Journal of Cranio-Maxillofacial Surgery (2008) **36**, 372–377

doi:10.1016/j.jcms.2007.12.002, available online at http://www.sciencedirect.com

Traumatic intracranial haemorrhage in conscious patients with facial fractures – A review of 1959 cases

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SUMMARY. Objective: **Facial fracture patients who are conscious with a Glasgow Coma Scale (GCS) score of 15 in the absence of clinical neurological abnormalities are commonly not expected to have suffered severe intracranial pathology. However, high velocity impact may result in intracranial haemorrhage in different compartments.** Methods: **Over a 7-year period, 1959 facial fracture patients with GCS scores of 15 and the absence of neurological abnormalities were analysed. In 54 patients (2.8%) computed tomography scans revealed the presence of accompanying intracranial haemorrhage (study group). These patients were compared with the 1905 patients without intracranial haemorrhage (control group).** Results: **Univariate analysis identified accompanying vomiting/nausea and seizures, cervical spine injuries, cranial vault and basal skull fractures to be significantly associated with intracranial bleeding. In multivariate analysis the risk was increased nearly 25-fold if an episode of vomiting/nausea had occurred. Seizures increased the risk of bleeding more than 15-fold. The mean functional outcome of the study group according to the Glasgow Outcome Scale was 4.7 ± 0.7.** Conclusion: **Intracranial haemorrhage cannot be excluded in patients with facial fractures despite a GCS score of 15 and normal findings following neurological examination. Predictors, such as vomiting/nausea or seizures, skull fractures and closed head injuries, enhance the likelihood of an intracranial haemorrhage and have to be considered. © 2008 European Association for Cranio-Maxillofacial Surgery**

Keywords: **intracranial haemorrhage, facial fractures, conscious patients, GCS, neurological injury, maxillofacial trauma**

INTRODUCTION

Sport and traffic accidents have emerged as some of the principal causes of severe injury in the western world (*Gassner* et al., 2003). In most cases, the victim has little facial protection commonly resulting in severe maxillofacial trauma. Patients sustaining facial fractures are at great risk of accompanying injuries. Localized injury to the face resulting in fractures may also involve the brain and meninges. In many cases, facial fractures distract attention from more critical, often life-threatening injuries (*Hohlrieder* et al., 2004). Conscious patients with a Glasgow Coma Scale (GCS) score 15 and no obvious neurological abnormalities are commonly not expected to suffer from severe intracranial pathology. However, high velocity impact with enough force to fracture facial bones may also cause rupture of intracranial vessels leading to haemorrhages in different compartments. Recognition of such life-threatening conditions is critical, because a traumatic intracranial haematoma is a major cause of morbidity and mortality. Early detection may lead to improved results as prompt evacuation of an intracranial haematoma is crucial to improve the outcome of head injury patients (*Mendelow* et al., 1979; *Haug* et al., 1992; *Jiang* et al., 2005; *Atzema* et al., 2006; *Bouamra* et al., 2006). The objective should be to identify patients at risk before deterioration takes place.

In this case–control study, we analysed a subgroup of alert, neurologically normal patients within a large number of facial fracture patients. All of them were admitted to the emergency department with GCS scores of 15 developing later signs of intracranial haemorrhage. By means of univariate and multivariate analyses the association between intracranial haemorrhage and the presence of potentially predictable clinical features were assessed.

PATIENTS AND METHODS

Over a 7-year period (1991–1997) 6649 patients with cranio-maxillofacial injuries were treated at the University Hospital of Innsbruck. Patient information was gathered and stored in databases allowing analyses of various parameters. Two thousand one hundred and ninety-five

patients (33.0%) sustained facial fractures, diagnosed on the basis of computed tomography (CT) and judged by a radiologist. Excluded from this study were 236 facial fracture patients (10.8%) because of admission with neurological abnormalities or GCS scores lower than 15 or not assessable because of deep sedation. The remaining 1959 patients (89.2%) were conscious with GCS scores of 15 and had no neurological abnormalities detected after standardized neurological examination including mental status evaluation as well as assessment of motor, sensory, cerebellar and reflex function. In 54 patients (2.8%) CT − mainly performed because of apparent or presumptive facial fractures − revealed the presence of an intracranial haemorrhage (study group). This patient cohort group was compared with the 1905 alert facial fracture patients without intracranial haemorrhage (control group).

Both groups were analysed and compared with respect to age, sex, cause of injury, mechanism of injury, type and location of the facial injury, associated injuries, type and location of intracranial haemorrhage and skull fractures, occurrence of vomiting/nausea, seizures, raised intracranial pressure (ICP), brain oedema, loss of consciousness or amnesia, duration of stay in critical care unit and duration of stay in the hospital overall. In the study group, we additionally assessed the necessity of surgical intervention and Glasgow Outcome Scale (GOS) scores in detail.

The causes of injury were classified into categories as follows: sports, traffic, industrial accidents, assault, activities of daily living (ADL) and play accidents. The classification of the injury mechanism included falls, collisions, hits/pushes/kicks and others. Concomitant injuries were included when they necessitated surgical care. Open brain injuries were diagnosed on the basis of cerebrospinal fluid leakage. Facial fractures were summarized as fractures of the orbit, zygoma, mandible, maxilla and nose. Fractures of the Le Fort I−III categories were also included in the zygomatic or nasal complex group as these bones were involved. Besides cervical spine fractures, skull fractures were reviewed and further classified as fractures of the cranial vault (CV) or the base of skull. The location of epidural haematoma (EDH) and subdural haematoma (SDH) was frontal, temporal, parietal or occipital. Intracerebral haematomas (ICHs) were additionally localised as being in the brainstem and the basal ganglia area. The category "closed head injury (CHI)" was defined by either a witnessed transient loss of consciousness or a patient's report of temporary loss of awareness and/or post-traumatic amnesia. The necessity of surgical intervention was exclusively related to the intracranial haemorrhage. The decision to perform craniotomy was based on the opinions of on-site neurosurgeons. The occurrence of vomiting/nausea and/or seizures was only registered at the trauma scene or during transport. Patients presenting with vomiting/nausea or seizures in the emergency department were classified as neurologically abnormal and were not considered within the group of 1959 neurologically normal, alert patients. Patients with nausea were only considered in the category "vomiting/nausea" if they were actually about to vomit. Functional outcome was evaluated at discharge from the hospital by the GOS as follows: 1 = death, 2 = vegetative state, 3 = severe disability, 4 = moderate disability and 5 = good recovery.

Statistics

Statistical calculations performed included descriptive as well as conclusive analysis. By means of univariate and multivariate analyses the association between intracranial haemorrhage and the presence of potentially predictive clinical features was assessed. To test for differences between the study and the control group the Pearson's chi-square test and the Fisher's exact test (when the expected counts were less than five) was used for categorical variables. Continuous variables were tested with the use of the Student's *t*-test. *p*-Values less than 0.05 were considered significant. Multivariate logistic regression analysis was performed to determine the odds ratios (ORs) and 95% confidence intervals (CIs) for different potential predictors of intracranial haemorrhage. Thus, selection of variables was based on clinical relevance and univariate comparisons (entry criteria $p < 0.05$).

RESULTS

Age and sex

During the study period 54 patients were identified with CT-proved intracranial haematomata (study group) while 1905 alert facial fracture patients without intracranial

Table 1 − Demographic characteristics, cause and mechanism of injury of 1959 patients in a univariate analysis

	Total (%, $n = 1959$)	Study group (%, $n = 54$)	Control group (%, $n = 1905$)	*p*-Value	OR	95%CI
Age	34.8 ± 19.1	41.1 ± 21.1	34.6 ± 19.1	<0.05	−	−
Sex	1443 (73.7)	43 (79.6)	1400 (73.5)	ns	1.41	0.72−2.76
Traffic accident	310 (15.8)	17 (31.5)	293 (15.4)	<0.005	2.53	1.41−4.55
Industrial accident	118 (6.0)	3 (5.6)	115 (6.0)	ns	0.92	0.28−2.98
Sports accident	731 (37.3)	18 (33.3)	713 (37.4)	ns	0.84	0.47−1.48
Assault accident	287 (14.7)	0 (0)	287 (15.1)	<0.001	−	−
ADL accidents	513 (26.2)	16 (29.6)	497 (26.1)	ns	1.19	0.66−2.16
Falls	851 (43.4)	22 (40.7)	829 (43.5)	ns	0.89	0.52−1.55
Hits/pushes/kicks	427 (21.8)	1 (1.9)	426 (22.4)	<0.001	0.07	0.01−0.48
Collisions	209 (10.7)	3 (5.6)	206 (10.8)	ns	0.49	0.15−1.57
Other mechanisms	472 (24.1)	28 (51.9)	444 (23.3)	<0.001	3.54	2.06−6.11

ns: Not significant.

Table 2 − Injury profile and predictors of intracranial haemorrhage in the cohort, the study and control groups in a univariate analysis

	Total (%, $n = 1959$)	Study group (%, $n = 54$)	Control group (%, $n = 1905$)	p-Value	OR	95%CI
Fractures						
Le Fort I	46 (2.4)	6 (11.1)	40 (2.1)	<0.0001	5.83	2.36−14.40
Le Fort II	52 (2.7)	4 (7.4)	48 (2.5)	<0.05	3.10	1.07−8.92
Le Fort III	34 (1.7)	9 (16.7)	25 (1.3)	<0.0001	15.04	6.64−34.06
Zygoma	854 (43.6)	32 (59.3)	822 (43.1)	<0.05	1.92	1.11−3.32
Orbit	862 (44.0)	38 (70.4)	824 (43.3)	<0.0001	3.12	1.73−5.63
Mandible	631 (32.2)	7 (13.0)	624 (32.8)	<0.005	0.31	0.14−0.68
Maxilla	199 (10.2)	8 (14.8)	191 (10)	ns	1.56	0.73−3.36
Nose	237 (12.1)	17 (31.5)	220 (11.5)	<0.0001	3.52	1.95−6.36
Associated injuries						
Thorax	54 (2.8)	4 (7.4)	50 (2.6)	<0.05	2.79	1.03−8.54
Abdomen	11 (0.6)	1 (1.9)	10 (0.5)	ns	3.58	0.45−28.44
Cerv. spine	31 (1.6)	4 (7.4)	27 (1.4)	<0.005	5.56	1.88−16.50
Extremities	38 (1.9)	2 (3.7)	36 (1.9)	ns	1.42	0.43−4.64
Open brain	36 (1.8)	22 (40.7)	14 (0.7)	<0.0001	92.86	43.62−197.71
CV fracture	45 (2.3)	25 (46.3)	20 (1.0)	<0.0001	81.25	40.63−162.47
BS fracture	62 (3.2)	29 (53.7)	33 (1.7)	<0.0001	65.8	34.83−124.31
Associated signs						
Brain oedema	17 (0.9)	12 (22.2)	5 (0.3)	<0.0001	108.57	36.61−322.0
Raised ICP	12 (0.6)	5 (9.3)	7 (0.4)	<0.0001	27.67	8.48−90.24
CHI	322 (16.4)	44 (81.5)	278 (14.6)	<0.0001	25.75	12.81−51.77
Vomiting	45 (2.3)	23 (42.6)	22 (1.2)	<0.0001	63.5	32.05−125.83
Seizures	12 (0.6)	7 (13.0)	5 (0.3)	<0.0001	56.6	17.33−184.84

ns: Not significant; inj: injury; cerv: cervical.

haematomas served as the control group. The average age was 34.8 ± 19.1 years, and men constituted 73.7% of the whole series. In comparison with the control group, the mean age in the study group was significantly higher. Age ranges were similar (study group 3−86 years, control group 1−98 years). Regarding the gender distribution there was no significant difference between the two groups (Table 1).

Cause and mechanism of injury

The most common cause of injuries was sporting accidents followed by ADL and traffic accidents. Assault and industrial accidents played a less important role. Group-specific analysis revealed a significantly higher proportion of traffic accidents in the study group. Sports accidents were almost equally frequent in both groups, while no injuries from assaults were found in the study group. Regarding the mechanism of injury, falls accounted for the majority of the injury mechanisms with no significant difference between the two groups. Hits, pushes and kicks ranked second with a particularly low incidence in the study group (Table 1).

Injury profile and predictors

The orbital and zygomatic region had the highest proportion of fractures. Except for mandibular fractures, all fractures were significantly more frequent in the study group. Regarding the ORs Le Fort III − fractures ranked first, followed by Le Fort I − fractures. One hundred and sixty patients (8.2%) sustained additional injuries of thorax, abdomen, cervical spine, or extremities, the most common being thorax injuries. Univariate analysis identified accompanying thorax injuries and cervical spine injuries to be significantly associated with intracranial bleeding. Other significant predictors were CV and basal skull (BS) fractures, open brain injuries and closed head injuries, as well as the occurrence of vomiting/nausea and seizures. The development of brain oedema and raised ICP were both significantly more frequent in the study group (Table 2).

Table 3 − Independent predictors of intracranial haemorrhage in multivariate analysis

	OR	95%CI	p-Value
Age	1.0133	0.9941−1.0328	0.1761
Open brain injury	4.7592	1.4263−15.8796	0.0112
BS fracture	5.5631	1.9034−16.2597	0.0017
CHI	8.0698	3.3602−19.3801	<0.0001
CV fracture	9.9144	3.7073−26.5143	<0.0001
Seizures	15.5141	1.2388−194.2949	0.0335
Vomiting/nausea	24.9540	8.3036−74.9921	<0.0001

Multivariate regression analysis

Multivariate logistic regression analysis identified vomiting/nausea and seizures as the strongest independent predictors of intracranial bleeding. The risk was increased nearly 25-fold if an episode of vomiting/nausea occurred and seizures increased the risk of bleeding more than 15-fold. Other significant predictors with ORs between 4 and 10 were CV fractures, CHI, BS fracture and open brain injury. Age was associated with an increasing risk of 1.33% per year (Table 3).

Intracranial haemorrhage

Overall 18 patients sustained extradural haematomas (0.92%), 15 SDHs (0.77%), 23 subarachnoid haemorrhages (SAHs) (1.17%) and 31 developed ICHs

Table 4 − Location of different intracranial haematomas and outcome according to the GOS (mean ± SD)

	No. (%, $n = 54$)	Location						Outcome
		fro	tem	par	occ	bga	bst	GOS
EDH	18 (33.3)	8	8	2	1	−	−	4.67 ± 0.67
SDH	15 (27.8)	9	8	10	7	−	−	4.53 ± 0.83
SAH	23 (42.6)	−	−	−	−	−	−	4.57 ± 0.66
ICH	31 (57.4)	26	14	11	4	3	3	4.55 ± 0.71

fro: Frontal; tem: temporal; par: parietal; occ: occipital; bga: basal ganglia area; bst: brainstem.

Table 5 − Outcome of the study group according to the GOS

	No. (%, $n = 54$)
GOS 1	0
GOS 2	0
GOS 3	5 (9.3)
GOS 4	9 (16.7)
GOS 5	40 (74.1)

(1.58%). Therefore, 33 patients suffered from only one type of intracranial haemorrhage and 21 from more than one. Extradural haematoma and ICH were predominantly located in the frontal and temporal area, while SDHs were most frequently in the parietal region. Table 4 shows the detailed locations of the haematomas. One in five patients (11, 20.3%) of the study group required surgical treatment of the ICH. Surgical options consisted of conventional craniotomy and evacuation of the clot under direct vision, or stereotactic aspiration via a burr hole.

Hospitalization and outcome of the study group

The period of hospitalization of the intracranial haemorrhage patients ranged from 3 to 94 days with an average of 16.3 ± 13.7 days. Two of three patients (18, 66.7%) required intensive care with an average duration of 4.4 ± 6.9 days (range 1−42 days). In contrast, the mean hospital stay in the control group (5.5 ± 7.3 days, range 1−55) as well as the mean duration of critical care (0.3 ± 2.5 days, range 1−12) was significantly lower ($p < 0.001$). The mean functional outcome of the study group patients according to the GOS was 4.7 ± 0.7. At the time of discharge, the GOS indicated good recovery (GOS 5) in 40 patients (74.1%), while 14 patients were still disabled (GOS 4 and GOS 3). None of the intracranial haemorrhage patients remained in a vegetative state (GOS 2) and the mortality rate was 0%. There was no significant difference in outcome between the four types of haemorrhage. In the control group no deaths and no GOS 2 and 3 were reported (Table 5).

DISCUSSION

Patients sustaining facial fractures are at high risk of accompanying injuries. *Gwyn* et al. (1971) analysed the records of 1517 facial fracture patients and reported a 19.2% incidence of life-threatening injuries. *Luce* et al. (1979) in their review of 1020 facial fractures found life-threatening injuries in 22.1%. The presence of any facial injury should always be of clinical concern, because it can be a marker for substantial transfer of energy to the brain, with associated brain damage (*Hohlrieder* et al., 2004). In such patients, rapid identification and assessment is vital because surgical therapy is often urgently required.

Diagnosis and management of head injury patients has been revolutionized by the introduction of CT, and its increasing availability has led to its routine use. Although it has become the method of choice for the identification of intracranial disorders, indications are unclear in head trauma patients with GCS scores of 15 (*Stiell* et al., 2001a; *Sifri* et al., 2006). A number of studies have been performed in the past 10 years to identify high risk findings that would clearly indicate which group of patients with minor head injury should undergo CT-imaging (*Stiell* et al, 2001a−*c*). The range of recommendations regarding the management of patients with head injury and GCS score 15 despite a history of loss of consciousness or amnesia, is confusing (*Livingston* et al., 2000). The reported incidence of a positive CT scan in this patient population is between 3 and 19%, and the need for neurosurgical intervention is 0.08−3.3% (*Stiell* et al., 2001c). Some studies have concluded that a patient with minor head injury and a normal neurological examination can be discharged home safely (*Taheri* et al., 1993; *Huynh* et al., 2006). *Miller* et al. (1996) found that routine CT scanning of patients with a history of loss of consciousness or amnesia, but with no symptoms of nausea, vomiting, headache or dizziness, or signs of depressed skull fracture has a minimal effect on trauma management and is not warranted. However, there are reports of patients requiring operative therapy despite presenting GCS score of 15 and minor head trauma (*Borczuk*, 1995).

Within this study we specifically analysed intracranial haemorrhage in conscious facial fracture patients assessing its incidence and significance. We found predictive clinical features by univariate and multivariate analyses. Our aim was to reduce or eliminate the likelihood of facial fracture patients being discharged from the emergency department with an undiagnosed intracranial haematoma.

The high proportion of males (73.7%) in our study was hardly surprising. During the period from March 1984 to January 1990, at the MetroHealth Medical Centre of Cleveland, 79% of the patients treated with facial fractures were male (*Haug* et al., 1990). This is only one example of the well known phenomenon of the male preponderance in facial trauma. The high proportion of sports-related injuries (37.3%) is quite unusual; in most comparable studies, vehicle accidents were the predominant cause of injury. The explanation could be the huge

number of injured skiers in our hospital located in the midst of the Alps, where skiing is one of the favourite leisure-time activities throughout the year. Nevertheless, traffic accidents were the most frequent cause of injury in intracranial haemorrhage patients. Obviously, in traffic accidents the CV is subjected more often to higher velocity and forces when compared with sporting accidents or assaults. This is supported by *Davidoff* et al. (1988) who found facial fracture victims to be at a significantly increased risk of CHI, when they sustain their trauma as a result of a traffic accident.

In this study, the orbital and zygomatic region had the largest proportion of the facial fractures. The anatomic position and composition (thickness of bone and hollow interiors) of the zygoma itself and the orbit make them more susceptible to fractures when compared with the mandible. *Hampson* (1995) reported in a review of biomechanical studies of facial injury, that the tolerance of the maxilla, nose and zygoma to energy forces is lower than that of the frontal and mandibular bones.

Among concomitant injuries, cervical spine injuries were found to be the most reliable associated predictor of intracranial bleeding. Although the general incidence of associated cervical spine injury in maxillofacial trauma victims is reported to be relatively low (0.2−6%), it is recognized that the combination of severe facial and cervical spine injuries has a high incidence of associated brain injury (*Merritt* and *Williams*, 1997; *O'Malley* and *Ross*, 1988; *Sinclair* et al., 1988; *Hackl* et al., 2001).

Based on the results of the univariate analysis, seven independent variables were selected for a multivariate logistic regression model. Highest ORs for accompanying intracranial haematomas were calculated for the occurrence of vomiting/nausea (OR 24.95, 95%CI 8.30−74.99) and seizures (OR 15.51, 95%CI 1.24−194.29). Other independent predictors included in the model were skull fracture, CHI, BS fracture, open brain injury and age. A number of authors have found predictors for intracranial injuries (*Miller* et al., 1996; *Fabbri* et al., 2004; *Da Dalt* et al., 2006). Loss of consciousness, altered mental status, amnesia, evidence of ICP, seizures, focal deficits, abnormal GCS score, and pupillary asymmetry are only a few of those reported in the literature. The only predictor common to nearly all the studies was the presence of neurological deficits (*Quayle* et al., 1997). However, our study sample and control group consist of alert, neurologically normal patients with facial fractures. The incidence of intracranial disorders in such patients has been reported as 3−7% (*Stiell* et al., 2001a). In total, we found a 2.8% incidence (54 of 1959 patients) of intracranial haematomas in alert, neurologically normal patients.

Vomiting was associated with an almost 25-fold risk of intracranial bleeding. This finding coincides with a review reporting on a 16-year experience with post-traumatic intracranial haematomas, in which vomiting (45.7%) was the most frequent immediate symptom, beside loss of consciousness (*Herrera* et al., 2000). Another study on extradural haematomas in children found recurrent vomiting in 70% of the cases (*Maggi* et al., 1998). The comprehensive Canadian CT Head Rule Study also identified vomiting as a high risk factor for neurosurgical intervention (*Stiell* et al., 2001a−*c*). The number of reports regarding seizures as a sign for intracranial injury is far lower. On the one hand, seizures seem to be very rare in head injury patients. We found a 0.6% incidence in our sample, which is responsible for the marginal significance and the large CI in the multivariate analysis. Most reports listed seizures among the category "neurological deficits" and did not provide an accurate incidence figure for seizures. However, the occurrence of seizures coincided with a 15-fold risk of intracranial bleeding. Lower ORs, but better comparability with the literature were found for skull fractures and closed head injuries (*Luce* et al., 1979; *Keenan* et al., 1999; *Tung* et al., 2000; *Stiell* et al., 2001b; *Fabbri* et al., 2004; *Sifri* et al., 2006). Both of these are already known as indicators of an elevated risk of acute traumatic intracranial haemorrhage at all ages.

Overall, the number of patients requiring neurosurgical procedures for decompression of intracranial haematoma was comparatively low. Only 20% of our intracranial haemorrhage patients had to undergo surgery because the majority of the haematomas in these conscious patients were small and did not result in raised ICP. This is supported by the rather short mean duration of stay in intensive care (4.4 $\pm$ 6.9 days) and a comparatively fair result outcome according to the GOS.

CONCLUSION

Significant brain injury cannot be excluded in patients with apparently minor head injury despite a GCS score of 15 and an unremarkable neurological examination on presentation in the emergency room. In this case−control study, thorough sound neurological examination failed to validate an intracranial haematoma in certain facial fracture patients. In 2.8% (54 of 1959 patients) intracranial haematomas in neurologically "normal" patients were observed. Predictors, in particular vomiting/nausea or seizures, in addition to skull fractures, can point to the possibility of an intracranial haemorrhage and have to be considered at all times. Our study does not support a limitation of CT scan use for patients with positive risk factors only, as this regime might fail to detect intracranial haematomas, thus potentially increasing complication and death rates in head trauma patients.

CONFLICT OF INTEREST STATEMENT

The authors do not have any financial or personal conflict of interest regarding the content of this article.

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Paper received 24 September 2007
Accepted 28 December 2007