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CT evaluation of intracranial subdural haematoma: an Accra experience

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Summary
This is a descriptive analysis of the Computed Tomographic (CT) findings in 50 cases of intracranial subdural haematoma in Accra. The majority of patients were adults, though no age group was immuned. The frequent aetiological factor in the series was trauma. Other remote causes such as meningitis and sickle cell disease were reported. The male to female ratio was 2.6:1, while the mean age was 32.4 years. Subdural Haematoma (SDH) was classified into various subtypes by their CT densities. The commonest type, the hyperdense or acute haematoma was reported in 56% of the patients. CT features noted in the series, include ventricular alteration, seen in 31 (62%) and were more often noted in the acute and isodense bleeds. Ipsilateral effacement of cerebral sulci was observed in only 10% of cases. Concave haematoma borders were reported in 76%. SDH were more often found on the left aspect of the cranium (52%), particularly in the frontal and pariental lobes. Evacuated surgery was done in only 9 of the series, where haematoma was demonstrated in more than 3 tomographic slices. Flake-like calcifications were found in three cases of long standing haematoma. Against the above background CT can be described as an appropriate diagnostic tool in clinical evaluation of SDH.

Keywords: *Subdural, haematoma, hyperdense, isodense and hypodense*

Résumé
C'est une analyse descriptive de la tomographie informatique des données de 50 cas d' hematome intra-craniea subdural á Accra. La majorité des patients etait des adultes malgre le pait que an cum group d'age n'etait immunize. Le facteur actologique frequent dans ces seues etait les accidents. La meningite et la drepanocytose etaient des causes pas conrrantes. La proportion male/ female etait 2.6:1 et al moyenne d'age etait de 32.4 ans. L'hematome subdural (HSD) etait classifiée en different sous-types en function de la densité tomographique-informatisée. Le type plus commun, l'hyperdense hematoma severe, avait ete reportée chez 56% des patients les characteristiques des valeurs tomographiques notes dans les series étaient l'alteration ventriculaire qui apparut chez 31 (62%) des cas et la plus spouvest chez ceux avec une hemorragie isodense severe. L'inflammation ipsilateral du sulci cerebral etait present a 10% seulement et (hematome des bordures concave chez 6% des patients. La HSD était le plus souvent demontre a l'aspect gauches du crane (52%) particulierement dans les lobes frontal at porietal. La chirugie d'evacuation avait ete faite seulement sur a patients dans les series, avec l'hematome demontre sur plus de 3 larres tomographiques. Des gocittes de calcification etaient trouvés sur 3 cas d'hematome chronique. Par rapport aux observations GI dessus, hematome tomographique informatisees peuvent etre decrit comme un ontil didiagnostique approprié dans l'evaluation clinique del'hematome subdural.

Introduction

Whereas CT has become a valuable diagnostic tool in the evaluation of various neurological conditions including subdural haematoma (SDH) especially in developed countries [1-6], SDH is still largely diagnosed in most of the West African sub-region by clinical evaluation. This is mainly due to the lack of facilities required for brain imaging in most parts of these countries. Records show a dearth of literature on SDH in Ghana, but a clinico-pathological study in Nigeria before the advent of CT reported only 25 cases of SDH in a period of $9^1/_2$ years[7]. This not only represents gross underdiagnosis among the group studied but also illustrates the inadequacy of the available diagnotic tool. This report describes the ease of diagnosis of SDH in 50 consecutive cases and also reviews the CT characteristics of this extra parenchymal haemorrhage.

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Materials and methods

The films and reports of fifty brain CT Scan examinations, radiologically diagnosed as subdural haematomas during an 8 month period (October 99 – May 2000) were reviewed. All scans were done at the Korle Bu Teaching Hospital Accra and patients were examined using a Philips CSQ Scanner. Pre and post contrast slices were done, except in cases of acute trauma or cardiovascular accident (CVA) when only non contrast slices were obtained. This is to avoid further complications from contrast administration in these categories of patients. Sixty mls of intravenous urrograffin was administered in the contrast studies.

Results

There were 36 males and 14 females giving a male/female ratio of 2.6:1. The mean age was 32.4 years

Table 1: This is a table of age distribution and the various causes of subdural haematoma. It shows that no age group was immuned to SDH and no definite peak age incidence was observed. The commonest cause of subdural haematoma was trauma which occurred in all age groups. This was followed by patients with Cerebrovascular accidents (CVA). The miscellaneous category was made up of 3 patients with sickle cell disease and 1 with meningitis.

Table 1: Age distribution and cause of subdural haematoma

Age	Trauma	CVA	Post craniectomy	Miscella-neous	Total
<9 yrs	3	-	-	2	5
10-19	3	-	-	1	4
20-29	5	-	1	-	6
30-39	5	-	1	-	6
40-49	6	-	1	1	8
50-59	2	3	-	-	5
60-69	-	4	1	-	5
70 yrs	2	3	2	-	7
Total	26	10	6	4	46

Ages were not stated in 4 patients involved in trauma
CVA -Cardiovascular Accident

Table 2: This illustrates the relationship between causative factor and the side of haematoma as demonstrated by CT SDH resulting from trauma and the miscellaneous category were found in both sides of the vault with an approximately equal incidence. In patients with CVA and previous craniectomy left sided SDH were more common. Generally, left sided subdural haematoma were the commonest (52%). Bilateral haematoma were however reported in cases of trauma and CVA with a total incidence of 12%.

Table 2: Subdural haematoma, causative factor and side of haematoma as shown by computed tomography.

Causative factor	Unilateral Left		Right		Bilateral		Total	
	No	%	No.	%	No	%	No	%
Trauma	13	(26)	12	(24)	5	(10)	30	(60)
CVA	7	(14)	2	(4)	1	(2)	10	(20)
Post Craniectomy	4	(8)	2	(4)	-	-	6	(12)
Miscellaneous	2	(4)	2	(4)	-	-	4	(8)
Total	26	(52)	18	(39)	6	(12)	50	(100)

Table 3: This describes the various CT findings in the 50 cases of subdural haematoma.

Table 3: Computed tomographic findings in 50 cases of subdural haematoma

CT findings	No.	%
Density of haematoma		
a. Hyperdense (acute)	28	56
b. Hypodense (chronic)	13	26
c. Isodense (subacute)	7	14
d. Hypodense on a hyperdense layer (acute on chronic)	2	4
Density (attenuation) of adjacent brain parenchyma		
a. Normal density	40	80
b. Hyperdense	10	20
Margins of Haematoma		
a. Concave	38	76
b. Straight	12	24
Venticular alteration	31	62
Ipsilateral effacement of cerebral sulci over the converxity of the brain	5	10
Calcification	3	6

Density (attenuation co-efficient)
Hyperdense haematoma demonstrating attenuation coefficients higher than that of brain parenchyma with CT (Hounsfield) units ranging from 40-65 were reported in 28 (56%). Isodense bleeds with density equal to that of brain parenchyma and CT units ranging from 25-40 were found in only 7(14%) of patients. The third category of haematoma which were hypodense had CT units ranging from 15-25 and they occurred in 13(26%) of patients.

Ventricular alteration
Ventricular alterations by haematoma was found in 62%. These were seen as displacement, deformity, compression and/or collapse usually in the lateral ventricles particularly the body. While 95% of the hyperdense (acute) SDH and 50% of the isodense (subacute variety) demonstrated dominant ventricular changes, only two of the hypodense haematoma showed the above characteristics.

Harmatoma margin
The margins of the outlined haematoma were most often concave in relation to the adjacent brain 38 (76%). The remaining 24% showed straight margins against the brain parenchyma.

Density of adjacent brain
In 80% of the cases there was normal attenuation of the brain tissue adjacent to the haematoma while only 12% demonstrated higher attenuation of the adjacent brain. However, none of the cases demonstrated low attenuation.

Contrast enhancement
Intravenous contrast medium was administered only to the 8 postoperative cases. An edge enhancement was the common pattern observed in six of the patients.

Effacement of ipsilateral cerebral sulci
This was noted only in the 5 patients with isodense haematoma Table 4: shows the site and incidence of SDH. Fronto-parieto-occipital haematomas were commonly observed 23(46%). Parietal SDH was reported in 15 (30%) while frontal was found in 10% of cases. From this table haematoma antecedent to surgery were usually frontal or fronto-parietal while those resulting from CVA were mostly parietal.

Table 4: Causative factor and relationship to site of subdural haematoma as demonstrated by CT.

Causative Factor	Site of Haematoma F	FPO	P	PO	O	TP
Post trauma	3	12	11	1	2	1
Post craniectomy	0	5	4	1	-	2
Post operative	2	6	-	-	-	-
Total	5	23	15	2	2	3
%	10	46	30	4	4	6

Key
O = Occipital
F = Frontal
FPO = Fronto0parieto-occipital
P = Parietal
PO = Parieto occipital
TP = Temporo-parietal

Other positive CT findings
A total of 28(56%) showed additional CT findings. Namely; vault fractures observed in 20% and vault defects from surgery in 14%. Intraventricular haematoma and intraparenchymal haematoma in 8% and 12% respectively. Hydrocephalus was noted in only 2%.

Discussion
Odeku [7] in his report described the various neurodiagnostic aids available for the evaluation of SDH before the advent of CT in Nigeria. These imaging modalities are not only invasive

but lack specificity, sensitivity and promptness [8], which must have accounted for an underdiagnosis of 25 cases in a 9' year period among the population studied. CT diagnosis of 50 cases in only 8 months at the Korle Bu Teaching Hospital suggest, improved sensitivity and specificity of this invaluable neuroimaging modality [8]. The ease of diagnosis is due to the differential attenuation coefficient of subdural collection when compared to that of the adjacent brain tissue especially in the hyper and hypodense varieties.

Classification of the subtypes of SDH is dependent on the density demonstrated by CT, which is also an indication of the age of the haematoma. Hyperdense collections, described as acute bleeds occur in an interval of 0-7 days (fig 1) [9]. Isodense or sub acute haematomas are usually 7-22 days old, while hypodense or chronic bleeds are more than 3 weeks old (fig 2) [9]. This change in density occurs as the blood in the subdural space undergoes resorbtion, reducing the density of haematoma and making it indistinguishable from the brain tissue (isodense phase) [6,9,10]. Eventually it becomes lower in density than the cerebral parenchyma (hypodense variety).

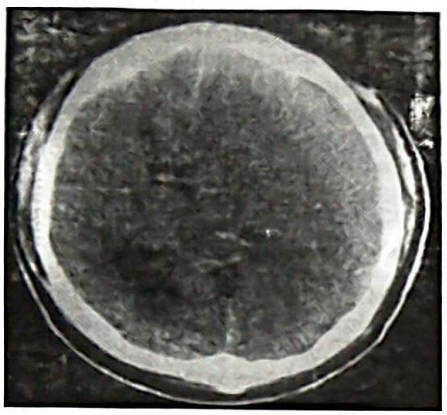

Fig. 1: Left hyperdense subdural haematoma in a non-contrast enhanced CT (NCECT) there is associated compression and [illegible] ipsilateral ventricle.

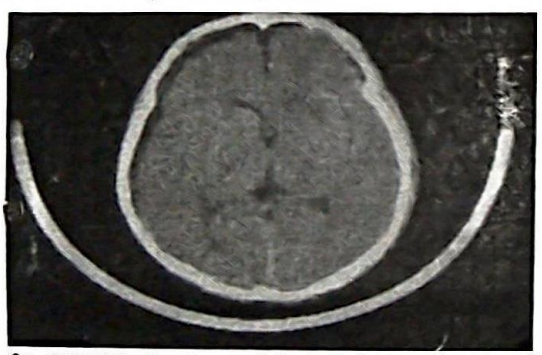

Fig. 2: NCECT showing a right hypodense, chronic subdural haematoma. Note the concave haematoma border adjacent to the brain parenchyma.

In our series hyperdense haematoma were mostly reported (56%) and were commonly seen as a sequeale to trauma, when they are normally produced by tearing of veins crossing the subdural space. The hypodense variety was found to be more common amongst stroke patients. Reasons can be traced to the timing of CT scan post trauma or ictus [11]. It is sometimes practically impossible to identify isodense haematoma because of the loss of the usual silhouette, between the bleed and the brain parenchyma [12] This must have been responsible for their low detection in our series (Fig. 3). However, secondary signs such as ventricular alteration and effacement of the ipsilateral sulci were often helpful criteria in such equivocal situations [12]

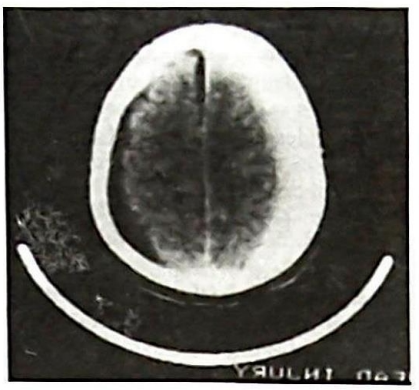

Fig. 3: NCECT showing bilateral subdural haematoma at different phases of resolution. Note the hyperdense or acute left haematoma and the older hypodense right haematoma. Both show concave haematoma borders.

Generally, haematoma were more on the left side of the cranium (52%). However of the six bilateral SDH only one patient demonstrated varying stages of resolution of haematoma. This implies a recent insult on the contralateral side of the old bleed (fig3). Two cases of acute on chronic haematomas were also reported amongst our CVA patients. They were recognised by a fluid/fluid level with the area of increased attenuation below the well delineated old hypodense collection. This represents a rebleed into the old haematoma.

Most of the haematomas showed concave margins adjacent to the brain. This is because haematomas are venous bleed which can spread freely in the subdural space consequently assuming the concavity of the bony vault. The density of the adjacent brain was also dependent on a concomitant insult of the brain parenchyma. In cases of cerebral contusion or haemorrhage, the adjacent brain was hyperdense as seen in only 20% of our cases. The rest of the series however showed normal brain density indicating non-parenchymal involvement of the insult.

Ventricular alteration was helpful in the diagnosis of SDH of the isodence variety where diagnosis would otherwise have been missed. Expectedly the degree of compression or collapse was proportional to the size of the haematoma. A shift of the midline was also noted with ventricular displacement. We observed that subdural haematomas were visualized on 1 to 4 adjacent tomographic slices. C T was useful in taking a decision between surgical evacuation and conservative management of SDH. In this series, surgery was performed on patients with collections seen on three or more slices especially in cases with associated ventricular alteration. Similar to a report by Krichett *et al* [4] and Scotti *et al* [8]. Only 9 (18%) of our cases had evacuative surgery done.

In favour of CT scan as a suitable investigative tool was the additional diagnosis of fractures, intraparenchymal and intraventicular haematomas as well as hydrocephalus. These coexisting findings were dependent on the causative factor of

the subdural bleed. SDH was associated with intraventricular bleeds in 5(50%) of the CVA patients.

Even though six of our cases had contrast studies intravenous enhancement was not remarkable. Those that enhanced contrast medium demonstrated a fairly regular edge enhancement as in previous reports [5,6,9] Long standing SDH also calcify. Flake like calcifications close to the vault were seen in 3 (6%) of our patients with longstanding haematomas as described by Bradshaw [9]

Motion artifarcts in extreme instances especially the very ill patients, degraded the quality of CT images. In such patients high reliance was placed on the presence of secondary signs as previously described. Surgical metalic clips also produced artifarcts in the scans which obscured subdural collections in some slices.

Conclusion

Our study has shown the ease of diagnosis of SDH by Computed Tomography, particularly in patients with head injury, CVA and those who had just undergone craniectomy. It has also affirmed the occurrence of these haematomas in all age groups. The density of the haematomas was found to be a useful criteria in the classification and age estimation of the bleeds. The number of tomographic slices through which the haematoma was demonstrated, was a useful parameter for evacuative surgery. Against this background CT can be described as a gold standard for prompt, non-invasive diagnosis of this extra parenchymal haematoma. The ability to evaluate the skull vault and brain parenchynea in the same slices are also added advantage of CT in the diagnosis of SDH.

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