

Causes of Delay in the Definitive Treatment of Compound Depressed Skull Fractures: A Five-Year Study from Nigeria

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Background: Calvarial fractures may be linear, depressed or ping-pong, and each can be compound (open) or simple (closed). When depressed fractures become compound, they cause contamination, resulting in intracranial sepsis. All depressed fractures with scalp breach are considered compound, whether or not the breach is contiguous with the fracture. They, therefore, become almost an absolute indication for operative treatment by elevation and debridement, to avert intracranial sepsis. Definitive treatment should be within 72 hours or else it would be unsafe to preserve the bone fragments.

Method: This was a retrospective study in which a review of the outcome from management of compound depressed skull fractures (CDSF) in the Institute of Neurological Sciences, University College Hospital, UCH, Ibadan Nigeria referral Centre for neurological diseases was undertaken from November 1997 to October 2002. Data was retrieved from ward, theatre and out-patient records and subsequently analyzed for the time interval between time of injury treatment and the cause of delays in surgery, if any.

Results: Of the 75 cases treated for CDSF with a Male: Female ratio of 11.5:1, only 54 had complete records and all were treated by elevation, debridement and craniectomy, resulting in cranial defects. There was delayed treatment in most of the cases at various stages from the time of injury, with most of them arising after presentation in our Centre.

Conclusion: Delayed treatment, was our major source of morbidity and most of the delay was traceable to our hospital processes. Scalp suturing before definitive treatment did not contribute to delay. Non-specialist care givers should be encouraged to refer patients without delay.

Introduction

Compound depressed skull fracture (CDSF) is a discontinuity in the skull in which one (or more) of the discontinuous edges is displaced below the inner table of the surrounding intact skull with a connection to the exterior, through the paranasal sinuses, the external auditory meatus or a scalp laceration. It constitutes about 80% of depressed skull fractures and 2.5% of head injuries, and is the most frequent reason for operative treatment in childhood head injury, in our Centre^{1,2,3,4}. Like all traumatic injuries especially to the head, more males than females are affected with about 70% of

cases diagnosed clinically⁵, requiring therefore the superior diagnostic sensitivity of computerized tomography, CT to reduce the incidence of false negatives⁶. Infection, from contamination at the time of trauma, is a major determinant of long term morbidity and neglected cases are a principal cause of intracranial abscess^{7,8}. Dural laceration is reported to be a major predictive factor for intra-axial sepsis and focal neurological deficits^{9,10}. It had been established in an earlier study, that closure of the scalp alone without adequate haemostasis and debridement, does not alter the infective sequelae from CDSF¹¹. Elevation and debridement done within 48hours,

historically, offers the best chance for an uneventful outcome, barring the severity of the parenchymal injury sustained from the trauma; recently however, a recent report advocated 72hours. A CDSF on its own, therefore, becomes a mandatory indication for operative treatment in most cases once the diagnosis is established. On rare occasions though, when the fracture directly involves a major dural venous sinus and elevation could precipitate life-threatening haemorrhage, conservative treatment has been exceptionally advocated^{12,13}.

In our own environment, CDSF has been reported as prone to neglect, with a protracted time lag as much as three weeks between injury and definitive treatment¹¹. This interval was reported as prolonged in patients whose wounds were sutured before definitive treatment. Why was the interval before definitive surgery longer in patients whose wounds were sutured? The previous study in our Centre had given three empirical reasons: (a) assumption that scalp wound closure was sufficient treatment; (b) expectation that scalp suturing would prevent intracranial sepsis; and (c) the fracture was simply not diagnosed.

In the present study, 10 years later, we evaluated the time lag before surgery, the reasons for pre-operative delay among the patients who did not get operated within 72hours, the points in the line of management where these delays occurred, the possible reasons for the neglect of CDSF in such cases including the influence of pre-operative scalp suturing on the timing of definitive treatment, and the impact of dural laceration on post-operative infection.

Patients and Methods

The 54 with complete records out of the 75cases (21 had incomplete records) treated for CDSF between November 1997 and October 2002, had their records reviewed. Essential data on each patient were collected using ward, theatre and out-patient records and subsequently, collated and analyzed. All

patients aged 15years or less were categorized as children, and above 15years as adults. Operating on a patient after 72hours following trauma (the maximum historical limit of reported uneventful replacement of elevated bone fragments), was regarded as delay.

Results

Of the 75 cases, 69(92%) were males, and 6(8%) were females, a male: female ratio of 11.5:1 (Table 1). Adults were 57 (76%) and children 18 (24%). Only 2 (2.67%) of the patients were aged above 60years (Table 1). Only 54 cases had complete records. Of these, road traffic accident accounted for 27 (50%) of the cases and was the most common aetiological factor, followed by assault 12 (22.2%), missiles 7(13%), domestic accidents 4(7.41%), industrial accident 2(3.7%), sport 1(1.85%) and fall 1(1.85%). Twenty-eight (51.9%) came with already sutured scalp wounds, and 26(48.1%) had unsutured wounds (Table 2). The affected regions of the skull were mainly frontal 30(55.6%) and parietal 16(29.6%). Dural lacerations accompanied 40(70.1%) of these fractures.

Fifty cases (92.6%) were referred from other medical facilities, and 4(7.4%) presented to us straight from the site of incident. The interval between trauma and presentation to our Centre in both groups ranged 5 hours - 27days (average 4.6days), and the mean trauma – surgery interval was 12.5days. A total of 45(83.3%) cases had delayed surgery (>72hours post-trauma), 43(79.6%) of whom were referred from other facilities. There were 14 different reasons for delay, 8 of which arose in our Centre, 5 occurred at the point of first medical contact, and 1 at the site of event (Figure 1). On the whole, however, the various reasons arose 87 times among the 45 delayed cases, some patients having as much as 4 reasons for their delay. A total of 45(83.3%) cases had delayed surgery out of which 25(46.3%) presented with already sutured scalp wounds (Table 2). Cranial

computerized tomography, CT was the imaging modality in 38(70.4%) cases. Seventeen patients (31.5%) had wound infections, pre-op 5, post-op 9, and combined pre-op/post-op 3; most of them (15) had delayed operations. The major complications were cranial defects 54(100%), wound sepsis 17(31.5%), post-

infective hydrocephalus 1(1.85%) and stitch granuloma 1(1.85%). Most 51(94.4%), made satisfactory recovery and 3(5.6%) died in the cause of treatment. Thirty-nine (72.2%) patients were followed-up after discharge for 1 – 56months (average 8.72months).

Table 1. Sex and Age Distribution in CDSF

Sex	Age (years)			No.
	≤15	≥15-60	>60	
Male	15	52	2	69
Female	3	3	0	6
Total	18	55	2	75

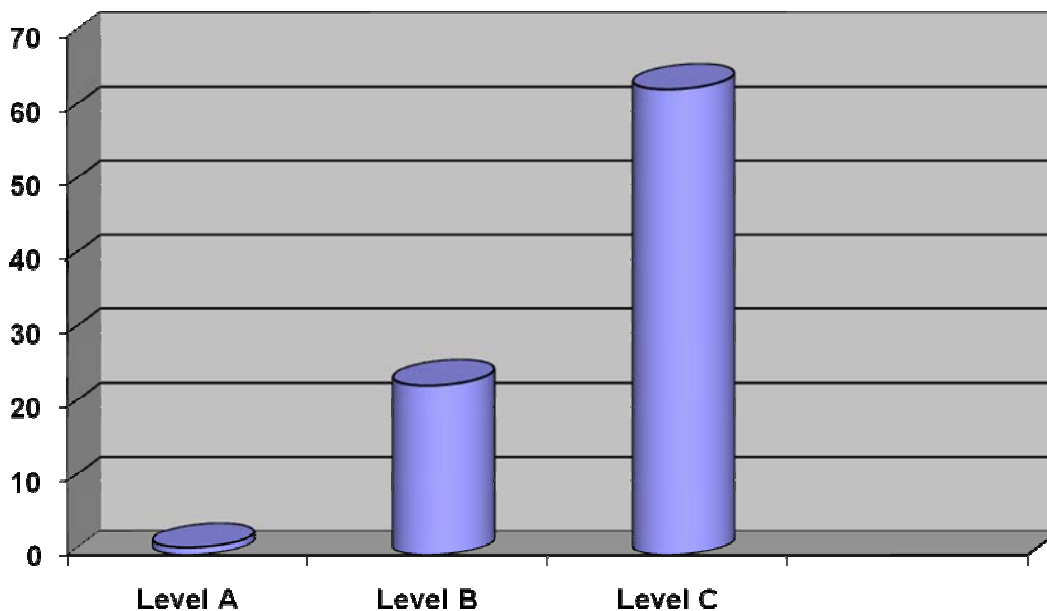


Figure 1. Delays at various levels of care – A (Site of incident), B (First health facility), and C (Our Centre)

Table 2. Presenting wounds in the infected and non-infected patients

Wounds	Infected		Non-infected		Total
	Delayed	Non-Delayed	Delayed	Non-Delayed	
Open	8	2	12	4	26
Sutured	7	0	18	3	28
Total	15	2	30	7	54

Discussion

In the 5-year period (1997-2002) under review, CDSF represented 10.2% of all the indications for operations done in our service. As with most traumatic injuries, more males than females were affected, and there were three times more adults than children. A retrospective study in our Centre, 10years before, had placed the age groups as equal¹¹. This suggests probably that even though CDSF is the most common reason for operating on children with head injury, over time, the incidence among our male adults has escalated, possibly due to a surge in traffic activities caused by the increased influx of second-hand vehicles in our country between the time of that previous report and the present study. The dominance of the frontal bone as the most commonly affected region of the skull possibly underscores the principal mechanism of injury, vehicular acceleration-deceleration, since the major aetiological factor in our study was road traffic accident. The implication of this is that road transportation represents a major factor in morbidity and mortality in our environment.

Most of our patients presented on referral from other health facilities 50(92.6%), compared to those presenting directly to our Centre 4(7.4%) from the scene of the incident. This appears to be a reversal of the

previous trend whereby only 39.2% of our cases came on referral from other facilities¹¹. Proliferation of private medical facilities in our environment seems to be responsible for this current trend, and the significance lies in the need for these health care providers to be adequately and regularly informed on the diagnostic indices of CDSF and its definitive (operative) treatment modality. This could engender precise and timely referral to reduce the morbidity burden on our patients. The need for this becomes even more pressing when it is realized that of the 54 cases reviewed, 45(83.3%) had delayed definitive treatment with 43(79.6%) of them coming on referral from other facilities. Even among the 9(16.7%) that had definitive treatment at optimum time (within 72hours), 7 were still outside referrals. Being the first port-of-call for almost all our patients, these facilities and the steps taken in them become very relevant in the overall outcome from management of our CDSF cases.

A little more than half of the delayed cases came with sutured wounds 25(46.3%), and a little less, 20(37.0%), came with unsutured scalp wounds, a minimal difference between the two groups in terms of delay in definitive treatment (Table 2). This, thus, challenges the previous report of pre-op scalp closure inducing delay in definitive treatment.

The surfacing of CT, as the major diagnostic modality has increased the diagnostic yield in CDSF, ensuring as it were, no missed diagnosis since all the patients had correct pre-op diagnosis. We had 3 reasons for CT: (a) confirmation of the clinical diagnosis of CDSF 16(29.6%); (b) confirmation of x-ray diagnosis 13(24.1%); (c) as part of routine evaluation of head injury for other intracranial lesions 10(18.5%). For the remaining 16(29.6%) diagnosed only by x-rays, the inability to afford CT bills 10(18.5%) and faulty CT machine 6(11.1%), were the reasons. Unless otherwise indicated, CT remains the gold standard in the diagnosis and follow-up of CDSF.

There were 17(31.5%) cases with infective complications, and almost all of them, 15(27.8%), had delayed operative treatment. Delay in operation, increases the chances of intracranial sepsis, and worsens prognosis, due to a secondary brain assault. Immediate elevation and debridement (with dural repair, if it is indicated) is the best treatment modality for CDSF [14]. It is noteworthy that even among the 37(68.5%) cases that did not have infective complications, 16 had unsutured, and 21 sutured wounds. It does appear, therefore, that with or without wound closure the more determinant factor in sepsis is the delay in definitive treatment. But curiously still, thirty other patients with dural lacerations and delayed treatment did not have infection. Possibly, the indiscriminate use of pre-op antibiotics or the degree of intracranial contamination may have influenced this observation.

The interval between trauma and definitive operation in this study ranged from 1 to 89days, with an average of 12.5days. Though this reduced the interval almost by half when compared with a previous study (24days), it still remains a far cry from the 2 days historically recommended as the maximum post-traumatic period to allow for a replacement of the fracture fragments without discarding them, and still way behind the 72hours post-traumatic time

lapse with successful replacement of bone fragments achieved recently by Wylen, Willis and Nanda^{7,11,14,15}. No wonder, all our patients had post-op cranial defects as replacement of fracture fragments was considered a risk not worth taking at late presentation. Perhaps, the recent report by Curry and Frim¹⁶, which proved that optimizing the cerebral perfusion pressure allowed for as long as 12 days of pre-op delay under antibiotic cover without neurological sequelae, could be quite instructive. Whether this allowed for a successful replacement of fragments should be the goal of further studies.

Reviewing the causes of delay in this study, we identified 3 different levels where delays occurred. Level A Site of incident to first health facility; B - First health facility to our Centre; C - Arrival at our Centre to definitive treatment. On the whole, there were 14 main causes occurring on several occasions to bring about a total of 87 reasons for delay among the 45 cases of delayed operations. Of these, 17(31.5%) had a single reason each, responsible for their delays, the other 28(51.9%) each had 2 to 4 reasons. Only 14 cases had delays at more than one level of care (Levels A and C 1; B and C 13 cases), none had delays at the 3 levels. There was a single incident of delay caused by transportation problems late at night at Level A; at Level B there were 23 incidents namely late/non-diagnosis of CDSF (15), ignorance of the correct treatment of CDSF (5), poor clinical status of patient (1), transportation difficulties (1) and attempted but failed debridement/elevation (1). At Level C, delays were in two patterns: (a) patient factors (b) hospital factors. Lack of funds reported by 21 patients, was the most common singular cause, whereas others arose from delays in the operating theatre (17), blood bank (7), radiology (7), laboratory (5), multi-disciplinary consults (5) and systemic infection (1), a total of 63 incidents. Considered separately, therefore, hospital delays (not caused by patients' factors) arose on 42 occasions - doubling

the 21 incidents of patients' factors - making the former, the most important cause of pre-op delays, by a ratio of 2:1 (Figure 1).

The 17 theatre problems were issues like unavailability of personnel due to other operations taking place in the different operating suites at the same time, since in our Centre, even with dedicated suites, there are no dedicated personnel allotted specifically to the different services; lack of sterile materials and instruments due to incessant electric power failure, and even lack of water supply, on some occasions. Simply put, despite all the delays before reaching our Centre, internal hospital logistics still caused a higher number of delays; meaning that most of the efforts in trying to optimize the outcome from CDSF should be geared towards making hospital processes easier and lighter for the patients, to ensure early surgical intervention.

On the long run however, 51(94.4%) patients made satisfactory clinical recovery returning to pre-morbid activities, while 3(5.6%) died in the cause of treatment. All 3 cases of mortality were males in the third and fourth decades of life, with severe head injuries (Glasgow Coma Scores of 3 - 8), and died within the first post-op week, most likely from severe primary brain assault. No patient had severe deficits or persistent vegetative state.

One patient had post-infective hydrocephalus following severe intracranial sepsis, and was treated by ventriculo-peritoneal shunting before discharge. Thirty-nine (72.7%) patients were followed up after discharge for 1 56 months (average 8.72 months), and only 2(3.7%) came back for cranioplasty.

Conclusion

CDSF, commonly an adult male traumatic injury, remains an important problem in our practice, with definitive treatment delayed

in a majority of cases by avoidable logistic problems within our Centre. Pre-op scalp wound suturing did not contribute to this delay, and the late treatment inevitably leads to craniectomy and cranial defects. Dural laceration did not increase the incidence of infection. The role of peripheral health facilities, where these cases were first attended to, appeared to be significant in the morbidity burden in our environment, and continuing education programmes stressing the need for early diagnosis and referral for specialist care, with re-certification of these care givers would go a long way in checking the negative trend.

References

1. Adeloje A, Olumide AA, Idowu LA, Ige AO and Akindele EO. Depressed skull fractures observed in Ibadan, Nigeria. *Afr J Neurol Sci* 1982; 1: 69 – 73.
2. Adeloje A and Olumide AA. Pattern of skull fractures in Nigerians. *Medicine d'Afrique Noire* 1976; 23: 129-132.
3. Adeloje A, Obiang HM and Olumide AA. Pattern of acute Head Injuries in Ibadan, Nigeria. *Medicine d'Afrique Noire* 1976; 23: 109-113.
4. Shokunbi MT and Olurin O. Childhood Head Injury in Ibadan : causes, neurologic complications and outcome. *West Afr J Med* 1994; 13: 38-42.
5. Zbinden B and Kaiser G. Specific aspects of depressed skull fractures in childhood. *Z. Kinderchir* 1989; 44: 3-7.
6. Geisler FH. Skull Fractures. Wilkins RH and Rengachary SS (eds). *Neurosurgery Vol II* 2nd ed. McGraw, New York 1996.
7. Jennet B and Miller JD. Infection after Depressed Fractures of the skull. Implications for management of non-missile injuries. *J Neurosurg* 1972; 36: 333-339.
8. Stephanov S. Brain abscesses from neglected open head injuries: experience with 17 cases over 20years. *Swiss Surg* 1999; 5: 288-292.
9. Munro D. Compound fractures of the skull. The results of surgical therapy in and eighteen cases. *N Engl J Med* 1943; 228: 737-745.
10. Shokunbi MT, Bamgbade AO, Malomo AO, et al. Focal Neurological Deficits are Predictive of Laceration of the Dura in

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- Children with Compound Depressed Skull Fractures. *Nig Qt J Hosp Med* April-June '97, Vol 7 (2).
11. Shokunbi MT, Komolafe EO, Malomo AO, et al. Scalp closure without fracture elevation does not reduce the risk of infection in patients with compound depressed skull fractures. *Afri J Med med Sci* 2000; 29: 293-296.
 12. Vender JR and Bierbrauer K. Delayed intracranial hypertension and cerebellar tonsillar necrosis associated with a depressed occipital skull fracture compressing the superior sagittal sinus. Case report. *J Neurosurg* 2005 Nov; 103: 458-461.
 13. Binder DK, Sarkissian V, Schmidt MH and Pitts LH. Resolution of intracranial hypertension after elevation of depressed cranial fracture over the superior sagittal sinus: case report. *Neurosurgery* 2004 Oct; 55: 986.
 14. Hammon WM. An analysis of 2187 conservative penetrating wounds of the brain from Vietnam. *J Neurosurg* 1974; 41: 208-216.
 15. Wylen EL, Willis BK and Nanda A. Infection rate with replacement of bone fragment in compound depressed skull fractures. *Surg Neurol* 1999; 51: 452-457.
 16. Curry DJ and Frim DM. Delayed repair of open depressed skull fracture. *Paed Neurosurg* 1999; 31: 294-297.