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CT EVALUATION OF NON-TRAUMATIC INTRACEREBRAL HAEMORRHAGE IN ADULTS

Radiodiagnosis

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ABSTRACT

Intracranial haemorrhage is a devastating disorder with poor prognosis and high mortality rates1and hence is an indication for urgent neuroimaging. Advances in neuroimaging techniques have improved diagnostic capabilities, enabling understanding of the underlying pathophysiology and etiology of intracranial haemorrhage, and helped to establish prognosis. CT with a high sensitivity in detection of intracranial haemorrhage can detect within a short time, also can image soft tissues &cranial bones with a significant degree of accuracy.

AIMS AND OBJECTIVES

- Evaluation of the site and type of intracranial haemorrhage.
- Evaluation of the volume of intracranial haemorrhage.
- Evaluation of the intraventricular extension of haemorrhage.
- Evaluation of mass effect in the form of midline shift and cerebral herniation.
- Evaluation of subarachnoid extension.

MATERIALS AND METHODS

- **Place:** This study was carried out from the cases collected from the Department Of Radio diagnosis, Alluri Sitaram Raju Academy of Medical Sciences, Eluru.
- **Duration:** 18 months between October 2016 and March 2018
- **Study Design:** Prospective study.
- **Source Of Data:** All patients with symptoms and signs suggestive of ICH referred for noncontrast plain CT scan of the head.
- **Study Population :** 100 cases
- **Technique:** Serial CT Sections of the brain were obtained from orbitomeatal line at 5 mm interval in posterior fossa and 10 mm interval thereafter. Thin cuts were taken wherever necessary. Scan time is reduced to up to 1 second in uncooperative patients.

Protocol

A) Standard Brain (Transverse Scan)

- Patients' position: Supine.
- Gantry angulation : -50 to orbito metal line.
- Start position: External auditory meatus.
- End position: Top of the head.
- Slice thickness: 10mm.

B) Standard Posterior Fossa (Transverse Scan)

- Patient Position: Supine
- Gantry Angulation: Parallel to orbito metal line.
- Start Position: Foramen magnum
- End Position: One slice above petrous bone.
- Slice thickness: 5mm

KEYWORDS

ICH : Intra Cranial Haemorrhage, SAH : Sub Arachnoid Haemorrhage. IVH: Intra Ventricular Haemorrhage

INTRODUCTION

ICH accounts for 10 to 15 percent of all cases of stroke[7]making it a common outcome of the acute cerebrovascular accident. It is associated with the highest mortality rate, with only 38 percent of affected patients surviving the first year. Craniocerebral trauma and hypertension are the two most important causes of intracranial haemorrhage.

CT with its high sensitivity of detection of intracranial haemorrhage, can swiftly image soft tissues as well as cranial bones with a significant degree of accuracy.

NON-TRAUMATIC INTRACRANIAL HAEMORRHAGE

ICH is an acute and spontaneous extravasation of blood into the brain parenchyma. 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 the brain and intraventricular when bleeding has occurred purely into the ventricle[8]. Rarely bleeding can occur within a tumor in the brain when it is termed intratumoral haemorrhage.

Depending on the underlying cause of bleeding, ICH is classified as either primary or secondary. Primary intracerebral haemorrhage (PICH), accounting for 78 to 88% of cases, originates from the spontaneous rupture of small vessels damaged by chronic hypertension or amyloid angiopathy.

Hypertension is a major risk factor both for ischaemic cerebrovascular disease and for intracranial haemorrhage. Secondary ICH occurs in a minority of patients in association with vascular abnormalities such as vascular malformation, vasculitis, moya-moya disease, aneurysm, cortical vein/ sinus thrombosis, neoplasm, trauma, postoperative event, hyperviscosity syndrome, hemorrhagic diathesis, or ischaemic stroke.

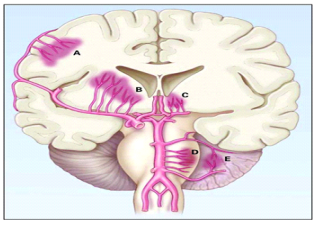

Figure : Most common sites and sources

ICH most commonly involve cerebral lobes, originating from

A. penetrating cortical branches of the anterior, middle, or posterior cerebral arteries
B. basal ganglia, originating from ascending lenticulostriate branches of the middle cerebral artery
C. the thalamus, originating from ascending thalmogeniculate branches of the posterior cerebral artery the pons, originating from paramedian branches of the basilar artery and
D. the cerebellum, originating from penetrating branches of the posterior inferior, anterior inferior, or superior cerebellar arteries.

PATHOLOGIC PROCESS

ICH commonly occur in the cerebral lobes, basal ganglia, thalamus, brain stem (predominantly the pons), and cerebellum. Extension into the ventricles occurs in association with deep, large hematomas. Edematous parenchyma, often discoloured by degradation products of hemoglobin, is visible adjacent to the clot. Histologic sections are characterized by the presence of edema, neuronal damage, macrophages, and neutrophils in the region surrounding the hematoma. The haemorrhage spreads between planes of white-matter cleavage with minimal destruction, leaving nests of intact neural tissue within and surrounding the hematoma. This pattern of spread accounts for the presence of viable and salvageable neural tissue in the immediate vicinity of the hematoma.[9]

Pathophysiology Of Evolution Of Intracerebral Haemorrhage

Intracranial hematomas follow well-described, predictable pathophysiological processes of evolution and resorption. Five distinct evolutionary phases can be identified:

- hyperacute (less than 12 hours after onset),
- acute (12 hours to two days),
- early subacute (two to seven days),
- late subacute (eight days to a month), and
- Chronic (more than one month).

Factors that influence hematoma evolution include

- location (intra or extra-axial, gray versus white matter),
- size (punctuate versus confluent, unifocal versus multifocal),
- etiology (arterial versus venous, trauma versus hypertension), and
- temporal occurrence of haemorrhage (acute event versus multistaged, recurrent haemorrhage).

It is a complex process with 3 distinct phases:

1. initial haemorrhage,
2. hematoma expansion, and
3. perihematoma edema.

The mechanisms of early hematoma growth are unclear but likely to be related to sudden increases in intracranial pressure (ICP), causing local tissue distortion and disruption, vascular engorgement secondary to obstructed venous outflow, blood-brain barrier disruption, and a local coagulopathy secondary to release of tissue thromboplastin.

Hematoma expansion is an important cause of early neurological deterioration, the severity of which depends on the original hematoma size and subsequent expansion rate.

There is an exponential increase in mortality when the hematoma volume exceeds 30 mL[4].Perihematoma brain edema develops early, evolves over many days, and is the primary cause of neurological deterioration after the first day. It occurs mainly as a result of an inflammatory response secondary to a local release from the hematoma of thrombin and other end products of coagulation, and also because of cytotoxic mediators and disruption of the blood-brain barrier.

CT IMAGING OF INTRAPARENCHYMAL HAEMORRHAGE

Progressive blood clot retraction increases haemorrhage density during the acute and early subacute phases, while progressive red blood cell lysis during the late subacute phase decreases the density of the hematoma. Progressive resorption of intra-axial hematoma in the chronic phase results in a hypodense brain defect filled with cerebrospinal fluid. Hematoma density is essentially determined by the degree of blood clot retraction, hematocrit, hemoglobin fraction, and protein concentration[3].Peripheral rim enhancement, may mimic focal brain tumors or infarctions. The degree of CSF dilution alters the density of extracerebral (subarachnoid, subdural) hematomas. Dilution of the hematomas by CSF may accelerate hematoma resolution.

A fresh haematoma on NECT appears as a homogeneously dense (55-90 HU) well-defined lesion with a round to oval configuration[10,11]. After 3-4 days additional low density on CTappears around haematoma spreads peripherally in the white matter. From 3rd to 7th-day mass effect may increase due to development of edema. Steroid treatment will control edema formation and reduce the surrounding increased mass effect both clinically and on brain imaging[12].

A haematoma if non homogeneously dense should be due to haemorrhage occurring within a tumor, 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[10].

Haematoma of 2 cm or less reach isodensity on or before 19thday, while large haematomas took 4-6 weeks to become isodense. The decreasing density is due to lysis of RBC. Haematoma loses their density from the periphery inward and therefore show a progressive decrease in apparent size. Mass effect may be prominent for as long as 4 weeks with a large haematoma.

Over the next 2-3 months on CT, the density of a haematoma is completely lost. After passing through the isodense stage, the haematoma becomes hypodense. At its end stage which may vary between 3-6 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.

RESULTS

Site Of Spontaneous Intracranial Haemorrhage

Site	No. of Patients	Percentage
Intraparenchymal haemorrhage	80	80%
Subarachnoid haemorrhage	19	19%
Intraventricular haemorrhage	1	1%
Total	100	100%

INTRAVENTRICULAR and SUBARACHNOID EXTENSION OF BLEED IN 80 PATIENTS OF INTRAPARENCHYMAL HAEMORRHAGE

Intraparenchymal Haemorrhage	No. of Patients	Percentage
With Intraventricular Extension	25	32%
Without Intraventricular Extension	55	68%
Total	**80**	**100%**
With Subarachnoid Extension	16	20%
Without Subarachnoid Extension	64	80%
Total	**80**	**100%**

INTRAPARENCHYMAL HAEMORRHAGE WITH MASS EFFECT IN THE FORM OF MIDLINE SHIFT

Intraparenchymal Haemorrhage	No of Patients	Percentage
With midline shift	20	25%
Without midline shift	60	75%
Total	80	100%

REPRESENTATIVE CASES

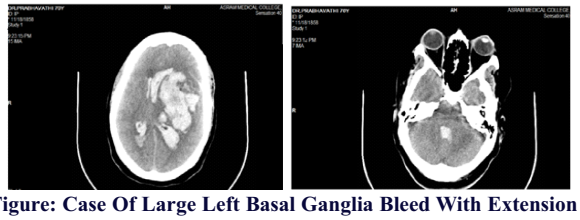

Figure: Case Of Large Left Basal Ganglia Bleed With Extension Into Lateral And Fourth Ventricles And Subdural Extension

DISCUSSION

CT provides a useful means of diagnosing intracranial haemorrhage.In this study of 100 patients with non-traumatic spontaneous intracranial haemorrhage 93 patients (i.e. 93%) were associated with hypertension and 7 patients (7%) without hypertension. Brottet al6 showed that hypertension is the most important risk factor for nontraumatic spontaneous intracranial haemorrhage. Adnan et al7 showed amyloid angiopathy and hypertension is the most factor for primary ICH.

In this study, non-traumatic spontaneous intracranial haemorrhage was higher among males (64%) as compared to females (36%). Franke et al 15 showed that higher incidence of intracranial haemorrhage is noted in males (57%).

Statistically, age is an important prognostic indicator in patients with non-traumatic spontaneous intracranial haemorrhage. In this study of incidence has gradually increased with age and is maximum around 60 to 69 years. A study conducted by Fewell ME2showed that men are more likely to suffer from spontaneous intracranial haemorrhage than women.

In this study, 75% of patients had the volume of haematoma in the range of 0-29 ml and 16 patients(20%) 30-59 ml and 5% of patients above 60 ml. Out of the 16 patients with volume in the range of 30-59 ml, 8 patients (50%) and all the patients (100%) with volume above 60 ml had a fatal outcome. There is an exponential increase in mortality when the hematoma volume exceeds 30 mL.

In this study, 80 patients had intraparenchymal haemorrhage (78%) and 19 patients had primary SAH (21%) and 1 patient had primary intraventricular haemorrhage (1%). Studies conducted by Broderick J.P.et al6 showed that IPH is more than twice as common as SAH. Obajami et al in his study showed that 84% of non-traumatic spontaneous intracranial haemorrhage were IPH and primary SAH accounted for 8% of the patients which is matching the present study.

The present study, one case of primary intraventricular haemorrhage had an association with advanced age (71years) and hypertension. In this study, it was proved that supratentorial location of the bleed (87%) was more common than infratentorial location (13%) and we have also shown that in lobar haemorrhage (75%) was commoner than lobar haemorrhage (25%).

In hypertensive IPH out of 80 patients, 36 patients had basal ganglia bleed (45%) followed by 20 cases of lobar bleed (25%), 9 cases of thalamic bleed (11.2%), 5 patients of thalamo ganglia bleed (6.2%), 6 patients of cerebellar bleed (7.5%), 4 patients of brain stem bleed (5%). Tatu L. Maulin T. et al23 showed the location of hypertensive ICH as lobar 36%, lenticular 32%, thalamic 15.7%, cerebellar 8.8%, brain stem 2%, intraventricular 2% and caudate 17%.

Mass effect in the form of a midline shift was noted in 20 cases of IPH (25%).

In this study of 80 patients with IPH, 25 patients (32%) had an intraventricular extension. Remaining 55 patients had no intraventricular extension (68%)

In this study of 80 patients with IPH, 16 patients (20%) have associated subarachnoid extension of the bleed. According to Carmen Corina Stancuet al71, the Subarachnoid extension is almost equally present in both sexes, but more frequently before 60 years and over 70 years. The typical features of ICH complicated with SAE included lobar site, mainly the lobar superficial areas, lobule shaped hematoma.

CONCLUSION

CT provides an excellent imaging modality for the early detection of intracranial haemorrhage which definitely improves patients management. In the present study, analysis of 100 patients with intracranial haemorrhage was done in above 20 years of age.

This study concludes following aspects of intracranial haemorrhage:

1) Hypertension is the factor for non-traumatic spontaneous intracranial haemorrhage.
2) The incidence of non-traumatic spontaneous intracranial haemorrhage is high around 60 years of age.
3) Sex incidence of intracranial haemorrhage is high in male.
4) IPH is thrice more common than SAH.
5) Thalamus and basal ganglia are the commonest sites of hypertensive IPH.
6) Peak age group of IPH was 60-69 years.
7) Peak age group for SAH was 40-59 years.

REFERENCES

1. History of neuroimaging [the American society of neuroradiology].
2. Fewell ME, Thompson GB. Spontaneous intracranial haemorrhage. A review of Neurosurgery 2002; 15: 1-8.
3. Osborne AG. Diagnostic neuroradiology, Intracranial hemorrhage. Harcourt Brace & Company, 1st edition Mosby 1997; 154-198.
4. Helen Fernandes. Spontaneous intracranial hemorrhage. Neurosurgery 2004; 22: 60-69.
5. Davis KR, Kistler JP, Heros RC et al. Neuroradiological approach to the patterns with diagnosis of subarachnoid haemorrhage. Radiological Clinics of North America. 1982; 20:87-95.
6. New England Journal Of Medicine, Vol. 344, No. 19 • May 10, 2001.
7. Adnan I, Oureshi M, Tuhrim S et al. Spontaneous intracranialhaemorrhage. New England Medicine Journal 2001; 3441:19-30.
8. Mathew P, Douglas. The central nervous system. In Robbins, Pathological basis of disease. Prism India 5th edition 1994, 1295-1355.
9. The Acute Management of Intracerebral Hemorrhage: A Clinical Review. Justine Elliott, FRCA, and Martin Smith, FRCA May 2010 , Volume 110 , Number 5, pages 1419-1427.
10. Brott T, Broderick J, Kothari R, Barsan W, Tomsick T, Sauerbeck L, Spilker J, Duldner J, Khoury J. Early hemorrhage growth in patients with intracerebral hemorrhage. Stroke 1997;28:1–5.
11. Mayer SA, Rincon F. Treatment of intracerebral haemorrhage. Lancet Neurology 2005;4:662–72
12. Tuhrim S, Horowitz DR, Sacher M, Godbold JH. Validation and comparison of models predicting survival following intracerebral hemorrhage. Crit Care Med 1995;23:950 – 4
13. Mayer SA, Sacco RL, Shi T, Mohr JP. Neurologic deterioration in noncomatose patients with supratentorial intracerebral hemorrhage. Neurology 1994;44:1379 – 84
14. Lee et al. Organized Intracranial haemorrhage with haemorrhage, CT patterns and pathological correlation. American Journal of Radiology 1986; 147: 113-118.
15. Franke J. Prognostic factors in patients with intracerebral haemorrhage. Journal of Neurosurgery, Neurology, Psychiatry 1991; 55: 653-657.
16. Carmen Corina Stancu et al. Morphoclinical study of intracerebral hemorrhage with subarachnoid effusion, Romanian Journal of Morphology & Embryology 2011, 52(1 Suppl):263–271