International Journal of
Science Inventions Today
www.ijsit.com
ISSN 2319-5436
Review Article

THE ROLE OF IMAGING IN DIAGNOSIS OF SUBDURAL HEMATOMA: REVIEW ARTICLE

*Dr. Sumendra Raj Pandey, Prof. Dr. Liu Pei WU, Dr. Sohan Kumar Sah, Dr. Lalu Yadav,

Md. Sadam Husen Haque and Rajan KR. Chaurasiya

Department of nuclear medicine and medical imaging, clinical medical college of Yangtze university, Jingzhou central hospital, province- hubei, PR china

ABSTRACT

A subdural hematoma (SDH) is a collection of blood below the inner layer of the dura but external to the brain and arachnoid membrane. SDHs are most commonly caused by shear forces, which tear the bridging veins transiting the subdural space.[1] Rarely, an SDH may be caused by rupture of an aneurysm or arteriovenous malformation[2]. SDH is the most common type of traumatic intracranial mass lesion. SDH occurs not only in patients with severe head injury but also in patients with less severe head injuries, particularly those who are elderly or who are receiving anticoagulants.

Key words: subdural hematoma (SDH), arteriovenous malformation, aneurysm

INTRODUCTION

A subdural hematoma is most often the result of a severe head injury. This type of subdural hematoma is among the deadliest of all head injuries. The bleeding fills the brain area very rapidly, compressing brain tissue. This often results in brain injury and may lead to death. Underlying brain injuries, such as hemorrhagic contusions and edema, are frequently seen with acute SDHs[3]. The degree of mass effect seen with an SDH is often disproportionate to the size of the SDH. This mass effect is due to the presence of other traumatic lesions such as hemorrhagic contusions and edema rather than the SDH[4]. The majority of acute subdural hematoma (SDH) occurs due to head trauma, and nontraumatic or "spontaneous" acute SDH is rare, which is caused by various etiologies such as cortical artery bleeding, vascular lesions, coagulopathy, neoplasms, spontaneous intracranial hypotension, cocaine, and arachnoid cyst.

This article provides an overview of the imaging of SDH, focusing on the correlation between radiological features and the underlying pathological processes.

APPEARANCE

The typical appearance of an acute SDH is a hyperdense crescent-shaped collection with a convex lateral border and concave medial border overlying the cerebral convexity [Fig. 1][5]. The majority of acute SDHs are hyperdense because of the attenuating properties of the hemoglobin molecule. An acute SDH may be isodense with brain parenchyma in patients with anemia as a result of a reduced concentration of haemoglobin[6]. Occasionally an SDH may be biconcave, simulating the appearance of EDH[7]. This biconcavity can be seen particularly when the SDH is large[8].

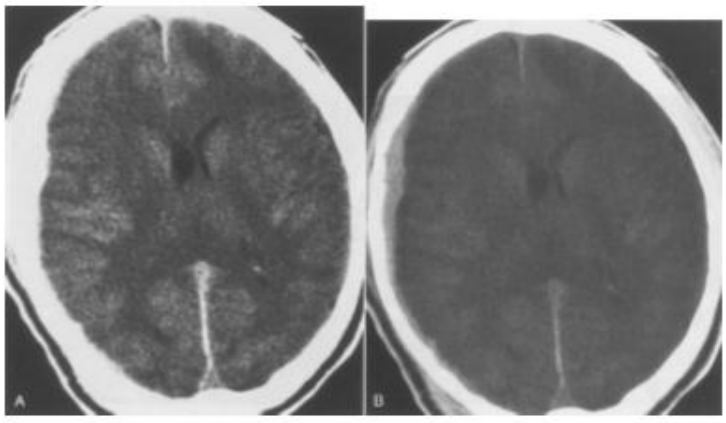

Figure 1: A, Crescentic right convexity acute subdural hematoma is evident from the irregular contour on the brain window setting. B, The subdural hematoma is better demonstrated with an intermediate window setting.

The classic homogeneous, hyperdense appearance of acute SDH is not always present. The SDH may appear mixed rather than homogeneous in attenuation[9]. It may have one of three patterns: marginal hypodensity, central irregular areas of hypodensity, or laminar areas of hypodensity [Figs. 2 and 3]. The low density may be secondary to unclotted blood or possibly CSF resulting from traumatic arachnoid tears. The mixed density SDH is usually larger and has more mass effect than the classic hyperdense SDH.

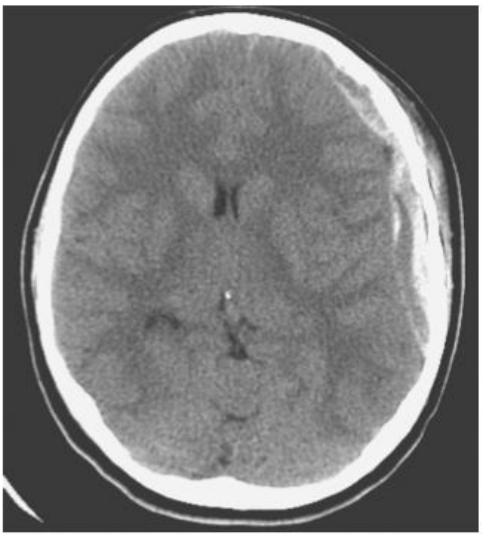

Figure 2: Acute left convexity subdural hematoma has marginal hypointensity representing unclotted blood or cerebrospinal fluid.

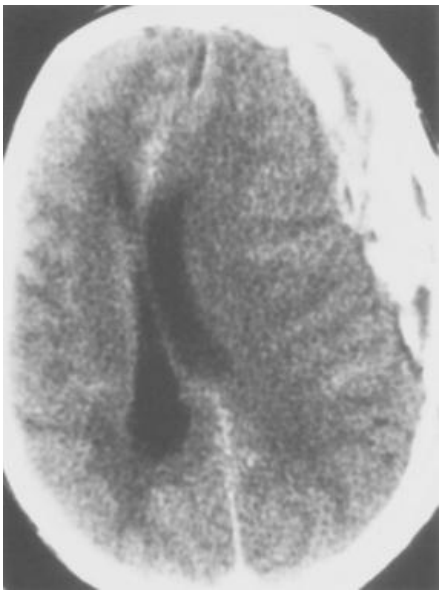

Figure 3: Irregular areas of hypointensity indicate that unclotted blood or cerebrospinal fluid is present in this acute left convexity subdural hematoma.

CLASSIFICATION

Subdural hematomas are divided into acute, subacute, and chronic, depending on the speed of their onset. Acute subdural hematomas that are due to trauma are the most lethal of all head injuries and have a high mortality rate if they are not rapidly treated with surgical decompression.

Chronic subdural bleeds develop over a period of days to weeks, often after minor head trauma, though such a cause is not identifiable in 50% of patients. The bleeding from a chronic bleed is slow, probably from repeated minor bleeds, and usually stops by itself. These are common in elderly

CT- IMAGING:

Chronic SDHs are most commonly hypodense on CT [Fig. 4][10]. Chronic SDHs are typically crescentic; however, they may be biconcave or lentiform as a result of fluid absorption into the hematoma[11]. Another possible cause is the formation of adhesions. A capsule composed of a capillary-rich membrane develops and surrounds the SDH[12].This capillary-rich membrane is responsible for repeated episodes of rebleeding and subsequent increase in size of the SDH. As the SDH enlarges, the patient becomes symptomatic, showing signs of increased intracranial pressure, hemiparesis, or intellectual and personality change. Repeated episodes of rebleeding may result in a mixed-density collection containing areas of hypodense, isodense, and increased density [Fig. 5]. A chronic SDH may appear hyperdense from acute hemorrhage, simulating an acute process[13]. Fluid-fluid levels may be seen as blood products settle in the dependent aspect of the subdural collection, which becomes hyperdense relative to the superior aspect. Enhancement of the SDH may be seen with delayed CT obtained 3 and 6 hours after intravenous administration of contrast material[14].

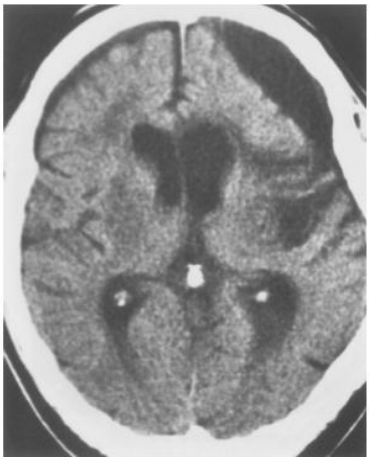

Figure 4: Bilateral frontal convexity chronic subdural hematomas are present. Note the shift of the septum pellucidum to the right.

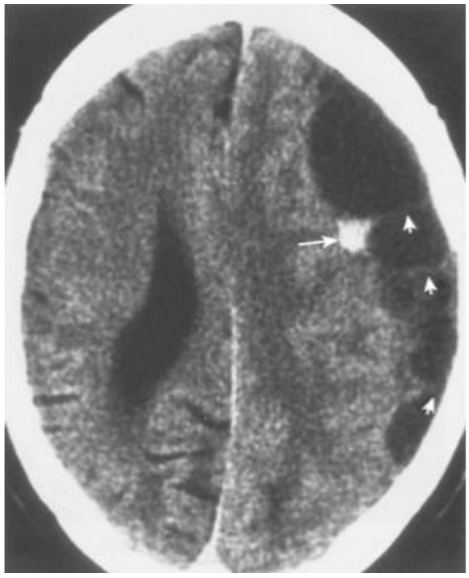

Figure 5: Large chronic subdural hematoma overlies the left cerebral convexity. The hyperdense area represents recurrent hemorrhage (long arrow). Note the septations within the hematoma (small arrows).

A subdural hygroma is a hypodense extracerebral collection equal in density to CSF [Fig. 6]. The lesions are crescentic and may be bilateral. It is not possible to distinguish a subdural hygroma from a chronic SDH with CT because the two lesions may be identical in appearance. At surgery, clear fluid and lack of a membrane allow the diagnosis of a subdural hygroma[15].

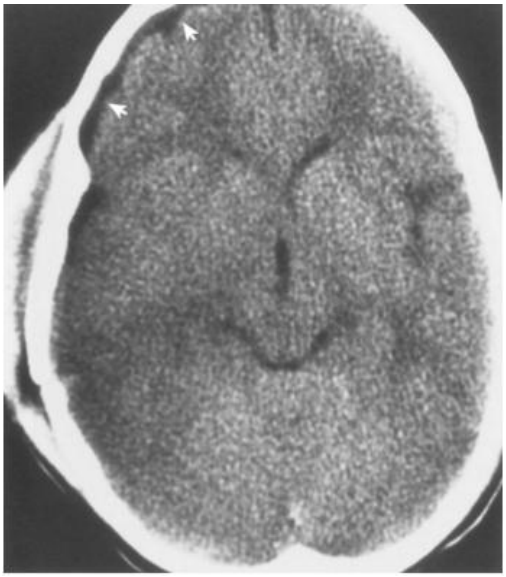

Figure 6: Cerebrospinal fluid–density collection representing a cerebrospinal fluid hygroma is present over the right frontal convexity (arrows). The initial CT scan 3 days earlier showed no subdural collection.

MR-IMAGING:

The MRI appearance of SDH has been well documented. In the acute phase (less than 1 week old), an SDH is isointense or slightly hypointense relative to gray matter on T1-weighted images and very hypointense on T2-weighted images. Early subacute SDHs (more than 1 week and less than 2 two weeks old) are characterized by a rim of hyperintensity surrounding a center of hypointensity on T1- and T2-weighted images. In the late subacute SDH (more than 2 weeks and less than 1 month old), the lesion is hyperintense on both T1- and T2-weighted images [Fig. 7]. Chronic SDHs (more than 1 month old) are isointense relative to gray matter on T1-weighted images and hyperintense on T2-weighted images [Fig. 8]. Usually no hypointensity is evident in chronic SDHs on T2-weighted images, most likely because of the lack of a blood–brain barrier and resorption of hemosiderin from the SDH. Occasionally hemosiderin may be seen in thickened membranes and areas of re-hemorrhage. Fluid-fluid levels of different signal intensity suggest rebleeding. It is possible to distinguish a chronic subdural hygroma from a chronic SDH because the hygroma is of CSF signal intensity on all pulse sequences [Fig. 9][16].

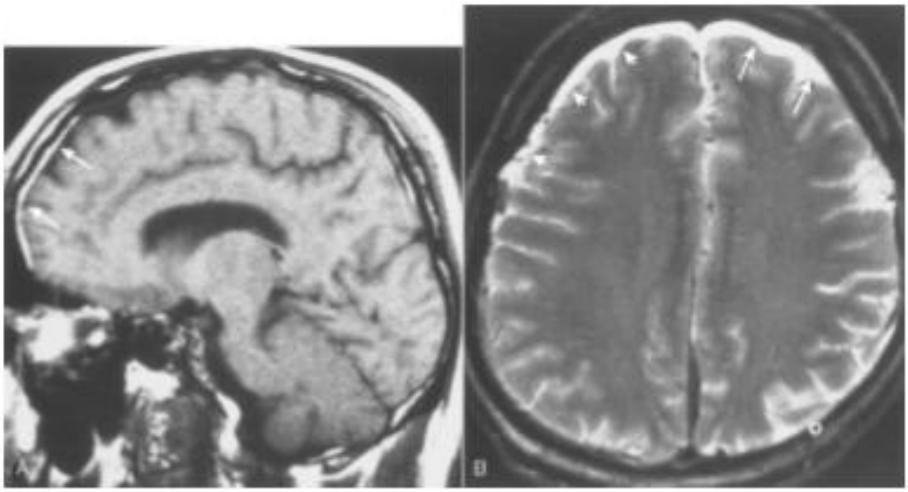

Figure 7: Sagittal T1-weighted (500/15) (A) and axial FSE T2-weighted (2500/80) (B) MR images demonstrate hyperintense left frontal convexity subacute subdural hematoma (large arrows in A and B). Right frontal convexity subacute hematoma (small arrows) is seen on the axial T2weighted image.

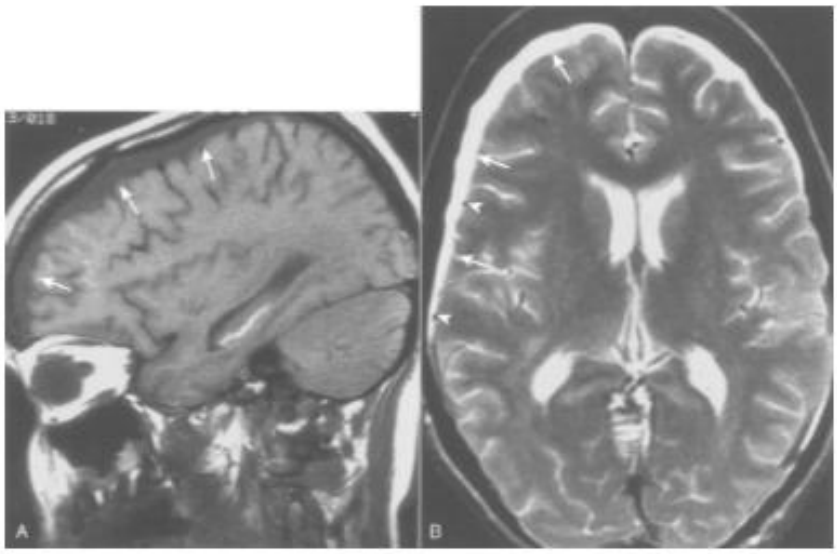

Figure 8: Right frontal convexity chronic subdural hematoma is slightly hyperintense on sagittal T1-weighted (500/15) (A) and isointense on axial T2-weighted (2500/80) (B) MR images relative to cerebrospinal fluid (arrows). Note displacement of the subdural veins away from inner table of the calvarium (arrowheads). A small left frontal convexity subdural hemorrhage is present.

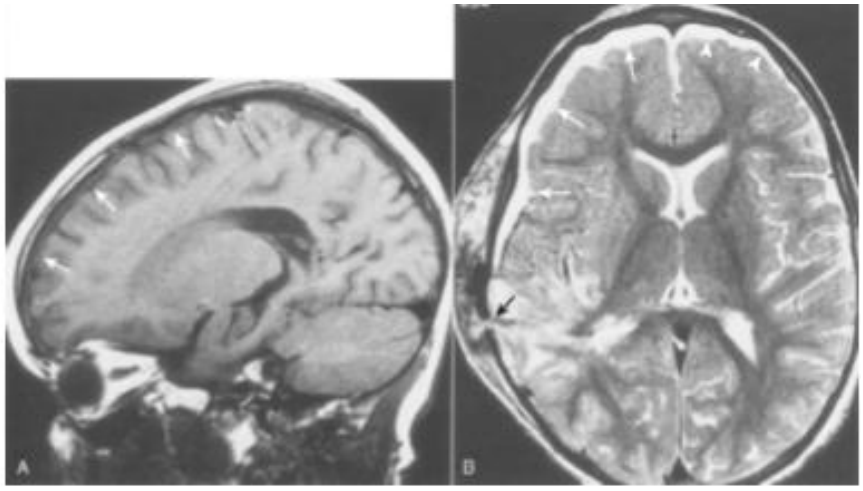

Figure 9: Same case as in Figure 6. Sagittal T1-weighted (500/15) (A) and axial FSE T2weighted (3500/108) (B) MR images show cerebrospinal fluid hygroma over right hemispheric convexity (arrows). The small left frontal convexity hygroma not evident on the CT scan is well visualized on the MR image (arrowheads). Note the right temporal lobe contusion and herniation of the temporal lobe through the temporal bone fracture (black arrow).

It is not always possible to distinguish a subdural hygroma from atrophy because the findings of mass effect and sulcal effacement associated with subdural hygroma may be subtle. Visualization of cortical veins adjacent to displaced cortex is indicative of subdural hygroma. A diagnosis of atrophy is made when cortical veins are seen traversing a widened subarachnoid space[17].

CONCLUSIONS

Imaging plays a central role in the diagnosis and management of subdural hematoma. CT and MRI are preferred mostly in imaging. Both are best modality in establishing the diagnosis of different lesions of brain.

REFERENCES

1. Gennarelli TA, Thibault LE: Biomechanics of acute subdural hematoma. J Trauma 1982; 22:680-685.
2. Rengachary SS, Szymanski DC: Subdural hematomas of arterial origin. Neurosurgery 1981; 8:166171.
3. Dublin B, French BN, Rennick JM: Computer tomography in head trauma. Radiology 1977; 122:365369.
4. Dolinskas CA, Zimmerman RA, Bilaniuk LT, Gennarelli TA: Computed tomography of post-traumatic extracerebral hematomas: Comparison to pathophysiology and responses to therapy. J Trauma 1979; 19:163-169.
5. Zimmerman RA, Bilaniuk LT, Gennarelli T, et al: Cranial computed tomography in diagnosis and management of acute head trauma. AJR Am J Roentgenol 1978; 131:27-34.
6. Smith Jr WP, Batnitzky S, Rengachary SS: Acute isodense subdural hematomas: a problem in anemic patients. AJNR Am J Neuroradiol 1981; 2:37-40.
7. Bergstrom AS, Ericson K, Levander B, et al: Computed tomography of cranial subdural and epidural hematomas: Variation of attenuation related to time and clinical events such as rebleeding. J Comput Assist Tomogr 1977; 1:449-455.
8. Forbes GS, Sheedy PF, Piepgras DG, Houser OW: Computed tomography in the evaluation of subdural hematomas. Radiology 1978; 126:143-148.
9. Reed D, Robertson WD, Graeb DA, et al: Acute subdural hematomas: Atypical CT findings. AJNR Am J Neuroradiol 1986; 7:417-421.
10. Cameron M: Chronic subdural haematoma: A review of 114 cases. J Neurol Neurosurg Psychiatry 1978; 41:834-839.
11. Forbes GS, Sheedy PF, Piepgras DG, Houser OW: Computed tomography in the evaluation of subdural hematomas. Radiology 1978; 126:143-148.
12. . Karasawa H, Tomira S, Suzuki S: Chronic subdural hematoma: Time-density curve and iodine concentration in enhanced CT. Neuroradiology 1987; 29:36-39.
13. Scotti G, Terbrugge K, Melancon D, Belanger G: Evaluation of the age of subdural hematomas by

computerized tomography. J Neurosurg 1977; 47:311-315.

14. Karasawa H, Tomira S, Suzuki S: Chronic subdural hematoma: Time-density curve and iodine concentration in enhanced CT. Neuroradiology 1987; 29:36-39.
15. Hoff J, Bates E, Barnes B, et al: Traumatic subdural hygroma. J Trauma 1973; 13:870-876.
16. Fobben ES, Grossman RI, Atlas SW, et al: MR characteristics of subdural hematomas and hygromas at 1.5 T. AJNR Am J Neuroradiol 1989; 10:687-693.
17. Kon AH, LaRoque RL: Evaluation of head trauma by computed tomography. Radiology 1977; 123:345350.