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Outcome and Management of Occipitofrontal Contrecoup Head Injury

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Abstract

Contrecoup brain injury refers to the classical opposite of the primary impact. Tamil Nadu has the highest rate of road traffic accident in India. Madurai has peak mortality due to accidents in India. Previous studies done on contrecoup head injury had shown patterns of injuries and mechanism of injury. Outcome and management of only occipitofrontal contrecoup head injury has been taken in this study. Mortality in this specific group is very high. Seventy-six patients of this specific head injury were admitted at Government Rajaji Hospital, Madurai. Patients were assessed for Glasgow coma scale (GCS), age, sex, progression of volume, mortality, traumatic brain injury-induced coagulopathy, and pillion rider outcome. Fourteen patients were surgically treated by decompressive craniectomy. Conservative management was done by antiepileptic and antiedema measures. Mostly affected were males ($n = 54$) followed by females ($n = 22$). GCS on admission mean value 9. In our study, mortality was 32% with sudden death of three patients due to hypothalamic compression. Hospital stay of the patient was significantly increased with progression of lesion with mean 9 days and *p*-value less than 0.01. Pillion riders ($n = 18$) were also affected in our study. Traumatic brain injury-induced coagulopathy ($n = 12$) was also detected, which was treated by injection tranexamic acid and injection vitamin K, thereby not leading to any death due to coagulopathy with significant *p*-value less than 0.01. We recommend helmet for both main and pillion rider with strict speeding regulations.

Keywords
- contrecoup head injury
- frontal hemorrhagic contusion
- mortality
- occipital bone fracture
- pillion rider
- traumatic brain injury-induced coagulopathy
- road traffic accidents

Introduction

Contrecoup brain injury refers to the classical opposite of the actual place of head hit. French for "counterblow" is "contre-coup." Hippocrates used the term contrecoup to describe a fracture that is located on the other side of point of impact.[1]

Regarding contrecoup injury, there are four theories discussed.[2] According to the positive pressure theory, stress is first created by the initial lagging of the brain with movement of the skull, then by the brain being compressed against a motionless, irregular skull.[3] According to the negative pressure or cavitation theory, when the brain moves in one way, the opposite part of the brain experiences stress, which can lead to cerebral damage. According to the angular acceleration theory, the brain is connected to certain regions, such as the

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brainstem, which makes some regions of the brain more susceptible to increased acceleration and deceleration.[4] The rotational shear stress theory takes both brain rotation and displacement along the trauma's axis into account.[5]

The most frequent causes are falls and vehicular accidents. At lower ages, males prevail in contrecoup, with an evener gender ratio in older adults.

Injury before the age of four is uncommon in pediatrics due to infants' open sutures and the elastic nature of their bones. After the age of 4, frequency rises rapidly.[1]

In comparison to the frontal and posterior locations of contrecoup, lateral contrecoup intracerebral hemorrhage is more prone to hemorrhagic progression.[6]

Materials and Methods

It is a prospective study in which patients were taken up from July 2021 to February 2023. Patient were admitted in neurosurgery head injury intensive care unit in Madurai Medical College. Computed tomography (CT) brain with bone window was taken for all patients and selected on the basis of the CT finding. Patient were selected on the basis of criteria mentioned in the following text.

Inclusion Criteria

(1) Coup injury should be only in occipital region with undisplaced linear fracture of occipital bone without underlying fracture hematoma or intracranial injury.
(2) Contrecoup injury should be only in frontal region, unilateral and bilateral, which includes hemorrhagic contusion, intracerebral hemorrhage, and subdural hemorrhage.

Exclusion Criteria

(1) Coup-contrecoup injury in temporal or parietal region.
(2) Contrecoup injuries in frontal region with fracture with underlying hematoma.
(3) Other associated injuries like extremity fracture, chest and abdomen injuries.
(4) Patients on any anticoagulant therapy or any congenital coagulation disorders.

Assessment of the selected patient was done on the basis of Glasgow coma scale (GCS), age, sex, progression of volume, mortality, and traumatic brain injury-induced coagulopathy.

The collected data were analyzed with IBM SPSS Statistics for Windows, Version 29.0. (IBM Corp, Armonk, New York, United States). To describe the data descriptive statistics frequency analysis, percentage analysis was used for categorical variables, and the mean and standard deviation were used for continuous variables. To find the significant difference between the bivariate samples in independent groups, the independent sample *t*-test was used. To assess the relationship between the variables, the Karl Pearson correlation was used. To find the significance in qualitative categorical data, chi-squared test was used similarly if the expected cell frequency is less than 5 in 2×2 tables, then the Fisher's exact test was used. In all the above statistical tools, the probability value 0.05 is considered as significant level.

p-Value	**Highly statistical significant at $p < 0.01$
p-Value	# No statistical significant at $p > 0.050$

Results

Seventy-six patients of specific injury were identified and treated. Fourteen patients progression of lesion with GCS deterioration was seen. Surgical decompression was done for these lesions (►**Figs. 1** and **2**). Frontal surgical decompression was done bilaterally in 10 cases and unilaterally in 4 cases.

Males were mostly affected by the road traffic accident (71%; $n = 54$) in compared to females (28.9%; $n = 22$). Females and aged males (>60 years) as a pillion rider were mostly affected (►**Fig. 3**). All patients were two-wheeler riders.

GCS on admission ranged from 3 to 15 (mean: 9). About 57.8% ($n = 44$) patients had GCS less than 8.

Patients were assessed per day by GCS analysis and serial CT brain.

In our study, mortality rate for this specific group was 32%. Twenty-five out of seventy-six patients died. Mean for death on posttraumatic day was 3.2 (►**Table 1**). Three cases with GCS of 15 on admission had a sudden deterioration leading to death.

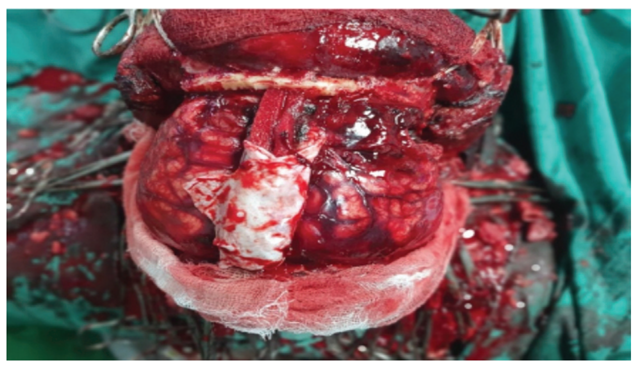

Fig. 1 Bilateral frontal decompressive, craniectomy done with no bone bridge with hemorrhagic contusion on right side.

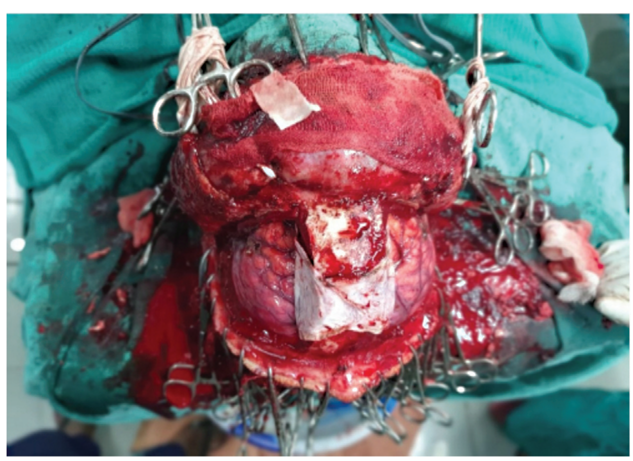

Fig. 2 Bifrontal decompressive frontal with bone bridge.

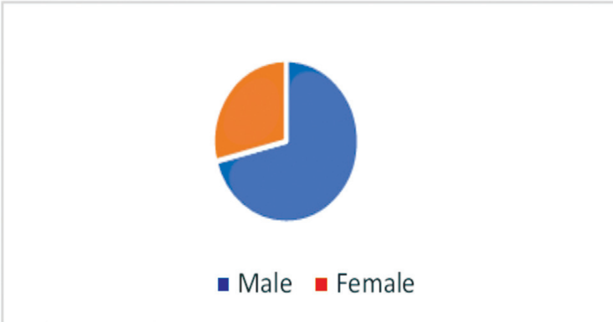

Fig. 3 Pie chart showing number of cases with M:F ratio (male [$n = 54$, 71%]); female [$n = 22$, 28.9%]).

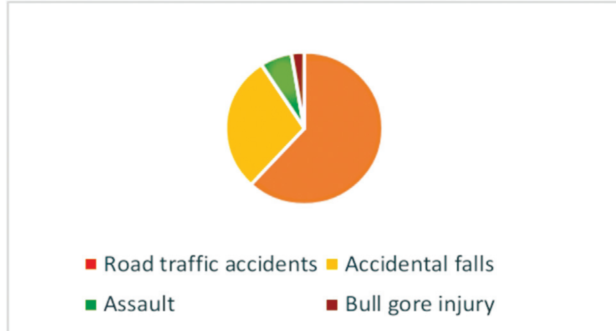

Fig. 4 Pie chart showing mode of injury (road traffic accidents—47%, accidental falls—22%, assault—5%, bull gore injury—2%).

Mean age in our study group was 43.8 (►**Table 1**). Road traffic accidents were the leading cause of injury followed by accidental falls (►**Fig. 4**). Contrecoup injuries in pillion rider patients were also high. About 23.6% ($n = 18$) patients were pillion riders. In our study, pillion rider injury was there but mortality was not significant (p-value >0.05; ►**Tables 2** and **3**). Forty-seven percent patients were affected by road traffic accidents 22% by accidental falls, 5% by assault, and 2% by bull gore injury

Pattern of injuries due to hemorrhagic contusion was the most common lesion followed by intracerebral hemorrhage, subdural hematoma (SDH) with contusion, and SDH with subarachnoid hemorrhage (SAH; ►**Fig. 5**).

Patients were managed by surgical management depending on the volumetric analysis (contusion size > 30cc), mass effect, and GCS deterioration (►**Fig. 5**). Patients with significant mass effect along with GCS 3 and brainstem reflexes absent with ionotropic supports were kept on conservative management. Conservative management was done by antiedema and antiepileptic measures.

Patients were also checked for traumatic brain injury-induced coagulopathy. Around 15.7% ($n = 12$) patients were having prothrombin time more than 13 and international normalized ratio more than 1.5. Injection vitamin K and injection tranexamic acid was given to the patients. Correlation of coagulopathy with outcome was also significant in our study with p-value less than 0.01 (►**Table 4** and **5**).

Table 2 Correlation of pillion rider head injury with the outcome

			Outcome		Total
			Died	Discharge	
Pillion rider	No	Count	20	38	58
		%	80.0%	74.5%	76.3%
	Yes	Count	5	13	18
		%	20.0%	25.5%	23.7%
Total	Count	25	51	76	
	%	100.0%	100.0%	100.0%	

Table 3 p-Value was not significant which denotes pillion rider mortality was not significant

	Value	df	p-Value
Pearson chi-squared test	0.280a	1	0.597

There was also significant increase in the days of hospitalization with progression of lesion or edema with mean of 10.6 with significant p-value less than 0.01 (►**Tables 6** and **7**).

Correlation of surgery with progression of lesion/edema was also significant with p-value less than 0.01 (►**Tables 8** and **9**). Surgery was done on posttraumatic day with mean of 3.2 (►**Table 1**).

Table 1 Age mean value 43.8. Progression of lesion and edema mean value 1.4. Period of hospitalization mean value 8.9. Death on posttraumatic day mean 3.2. Surgery on posttraumatic day mean 3.2

Descriptive statistics					
	n	Minimum	Maximum	Mean	SD
Age	76	15.0	74.0	43.8	14.4
Progression lesion and edema	76	1	2	1.4	0.5
Period of hospitalization	76	1.0	22.0	8.9	5.7
Death at posttraumatic day	25	1.0	6.0	3.2	1.2
Surgery on posttraumatic day	14	1.0	5.0	3.2	1.5

Abbreviation: SD, standard deviation.

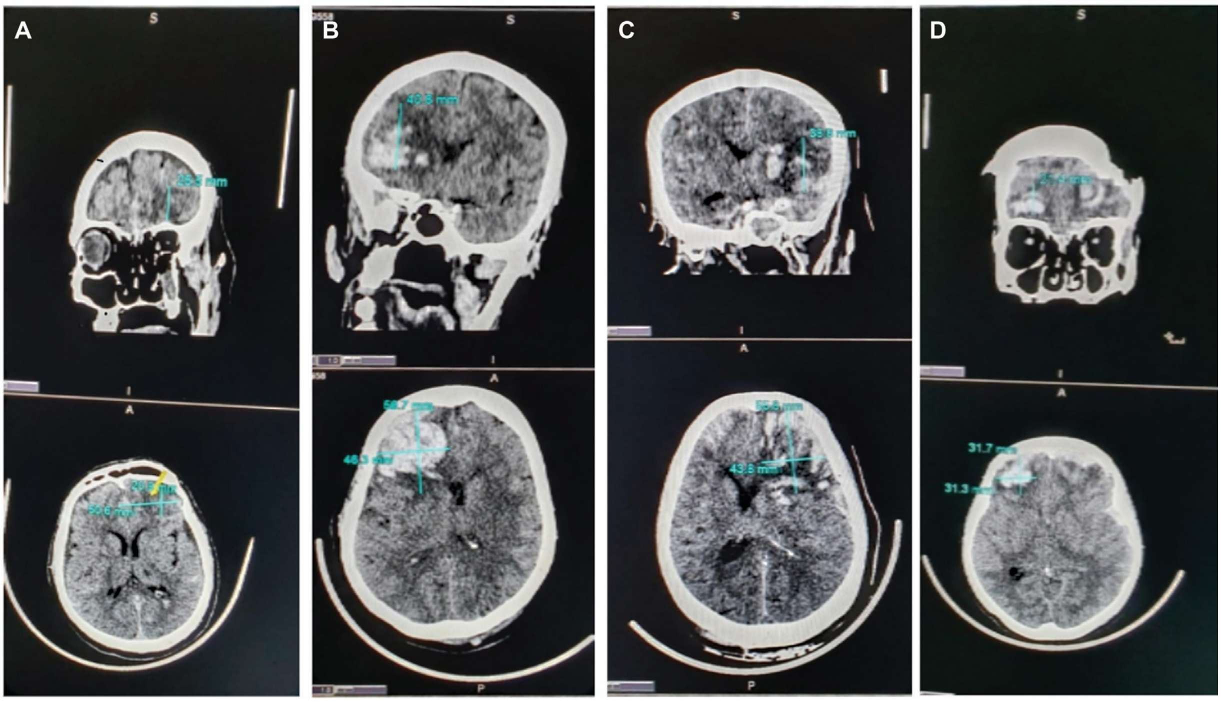

Fig. 5 Serial computed tomography showing volumetric analysis and different pattern of injuries. (**A**) Left frontal Intracerebral hemorrhagic contusion. (**B**) Right frontal intracerebral hemorrhage. (**C**) Left frontal hemorrhagic contusion with subarachnoid hemorrhage. (**D**) Right frontal intracerebral hemorrhage with left frontal acute subdural hematoma.

Table 4 Correlation of coagulopathy with outcome

			Outcome		Total
			Died	Discharge	
Coagulopathy	No	Count	25	39	64
		%	100.0%	76.5%	84.2%
	Yes	Count	0	12	12
		%	0.0%	23.5%	15.8%
Total		Count	25	51	76
		%	100.0%	100.0%	100.0%

Table 5 Significant *p*-Value denotes that the mortality was not increased due to traumatic brain injury induced coagulopathy, if early detection and management by Injection vitamin K and tranexamic acid was done

	Value	df	Asymptotic significance (2-sided)	*p*-Value
Pearson chi-squared test	6.985[a]	1	0.008	
Fisher's exact test				0.007

Note: [a] denotes 1 cells (25%) have expected count less than 5. The minimum expected count is 0.05.

Table 6 Correlation between progression of lesion and edema with period of hospitalization

Progression of lesion and edema		*n*	Mean	SD
Period of hospitalization	Yes	42	10.6	5.8
	No	34	6.7	4.9

Abbreviation: SD, standard deviation.

Table 7 Significant *p*-Value denotes period of hospitalization is increased due to progression of lesion

		Levene's test for equality of variances		t-Test for equality of means						
		F	Sig.	*T*	df	*p*-Value	Mean difference	Std. error difference	95% confidence interval of the difference	
									Lower	Upper
Period of hospitalization	Equal variances assumed	1.025	.315	3.159	74	0.002	3.9426	1.2479	1.4560	6.4291

Table 8 Correlation of surgery done with/without progression of lesion

Surgery		*n*	Mean	SD
Progression of lesion/edema	Yes	10	35.9	3.2
	No	29	22.3	3.1

Table 9 *p*-Value significant means that progression of lesion mostly leads to surgical intervention

		Levene's test for equality of variances		t-Test for equality of means						
		F	Sig.	t	df	*p*-Value	Mean difference	Std. error difference	95% Confidence interval of the difference	
									Lower	Upper
Progression of volume	Equal variances assumed	0.322	.574	11.899	37	0.0005	13.624	1.145	11.304	15.944

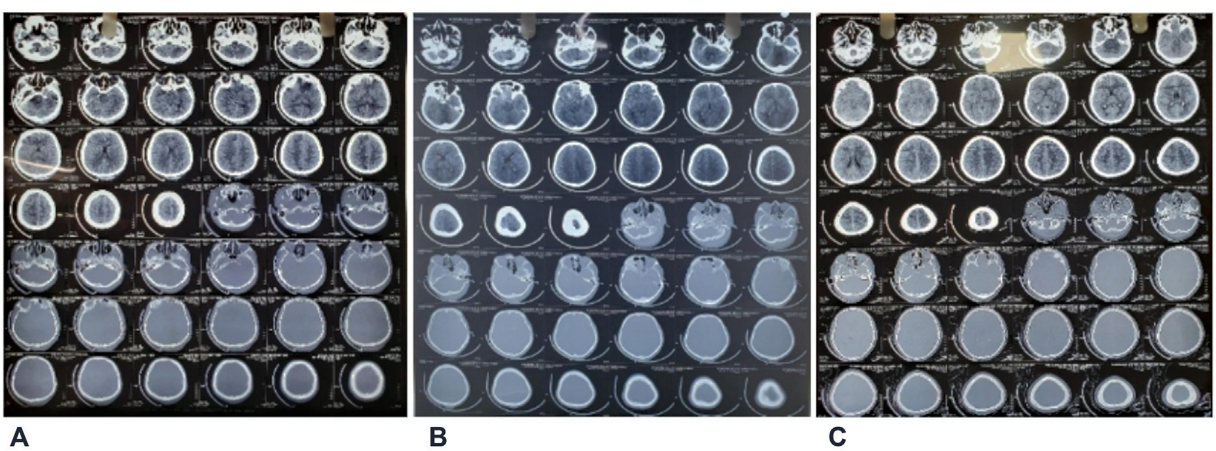

Fig. 6 Computed tomography of three patients with Glasgow coma scale (GCS) of 15 on admission who had sudden deterioration in GCS leading to death. (**A**) Hemorrhagic contusion in bilateral frontal lobe basifrontal region with perilesional edema with left frontal lobe intraparenchymal bleed. (**B**) Hemorrhagic contusion in bilateral frontal lobe extending to basifrontal region with perilesional edema and slight effacement of right lateral ventricle of frontal horn. (**C**) Hemorrhagic contusion in basifrontal region with perilesional edema with subarachnoid hemorrhage in the left frontal region and intraparenchymal bleeding in right frontal lobe.

Discussion

Head injury has deep impact on psychosocial and economic status of the country. Due to the increase in number vehicles, poor safety guidelines, and high-speed vehicles, head injury incidence has increased all over world.

Brain damage in head injury is classified as diffuse and focal. Contrecoup injury is a form of focal injuries.[7] Graham and Lantos described the biomechanics of contrecoup injuries that is observed as when one slips with his or her feet moving forward and skull rotating backward with occiput eventually hitting the surface.[8]

➢ The brain is displaced by cerebrospinal fluid, with the brain being propelled upward toward the contrecoup location.
➢ The buoyancy of brain causes further tendency for the brain to move toward the contrecoup location.
➢ The relatively small surface areas of frontal region result in impact forces in contrecoup areas being absorbed by a relatively small amount of brain substance, increasing force per unit area thereby increasing the amount of contusion.
➢ The irregular surfaces of the anterior cranial fossa cause focal shear stresses tearing the adjacent brain surfaces and increasing the degree of injury.

In our study, mean age for specific type of contrecoup injury was 43.8. Kraus reported the most common age group affected between the age of 20 and 40 years and low incidence at extremes of age.[9]

Most common mode of injury was road traffic accidents that was more than 60%, which was similar to the study by Bhateja et al.[10] In our study, we also found pillion rider head injury was high (23.6%; $n = 18$). Most common pattern of head injury was hemorrhagic contusion followed by intracerebral hemorrhage, acute SDH, acute SDH with contusion, acute SDH with SAH that is not similar with study of Manpreet et al where acute SDH was the most common pattern of injury.[11] In our study, contrecoup traumatic brain injury-induced coagulopathy has been treated with injection vitamin K and injection tranexamic acid with significant result. Previously, no study for contrecoup head injury has been reported.

In our study, mortality rate for this specific group was 32.9% ($n = 25$) that was better than the studies by Bhateja et al and Jayakumar et al in which the mortality rates were 44 and 53%, respectively.[10,12] Three cases with GCS 15 on admission had a sudden deterioration leading to death. All three cases were devoid of any comorbidities and no other associated injuries were present. Sudden deterioration in patients may due to mass effect on hypothalamus.

Conclusion

Prevention is better than cure. As contrecoup injury mortality is very high; so, we should prevent it by helmets and speeding regulations. Even in our study pillion riders were also having high incidence of head injury; so, we recommend helmet in this group.

Conflict of Interest
None declared.

References

1 Courville CB. Contrecoup injuries of the brain in infancy. Arch Surg 1965;90:157–165
2 Drew LB, Drew WE. The contrecoup-coup phenomenon: a new understanding of the mechanism of closed head injury. Neurocrit Care 2004;1(03):385–390
3 Allen FJ. The mechanism of contrecoup and of certain other forms of intracranial injury. BMJ 1896;1(1846):1196–1197
4 Bayly PV, Cohen TS, Leister EP, Ajo D, Leuthardt EC, Genin GM. Deformation of the human brain induced by mild acceleration. J Neurotrauma 2005;22(08):845–856
5 Goggio AF. The mechanism of contrecoup injury. J Neurol Psychiatry 1941;4(01):11–22
6 Cepeda S, Gómez PA, Castaño-Leon AM, Munarriz PM, Paredes I, Lagares A. Contrecoup traumatic intracerebral hemorrhage: a geometric study of the impact site and association with hemorrhagic progression. J Neurotrauma 2016;33(11):1034–1046
7 Mahapatra AK, Jaiswal A. Epidemiology of head injury. Neurosci Today 2003;7:29–32
8 Graham DI, Lantos PL. Greenfield's Neuropathology. 7th ed. London:: Arnold; 2002
9 Kraus JF. Epidemiology. In: Elizabeth F, ed. Head Injury Clinical Management and Research. Geneva, Switzerland: AIREN; 1990: 113–124
10 Bhateja A, Shukla D, Devi BI, Sastry KV. Coup and contrecoup head injuries: predictors of outcome. Indian J Neurotrauma 2009;6 (02):115–118
11 Banga MS, Sandeep BV, Roy K, Saha KS, Dixit S, Ghosh P. Contrecoup head injury. Indian J Neurosurg 2017;6:103–106
12 Jayakumar PN, Sastry Kolluri VR, Basavakumar DG, et al. Prognosis in contre-coup intracranial haematomas–a clinical and radiological study of 63 patients. Acta Neurochir (Wien) 1991;108(1-2):30–33

