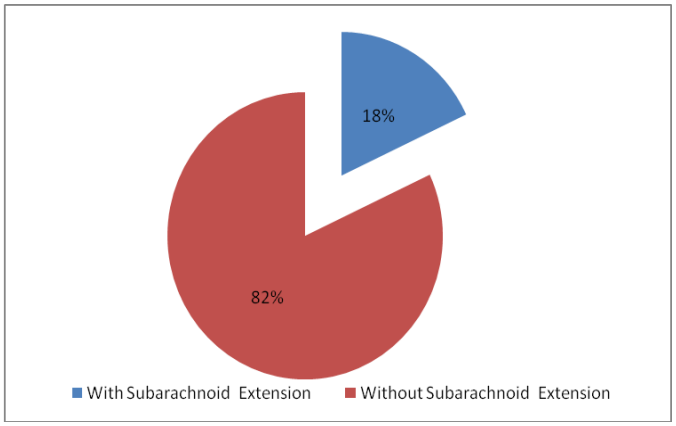



**Intraventricular Extension of Intraparenchymal Bleed:**

Out of 39 patients of intraparenchymal haemorrage 13 patients had intraventricular extension of the bleed (32%).

**SUBARACHNOID EXTENSION OF BLEED IN 78 PATIENTS OF INTRAPARENCHYMAL HAEMORRHAGE**

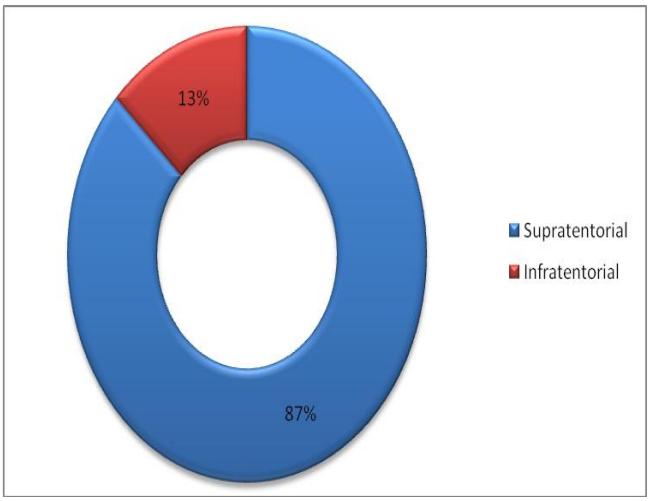
| | No. of Patients | Percentage |
|---|---|---|
| With Subarachnoid Extension | 7 | 18% |
| Without Subarachnoid Extension | 32 | 82% |
| Total | 39 | 100% |




**Subarachnoid Extension of Intraparenchymal Bleed:**
Out of 39 patients of intraparenchymal haemorrage 7 patients had subarachnoid extension of the bleed (18%).

**DISTRIBUTION OF INTRAPARENCHYMAL AEMORRHAGE**

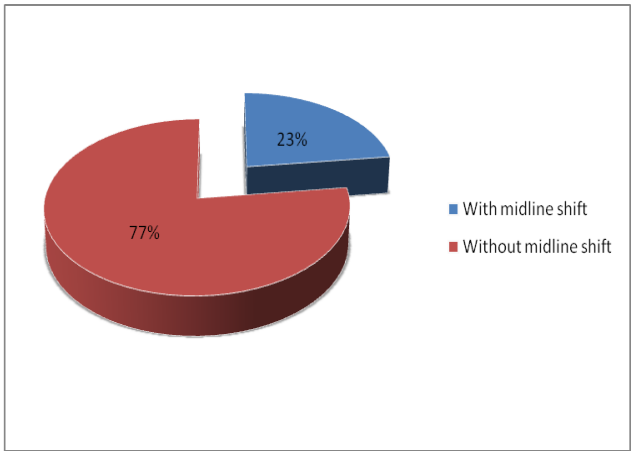
| Location | No. of Patients | Percentage |
|---|---|---|
| Supratentorial | 34 | 87% |
| Infratentorial | 5 | 13% |
| Total | 39 | 100% |




**Distribution of Intraparenchymal Haemorrhage**
Total number of patients with intraparenchymal haemorrhage is 39.
Supratentorial location of bleed is 34 (87%).
Infratentorial location of bleed is 5 (13%).

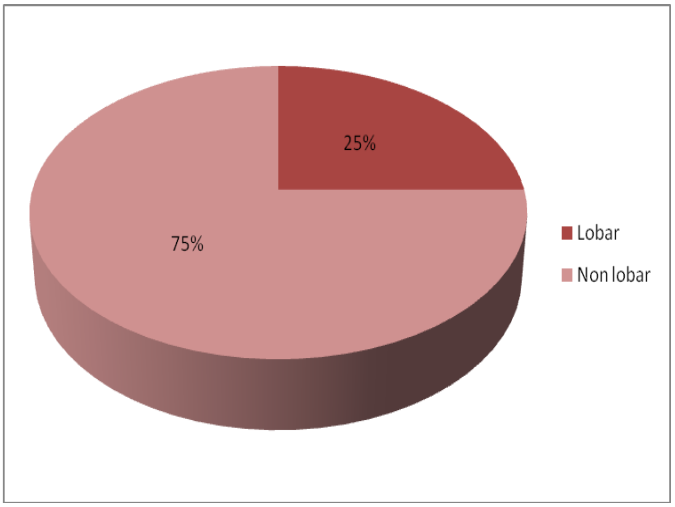
**INTRAPARENCHYMAL HAEMORRHAGE WITH MASS EFFECT IN THE FORM OF MIDLINE SHIFT:**

| Intraparenchymal Haemorrhage | No. of Patients | Percentage |
|---|---|---|
| With midline shift | 9 | 23% |
| Without midline shift | 30 | 77% |
| Total | 39 | 100% |




**LOCATION OF INTRAPARENCHYMAL HAEMORRHAGE AS LOBAR AND NON-LOBAR SUBTYPES**

| Location | No. of Patients | Percentage |
|---|---|---|
| Lobar | 10 | 25% |
| Non lobar | 29 | 75% |
| Total | 39 | 100% |




**Location of Intraparenchymal Haemorrhage as Lobar and Non-Lobar Subtypes:**In this study of 78 patients of intraparenchymal haemorrhage 75% of patients (29) showed non lobar location and 25% of patients (10)showed lobar location of the bleed.

**SITES OF HYPERTENSIVE INTRAPARENCHYMAL HAEMORRHAGE**

| Sites | No. of Patients | Percentage |
|---|---|---|
| Lobar | 10 | 25.6% |
| Basal Ganglia | 17 | 43.6% |
| Thalamus | 4 | 10.2% |
| Thalamo Ganglia | 3 | 7.6% |
| Cerebellum | 3 | 7.6% |
| Brain Stem | 2 | 5.1% |
| Total | 39 | 100% |

## 5.CONCLUSION

In our study we have concluded that, sex incidence of non traumatic spontaneous intracranial haemorrhage is higher among males. Incidence of non traumatic spontaneous intracranial haemorrhage is high around 60 years of age. Intra parenchymal haemorrhage was the commonest variety. Thalamus and basal ganglia are the commonest sites of hypertensive intraparenchymal haemorrhage, intraparenchymal haemorrhage twice the more common than subarachnoid haemorrhage. Hypertension is the major causative factor for non-traumatic spontaneous intracranial haemorrhage. Peak age group for subarachnoid haemorrhage was 40-59 years and peak age group of intraparenchymal haemorrhage was 50-69 years.

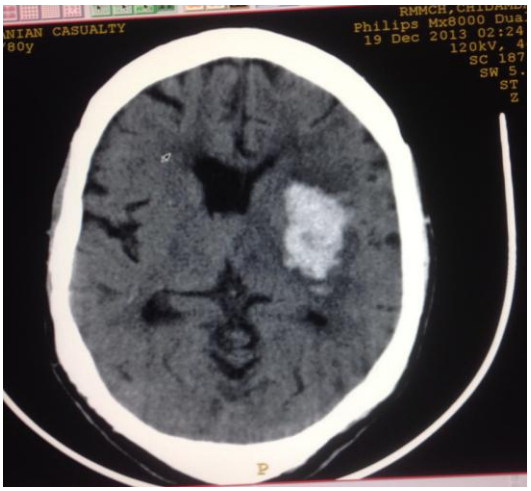
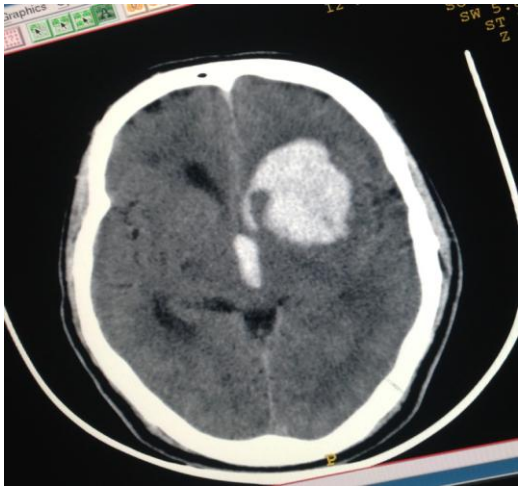
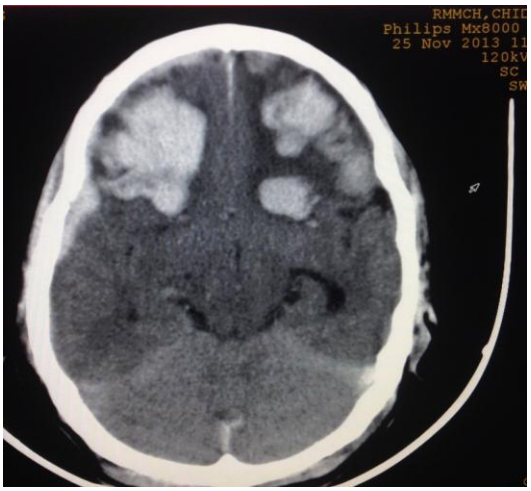


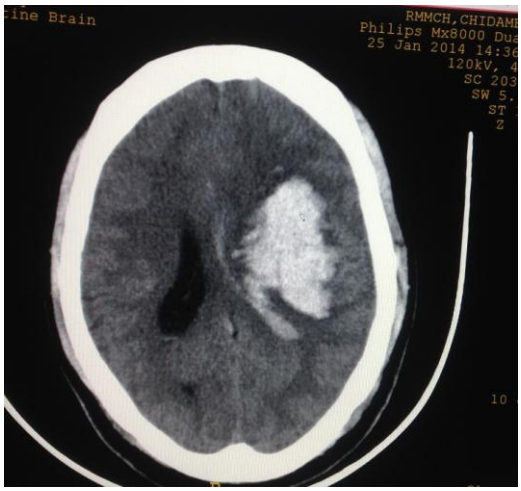

Image Showing left intraparenchymal hemorrhage with surrounding edema



(L) Intraparenchymal hemorrhage with intraventicular extension into third ventricle




Bilateral frontal intraparenchymal hemorrhage with subarachanoid space hemorrhage




(L) Intraparenchymal hemorrhage with extension into (L) lateral ventricle

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