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ACQUIRED BRAIN HERNIATIONS: ACCOUNTING OF BRAIN COMPUTED TOMOGRAMS IN A NIGERIAN TERTIARY HOSPITAL
Felix U. Uduma (MBBCh, FWACS), Associate Professor, Department of Radiology, Faculty of Clinical Sciences, University of Uyo, P.M.B 1017, Uyo, Nigeria. Ubong U. Ekpene (MBBCh, FWACS), Lecturer, Neuro-surgical Unit, Department of Surgery, Faculty of Clinical Sciences, University of Uyo, P.M.B 1017, Uyo, Nigeria. Ifeoma J. Okoye (MBBS, FMCR), Professor, Department of Radiation Medicine, Faculty of Medical Sciences, University of Nigeria, P.M.B. 01129, Enugu Nigeria.

Corresponding author: Dr Felix U. Uduma, Associate Professor, Department of Radiology, Faculty of Clinical Sciences, University of Uyo, Uyo, Nigeria. E-mail: felixuduma@yahoo.com

ACQUIRED BRAIN HERNIATIONS: ACCOUNTING OF BRAIN COMPUTED TOMOGRAMS IN A NIGERIAN TERTIARY HOSPITAL

F. U. Uduma, U. U. Ekpene and I. J. Okoye

ABSTRACT

Background: **Acquired brain herniation (ABH) is mechanical displacement of a brain tissue from its normal anatomical location into an adjacent space. Four known subtypes are sub-falcine, trans-tentorial, tonsillar and transalar herniations. ABH is life threatening, thus demands prompt diagnosis and intervention as it compresses on vital brain structures.**
Objective: **To evaluate brain computed tomograms (CT) for the sub-types and causes of acquired brain herniations.**
Design: **A retrospective cross-sectional study**
Setting: **Department of Radiology, University of Uyo teaching hospital, Uyo, Nigeria.**
Material and methods: **Three hundred and ninety-six (396) brain CT from 14th November 2012 to 30th July 2019 were obtained from the archives of Department of Radiology, UUTH, Uyo.**
Results: **14.65% (n-58) of all patients had acquired brain herniations with age range of 12- 86 years, mean age of 50.14years and male predominance of 2.62: 1. Only two subtypes of herniations namely subfalcine (n-55, 94.83%) and transtentorial (n-3, 5.17% were observed).**
The causes of ABH were trauma (n-38, 65.52%), cerebrovascular accident (CVA) (n-18, 31.03%) and intracranial tumours (n-2, 3.45%). Majority of the patients in traumatic ABH were less than 50years whereas CVA and tumour were predominant above 50. The commonest causes of traumatic ABH in descending order were subdural haematoma (39.47%), intra-cerebral haematoma (28.95%), epidural haematoma (23.68%), cerebral contusion (21.05%) and subarachnoid haematoma (10.53%).
Conclusion: **The commonest aetiology and subtype of acquired brain herniations in Uyo, Nigeria are trauma and subfalcine herniation respectively. Traumatic causes are commoner in younger age with reversal to mainly cerebrovascular accident in older age.**

INTRODUCTION

Acquired brain herniation (ABH) is otherwise called cerebral herniation. It is defined as mechanical displacement of a portion of a brain tissue relative to an adjacent intracranial anatomic space (1,2). ABH is divided into subtypes like sub-falcine herniation, trans-tentorial herniation, tonsillar herniation and transalar (trans-sphenoidal) herniation. Trans-tentorial herniation is further sub classified into central [ascending and descending] and lateral [anterior and posterior] (1,3,4).

This sub-classification of ABH is anchored on the anatomic space through which the brain herniates. Subfalcine herniation is a herniation beneath the anterior component of the falx cerebri that separates both cerebral hemispheres. Ipsilateral cingulate gyrus by virtue of its anatomic position remains the commonest and earliest brain structure that herniates in subfalcine herniation (3). On the other hand, trans-tentorial herniation is herniation through the incissura or notch that exists between the two-tentorium cerebelli. This tentorium separates the supratentorial fossa from the infratentorial fossa. Foramen magnum is a large orifice in the floor of the posterior cranial fossa that permits the exit of spinal cord, nerves and blood vessels (4). Herniation through this foramen is called tonsillar herniation. Whereas transalar herniation is herniation around the middle cranial fossa across the greater sphenoid wing.

The underlying factor in many aetiologies of ABH is raised intracranial pressure. These aetiologies include intracranial haematoma like (subdural, epidural, subarachnoid and intra-parenchymal), cerebral edema, cerebral contusion, cerebrovacular accidents, cerebral venous thrombosis, intracranial neoplasm, pneumocephalus, tension arachnoid cyst, meningitis, brain abscess, hydatid cyst, empyaema and metabolic-hepatic encephalopathy [see figures 4,5,6] (1,4,6-10). Others are spontaneous intracranial hypotension and lumbar puncture.

Acquired brain herniation causes squeezing of the brain parenchyma, blood vessels, nerves, cisterns and ventricles. The consequences will be severe neurological morbidities like infarction, nerve palsy, hydrocephalus and sometimes death if not relieved. Specifically, each subtype of ABH will demonstrate peculiar regional brain infarction based on vascular territorial compression with its attendant ischaemic stroke. For example, pericallosal and callosomarginal arteries of anterior cerebral artery are compressed in subfalcine herniation, leading to corresponding motor homunculus infarction with consequent lower limb weakness (9). Descending (uncal) trans-tentorial herniation is known to cause medial temporal lobe and hypocampal infarctions from middle cerebral artery stenosis (1,3). Uncal herniation can also compress calcarine branch of the posterior cerebral artery leading to occipital infarction (3). Whereas ascending transtentorial herniation compresses posterior cerebral or superior cerebellar arteries leading to occipital and cerebellar infarctions (3). In tonsillar herniation, compression of the posterior inferior cerebellar arteries, vertebral arteries and anterior spinal artery, lead to ischemia of brainstem, tonsils and lower cerebellum (3,11).

These adverse outcomes of herniations merit evaluations of the frequency of the different subtypes. This is especially so when one takes into cognizance the paucity of local data on this critical condition. Since computed tomogram (CT) is among the available neuroimaging armamentarium in our centre, a tertiary hospital, it becomes pertinent to retrospectively evaluate our archival data on this significant entity. Though CT and MRI are the imaging modalities of choice used for establishing correct diagnosis and guiding therapeutic decisions of cerebral herniations, the preferred imaging modality is CT due to its short acquisition time (1). Moreover, the critical nature of this condition, demands prompt diagnosis and intervention. Therefore, we consider this study is designed to evaluate brain computed tomograms to ascertain the commonest subtypes and causes of acquired brain herniations.

PATIENTS AND METHODS

A retrospective study of brain computed tomograms from 14th November 2012 to 30th July 2019. All the brain computed tomographic results archived in the Department of Radiology, University of Uyo teaching hospital (UUTH), Uyo, Akwa Ibom State, Nigeria for the period under review were collated and evaluated.

The CT examinations were done with Toshiba Activion 16 helical CT machine, manufactured in Japan in December 2011. Axial non-contrast scans were done from the base of the skull to the vertex using 3 to 5mm slice tissue thickness. Contrast examinations were also added when indicated. Images were reformatted into sagital and coronal planes. Volume rendered 3D images, which aid in the detection of subtle intracranial haemorrhages and fractures were also employed.

CT reports with acquired brain herniations in the body of the text or in the conclusion were separated for evaluations. The demographic data and clinical history were documented. The type of herniation causes of herniation, degree of herniation, localizations of the aetiologies of herniation, associations with the herniations like calvarial fractures were evaluated. Patients with significant shift but without frank brain herniation as well as reports without demographic documentations were excluded. Data analysis was done with Statistical package for social studies (SSPS, 13.0, 2011).

Ethical approval with reference number UUTH/AD/S/96/VOL.XXI/380 was obtained from the institutional health research ethical committee.

RESULTS

396 brain computed tomograms were evaluated in the study period. 58 patients (14.65%) had acquired intracranial herniations with male to female ratio of 2.62: 1. The youngest age was 12 years while the oldest age was 86 years. The mean age was 50.14years. The commonest age affected was 50-69years with the highest frequency among males and females being 50-59years and 60-69years respectively.

No type of herniation was seen in the first decade of life in this study. The frequency of herniations among males increased from 3rd decade to the 5th decade and subsequently decreased to the 7th decade.

The commonest types of acquired brain herniations were subfalcine herniation (n-55, 94.83%) and descending transtentorial (uncal) herniation (n-3, 5.17%). The commonest age ranges for subfalcine herniations among males were 50-59years (n-10, 18.18%) and 60-69years (n-10, 18.18%). But it is commonest in the 60-69years (n-7, 12.72%) in females. The least cases were seen in the 70-79 years in both sexes with 1.82% in each. The commonest causes of acquired brain herniations were trauma (n-38, 65.52%), cerebrovascular accident (CVA) (n-18, 31.03%) and intracranial tumours (n-2, 3.45%). Of the traumatic causes, the commonest aetiology was a combination of lesions. But in terms of single entity, the commonest causes in descending order were subdural haemotoma (39.47%), intra-cerebral haematoma (28.95%), epidural haematoma (23.68%), cerebral contusion (21.05%) and subarachnoid haematoma (10.53%).

Only 13 cases (22.41%) of all herniations were quantified. On this basis, we adopted a grading formula where any herniation that is less than 5mm is regarded as mild herniation, moderate when the herniation is from 5mm to less than 10mm and severe herniation when the shift is from 10mm and above. Mild herniations were 4 in number (30.77%), moderate (n-1, 7.69%) and severe (n-8, 61.54%). All the severe cases were caused by trauma and they were accompanied by calvarial fractures in 37.50% of cases. Herniation to the right cerebral hemisphere (n-46, 79.31%) was commoner than herniation to the left (n-12, 20.69%).

In traumatic acquired brain herniations, majority of the patients (57.89%) were less than 50years. Whereas in acquired brain herniations due to CVA and intracranial tumours, 90.00% were older than 50years. Majority of the intracranial haematoma in traumatic acquired brain herniation affected many lobes. The frontal and parietal lobes were involved at same time in 51.72% of cases. In the age ranges of 70-79years and 80-89years, all the causes of ABH were either due to cerebrovascular accident (83.33%) or intracranial tumour (16.67%).

Table 1

Study population showing age and sex distribution with causes of acquired brain herniations. (M-Males, F-Females, ICH-Intracranial haematoma, CONT-cerebral contusion, EPID- -epidural haematoma, SUBD - subdural haematoma, SUB-A- Subarachnoid haemorrhage, TUM-Intracranial tumour, SUPRA-T-supra-tentorial fossal tumour, INFRA-T-Infra-tentorial fossal tumour, CVA-Cerebrovascular accident, HGIC-haemorrhagic CVA, ISH-Ischaemic CVA)

			ICH	**ICH**	**ICH**	**ICH**	**ICH**	**TUM**	**TUM**	**CVA**	**CVA**
AGE RANG	**M**	**F**	**INT-CE**	**CO NT**	**EPID**	**SUBD**	**SUB-A**	**SUPRA-T**	**INFRA-T**	**HGIC**	**ISH**
0-9	0	0	0	0	0	0	0	0	0	0	0
10-19	1	2	0	3	1	0	0	0	0	0	0
20-29	6	0	2	0	3	2	0	0	0	0	0
30-39	4	3	3	1	2	2	1	0	0	0	0
40-49	6	0	3	0	0	4	0	0	0	0	0
50-59	12	1	2	3	0	2	2	1	0	3	1
60-69	11	7	1	0	3	5	1	0	0	4	3
70-79	2	1	0	0	0	0	0	1	0	2	1
80-89	0	2	0	0	0	0	0	0	0	2	0
TOTAL	42	16	11	8	9	15	4	2	0	13	5

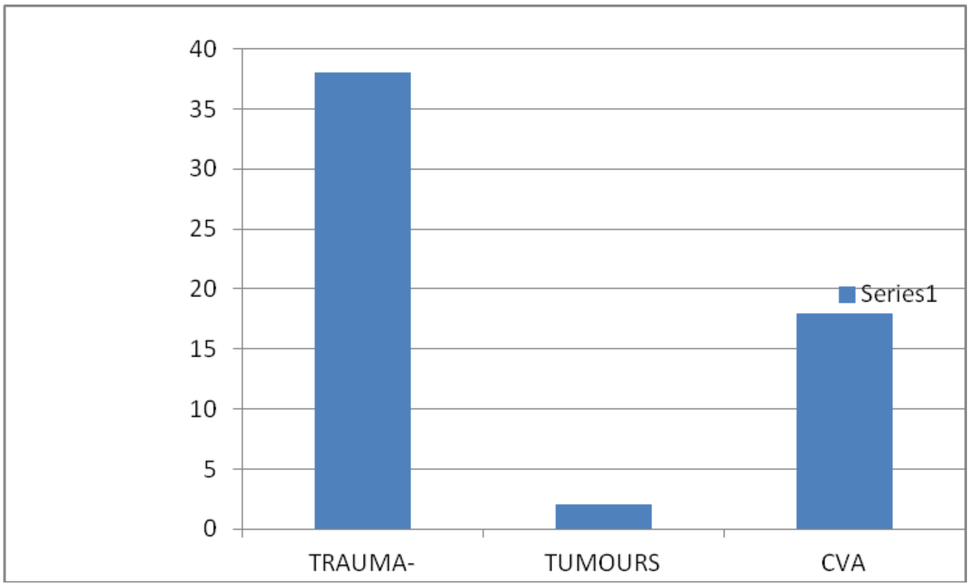


Figure 1-Bar chart showing the different causes of acquired brain herniation.

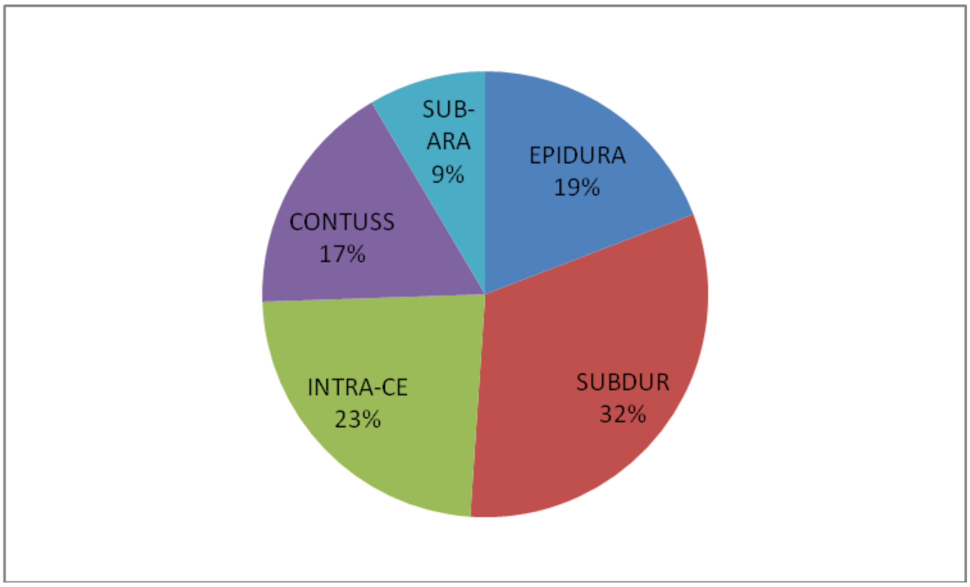


Figure 2-Pie-chart showing the percentages of different aetiological factors in the traumatic causes of acquired brain herniations.

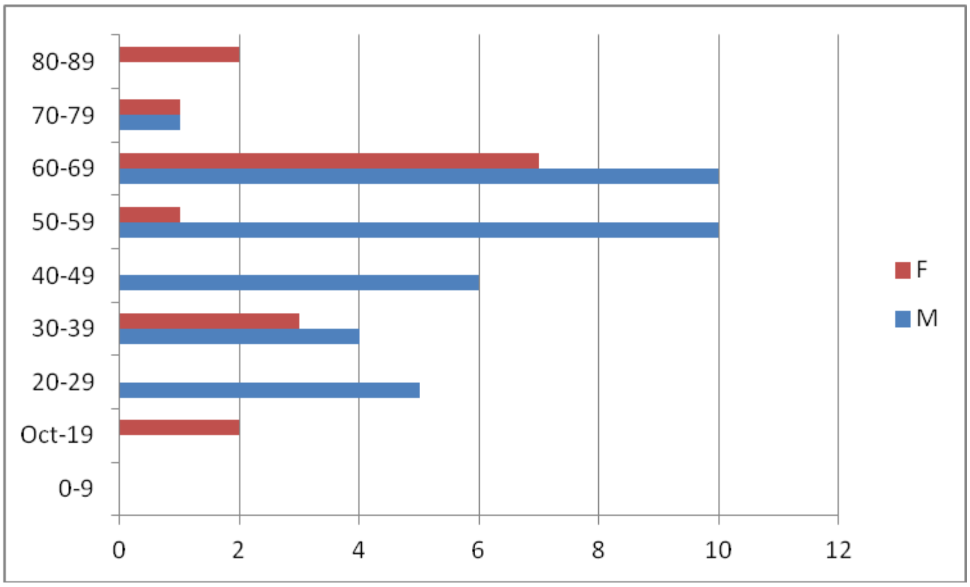


Figure 3-Age and sex distributions of subfalcine herniations

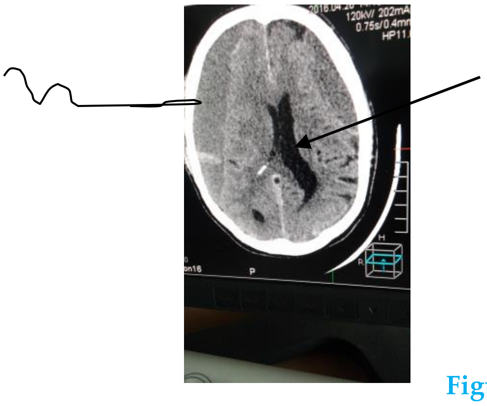


Figure 4- Axial brain CT at the level of lateral ventricles showing subfalcine herniation(straight arrow) to the left due to bilateral subdural haematoma (curved arrow) with right worse than left.

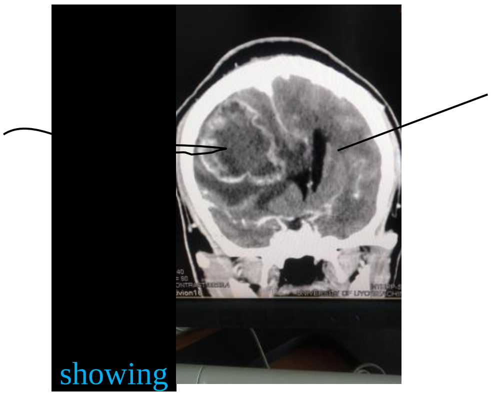
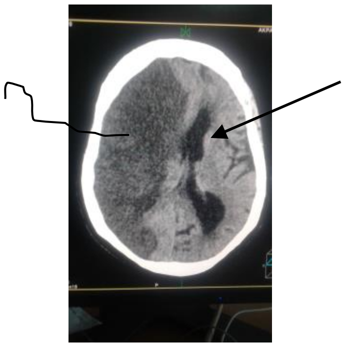


Figure 5-Coronal enhanced brain CT showing ring enhancing intra-cerebral Glioblastoma multiformis (curved arrow) in the right cerebral hemisphere with subfalcine herniation(straight arrow) to the left.



Fihure 6-Axial CT with right hemispheric hypodense ischaemic infarct (curved arrow) with subfalcine herniation (straight arrow) to the left.

DISCUSSION

This retrospective observational study has highlighted subfalcine herniation as the most frequent subtype of acquired brain herniations in this study. This may not be unconnected with the larger anatomic space left beneath the dural reflections of the anterior falx cerebri, thus creating potential capacious window for any shift. As such adjacent brain structures like cingulate gyrus, peri-calloasal cistern, peri-callosal arterial branch of anterior cerebral artery, ipsilateral frontal lobe and ipsilateral lateral ventricle can easily shift through this space to the contralateral side [see figures 4,5,&6]. The neurological sequelae will be regional brain infarction, hydrocephalus, motor dysfunction, impaired memory and even loss of consciousness. Nevertheless, our observation of subfalcine herniation as the most frequent herniation (94.83%) is at variance with the findings of Probst et al who discovered transtentorial herniation (49%) as the most frequent (12). However, subfalcine herniation still narrowly followed as their second commonest with 42% unlike the wide disparity between the two entities in our study. No type of herniation was seen in the first decade of life in this study [see table 1]. This is not astonishing as brain herniation is rare among neonates because of the presence of open fontanelles and cranial sutures that allow for any intra-cranial expansion (13).

It is noteworthy that we recorded low frequency of transtentorial (uncal) herniation and no tonsilar herniation. This is because of the grave clinical conditions associated with such herniations (14). Transtentorial herniation causes ipsilateral pupil dilatation (third cranial nerve compression) and obstructive hydrocephalus (aqueduct of Sylvius compression) (1,3). Trans-tentorial herniations can also cause Duret's haemorrhages and diplegia. Duret's haemorrhage is bleed in the midbrain/upper pons resulting from stretching and tearing of small vessel perforators (15). This haemorrhage has frequently been associated with prolonged consciousness disorders and devastating functional disability (15). Diplegia is however due to Kernohan notch phenomenon. This phenomenon is defined as ipsilateral hemiplegia caused by compression of the contralateral cerebral peduncle against the tentorial edge (3,16). It means that direct compression of the ipsilateral cerebral peduncle by the uncus and the contralateral ones against the tentorial edge will produce diplegia (13). Worsening trans-tentorial herniation, will translate to conversion of abnormal flexor posturing to abnormal extensor response (decorticate and decerebrate posturing) through the involvement of rubrospinal and vestibulospinal tracts from brain stem compression (3). Further compression of the reticular activating system will change consciousness level whereas medullary compression will lead to moribund state (3). Parinaud syndrome along with features of diabetes insipidus arising from injuries to pituitary stalk will accompany ascending transtentorial herniation (3).

On the other hand, tonsillar herniation is known to affect the cardiac and respiratory centres in the medulla oblongata (3). This will lead to Cushing triad of hypertension, bradycardia and hypoxia (3). Sometimes Cheyne-stokes breathing with stages of hyperventilation, ataxia and finally apnoea may occur (3). The endpoints may be vegetative state or even death.

Notably, the most observed instigating aetiology in this study is trauma [see figure 1]. This explains the involvement of younger age groups and male predominance as they are the most itinerant being their family bread winners. This also rationalizes the increasing incidence of traumatic ABH from the 3rd to 5th decade of life. In contrast, waning of mobility at older age reinforces the somersault of the aetiology of ABH to CVA and intracranial tumours in the 7th to 8th decade of life with bias towards CVA [see figure 3].

When trauma is the cause of ABH, combined lesions like intra-axial and extra-axial lesions account for most causes. This explains the observed lesions outnumbering the number of patients. But as a single entity lesion, the commonest cause of traumatic ABH is subdural haemorrhage as seen in another study [see figure 2] (12). Subdural haemorrhage is sometimes known to co-exist with unidentifiable accompanying brain lesions like brain contusion and diffuse axonal injury (17.18). This adds to morbidity, thus producing incongruent imaging and clinical findings. The observed descending order of this single entity lesion as a cause of traumatic sub-falcine herniation in this study are subdural haematoma, intra-cerebral haematoma, epidural haematoma, cerebral contusion and subarachnoid haematoma.

The prevalence of ABH in our tertiary hospital is 14.65%. This may not all be encompassing as some cases of mild herniation may have escaped imaging when the patient was triaged and dismissed as a case of mild traumatic brain injury and considered to be outside the scope of imaging. The choice of quantitative grading system into mild, moderate and severe herniation as developed by the authors was computed based on the distance of shift from midline. This added objectivity and reproducibility to the study, thus creating room for comparable future research and diagnosis. But a pointer to the greater degree of impact on the head in severe ABH was the observed consistent calvarial fractures. This was seen localized to the frontal and parietal bones in subfalacine ABH in this study. This may suggest that the impact causing subfalacine herniation may be on the anterior cranial fossa.

The significance of this study is manifest when one considers grave neurological and vital centres damages that will result from delayed diagnosis of cerebral herniation and absence of early prompt interventions.

It is possible we may have under-counted or even over-counted the prevalence of acquired brain herniations. The under-counting could result from the fact that the patients with minor herniation may have been neurological stable and therefore suffer selection bias from the clinician based on pre-determined criteria. One in 1500 neurologically intact patients undergoing CT head imaging may have brain herniations (12). Besides, resource constraints may have prevented CT request compliance, thus limiting our sample population. Some may have been lost to death even before CT examinations, thus potentially decreasing our sample size.

Misclassification of significant shift as frank herniation may contribute to false positives, hence over-counting.

Retrospective single institution study which has been marred by incessant breakdown of the CT machine and poor archiving system also contributed to study limitations. These may account for the limited number of patients evaluated. Another compounding issue is that these are exhumed archived reports. The Radiologist were not mandated to thoroughly evaluate any existing brain herniation. Little wonder just above 20% of all reports carries any objectivity in terms of quantitative calculation of the level of shift.

CONCLUSION

The most frequent subtype of acquired brain herniations in Uyo, Nigeria is subfalcine herniation. Its commonest cause is traumatic brain injury with sub-dural haematoma being the single most frequent lesion. In descending order, lesions seen in traumatic causes of acquired brain herniations are subdural haematoma, intra-cerebral haematoma, epidural haematoma, cerebral contusion and subarachnoid haematoma. Traumatic aetiologies are commoner in earlier age with reversal to cerebrovascular accident and intra-cranial tumours at elderly age.

RECOMMENDATIONS

Proper archiving system of all radiological reports should be encouraged. Pre-determined quantitative criteria should be adopted for objective evaluation of cerebral herniations. A proposed example is the one adopted by the authors of this study.

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