Occipital Condyle Fractures

Nicholas Theodore, MD*
Bizhan Aarabi, MD, FRCSC‡
Sanjay S. Dhall, MD§
Daniel E. Gelb, MD¶
R. John Hurlbert, MD, PhD, FRCSC‖
Curtis J. Rozzelle, MD#
Timothy C. Ryken, MD, MS**
Beverly C. Walters, MD, MSc, FRCSC‡‡§§
Mark N. Hadley, MD§§

*Division of Neurological Surgery, Barrow Neurological Institute, Phoenix, Arizona; ‡Department of Neurosurgery, and ¶Department of Orthopaedics, University of Maryland, Baltimore, Maryland; §Department of Neurosurgery, Emory University, Atlanta, Georgia; ‖Department of Clinical Neurosciences, University of Calgary Spine Program, Faculty of Medicine, University of Calgary, Calgary, Alberta, Canada; #Division of Neurological Surgery, and §§Division of Neurological Surgery, Children's Hospital of Alabama, University of Alabama at Birmingham, Birmingham, Alabama; **Iowa Spine & Brain Institute, University of Iowa, Waterloo/Iowa City, Iowa; ‡‡Department of Neurosciences, Inova Health System, Falls Church, Virginia

Correspondence:
Mark N. Hadley, MD, FACS, UAB Division of Neurological Surgery, 510 – 20th Street South, FOT 1030, Birmingham, AL 35294-3410.
E-mail: mhadley@uabmc.edu

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RECOMMENDATIONS

Diagnostic:

Level II:

- Computed tomographic (CT) imaging is recommended to establish the diagnosis of occipital condyle fractures (OCFs).

Level III:

- Magnetic resonance imaging (MRI) is recommended to assess the integrity of the craniocervical ligaments.

Treatment:

Level III:

- External cervical immobilization is recommended for all types of OCFs. More rigid external immobilization in a halo vest device should be considered for bilateral OCF.
- Halo vest immobilization or occipitocervical stabilization and fusion are recommended for injuries with associated atlanto occipital ligamentous injury or evidence of instability.

RATIONALE

Acute traumatic OCF was first described by Bell in 1817. More frequent observations of this injury have been reported during the past 2 decades. Improvements in CT imaging technology and the use of CT imaging of head-injury patients that includes visualization of the craniovertebral junction have resulted in more frequent recognition of this injury. Despite this, acute traumatic OCF remains an infrequent occurrence.

An analysis of all of the reported cases of OCF in the scientific literature may facilitate development of diagnostic and treatment recommendations for this disorder and is undertaken in this review. Specific questions that were evaluated include: accuracy of plain radiographs and CT imaging in the diagnosis of OCF, and the safety and efficacy of various treatment strategies for OCF including no treatment, traction, external immobilization, and surgical decompression with internal fixation and fusion.

The guidelines author group of the Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons (AANS) and Congress of Neurological Surgeons (CNS) produced a medical evidence-based guideline on this topic in 2002.[1] The purpose of the current review is to update the medical evidence on the diagnosis and treatment of OCF since that early publication.

SEARCH CRITERIA

A National Library of Medicine (Pubmed) computerized literature search of publications from 1966 to 2011 was performed using the following headings: occipital bone and fracture (spinal, skull, or fracture alone), and led to 2105 and 71 182 citations, respectively. A subset of 235 citations contained both headings. The bibliographies of the identified articles were scanned to identify additional citations. The articles were reviewed using the following criteria for potential inclusion in diagnosis: human subjects, type of fracture, and tomographic or plain radiographic findings. The articles were separately considered for inclusion in treatment within the following parameters: human subjects, type of fracture, management, and outcome. The observations from all of the citations were combined because the usual methods for analysis were precluded by the infrequent occurrence of this injury type. Fifty-one articles met the selection criteria. All but 2 articles contained Class III medical evidence of either single case

studies or case series. The 2 exceptions were prospective studies to evaluate the use of clinical criteria in blunt trauma patients to prompt CT imaging of the skull base.[2,3] The duration of follow-up in the clinical articles ranged from not reported to 5 years. The data provided by these reports were compiled and make up the basis for this guideline. Summaries of the articles are provided in Evidentiary Table format (Table).

SCIENTIFIC FOUNDATION

OCFs have been classified by Anderson and Montesano[4] into 3 types: Type I (comminuted), Type II (extension of a linear basilar skull fracture), and Type III (avulsion of a fragment). The current literature review identified 415 patients reported to have OCF. Clinical series and case reports provided data on 84 Type I, 125 Type II, 207 Type III unilateral OC fractures, 37 bilateral OCFs, and 2 old fractures.

OCFs are relatively uncommon injuries. Various reports have estimated a 1% to 3% frequency of OCF in patients sustaining blunt craniocervical trauma.[5,6] Link et al described the results of craniocervical CT imaging on 202 consecutive acute blunt patients with a Glasgow Coma Score between 3 and 6.[3] OCF was identified in 9 of the 202 impaired patients they imaged (4.4%). In 1997, Bloom et al performed a prospective study, with 1 year follow-up to identify the frequency of OCF in patients meeting certain clinical criteria.[2] Fifty-five consecutive patients with high-energy blunt craniocervical trauma underwent thin-section craniocervical junction CT imaging. Supplemental criteria included reduced Glasgow Coma Scale (GCS) on admission, occipitocervical tenderness, reduced craniocervical motion, lower cranial nerve abnormality, and retropharyngeal soft tissue swelling (STS). Nine (16.4%) of 55 patients meeting their criteria following trauma were identified with OCF. In 2009, Malhan et al, determined that the incidence of OCF was 1.7/1000 trauma patients per year.

Diagnosis

Plain radiographs of the cervical spine were reported to have been performed in 359 patients culled from the literature review. These radiographs were reported as "normal" in 42 patients. Twenty-four patients had prevertebral STS.[7-10] Forty-eight OCF injury patients were reported to have sustained multiple cervical fractures. Associated fractures reported with OCF included fractures of the: atlas (n = 22); Type II odontoid (n = 7); axis (n = 14); isolated subaxial fractures (n = 13); and an unspecified cervical fracture (n = 1). Seven patients had atlantoaxial widening and 1 had a C5-6 subluxation without fracture.

Only 3 patients with OCF had their injury directly identified on plain radiographs of the skull or of the cervical spine.[11,12] The results of plain radiographs were not reported in 141 patients with OCF. The calculated sensitivity of plain radiographs from these reports aiding in the diagnosis of OCF is 1.4% (3 of 218). Since the data were obtained from case reports and case series of patients known to have OCF, comparison with the findings of plain radiographs in patients without OCF could not be accomplished. As a result, calculations of specificity, positive predictive value, and negative predictive value of plain x-rays for OCF could not be performed.

Three hundred sixty-three of the reported patients with OCF underwent CT imaging. Two patients had OCF diagnosed from retrospective review of CT images that were initially interpreted as normal. The diagnosis of OCF could be made in every patient with OCF on whom a CT scan was performed. The collective evidence (including several recent comparative studies) derived from the 363 cases identified in the literature review suggests that the sensitivity for CT imaging to depict OCF is 100%, and provides Class II medical evidence on this issue.

Only 59 patients with OCF have been reported to have been studied with MRI. Cervicomedullary hemorrhages were identified in 13 patients, 12 had a retrodental hemorrhage, 1 had a torn tectorial membrane, 4 identified the fracture (3 displaced), 24 had prevertebral or nuchal ligament edema and hemorrhage, and 5 patients had normal imaging. Acute MRI has been infrequently reported after OCF (11 described in various reports); therefore, no inferences about the role of early MRI in this setting can be offered. In 1997, Tuli et al proposed a new classification scheme using MRI to differentiate stable from unstable OCF (41). However, the case example they cited had concurrent atlantoaxial instability (rather than isolated atlanto-occipital instability).

Clinical clues suggesting OCF are variable and have not been consistently documented in the literature. Of the 415 patients with OCF described in the literature, there is detailed clinical information provided on only 119 patients. Loss of consciousness (poor GCS) was reported in 36 patients (30%). Thirty-five patients were reportedly normal (30%). Forty-eight had neurological deficits (40%), including acute or delayed cranial nerve deficits alone, cranial nerve deficits with limb weakness, mild to severe limb weakness without cranial nerve deficits, vertigo, hyperreflexia, and diplopia. Neck pain was reported as a consistent feature of OCF in responsive patients. Only 4 patients were described who did not complain of occipitocervical pain in the absence of significantly impaired consciousness.[8,10,13] Of these 4 patients, 1 was intoxicated, 1 had severe extremity injury pain, and the other 2 had severe facial trauma.

In summary, the diagnosis of OCF is rarely made on plain radiographs. Evaluation of the craniocervical junction utilizing multiplanar CT with reconstruction images is recommended to evaluate for the presence of OCF. Blunt trauma patients sustaining high-energy craniocervical injuries are more likely to sustain OCF. Consequently, cranial imaging of these patients should include CT imaging of the craniocervical junction. Clinical criteria including altered consciousness, occipital pain or tenderness, impaired craniocervical motion, lower cranial nerve paresis, and neurological deficits potentially referable to the proximal spinal cord should prompt CT imaging of the craniocervical junction to exclude OCF.

Treatment

Of the 415 patients with OCF reported in the literature, treatment information is offered on 259 patients. Follow-up of these patients ranged from not reported to 5 years duration. Forty-three OCF patients (2 Type I; 14 Type II; 5 Type III; and 22

unknown type) did not receive treatment. Nine of these patients (1 Type I; 4 Type II; and 4 Type III) on whom clinical follow-up was described developed cranial nerve deficits within days to weeks after injury.[6,14-21] One hypoglossal nerve palsy resolved, 2 hypoglossal nerve deficits improved, 3 other cranial nerve deficits persisted (2 hypoglossal, 1 glossopharyngeal and 1 vagal), and 3 outcomes were not reported. Six additional patients were identified in the literature with untreated OCF who developed delayed deficits or symptoms. Two of these initially untreated patients (1 Type II and 1 Type III) developed multiple lower cranial nerve deficits that reportedly improved with 6 weeks of cervical immobilization.[22] Another initially untreated patient (Type III) developed vertigo 3 months after injury that resolved after 8 weeks of collar immobilization.[23] One patient (Type III) developed nystagmus and a lateral rectus palsy after precautionary collar immobilization was discontinued. The deficit resolved after resuming cervical immobilization.[24] One patient (Type III) developed double vision during cervical traction, which resolved with surgical decompression.[21] Finally, 1 patient (Type III) developed delayed vagal, spinal accessory and hypoglossal nerve palsies during cervical immobilization in a cervical collar.[25] The cranial nerve X and XI palsies improved. However, the hypoglossal palsy persisted at 1 year.

One hundred ninety patients with OCF, including several patients with bilateral OCF injuries, were initially treated with cervical collar immobilization. Outcome following treatment was inconsistently reported. Sixty-eight with OCF treated with a collar on whom follow-up information was provided had complete recovery at last follow-up. Another patient had modest reduction of neck rotation after treatment. One patient had a hypoglossal nerve deficit at 50 months follow-up.[26] Two patients were described who had persistent mild dysphonia.[27] Three had persistent neck pain. Two of the 3 were unable return to work; however, 1 was reportedly without disability.[28]

Thirty-two patients with OCF were treated with halo/Minerva immobilization devices. One of those patients had slight improvement of Collet-Sicard syndrome at last follow-up.[26] Another was reported with persistent trapezius weakness.[27] Two other patients were reported to have chronic neck pain. Two patients were reported to have had a complete recovery at last follow-up.[28]

Of 37 patients with bilateral OCF reported in the literature, there is some treatment data on 29 patients. In the single largest series of these patients, Hanson et al described 22 patients with bilateral occipito-atlanto-axial injuries in a series of 95 OCF patients. Four of these patients died, 8 underwent occipitocervical fusion, and 4 were treated with halo immobilization. OCF patients in Hanson et al's series included patients with atlanto-occipital dislocation, precluding the ability to identify a treatment distinction between these 2 diagnoses.[29] In the remaining 7 bilateral OCF cases, there was 1 report of halo immobilization and 6 cases of conservative management, which was defined as either a rigid cervical orthosis or, "no specific treatment".[27,30] The patient who was treated with halo immobilization had a good outcome. In the conservatively treated group, there was 1 case of dysphonia at 2-year follow-up.

Seventeen patients with OCF were treated with surgery. Fourteen of them were treated with occipitocervical internal fixation and fusion (1 unknown type, 2 Type II and 11 type III OCF injuries). Three of these patients underwent surgery for decompression of the brainstem (1 type II and 2 type III injuries), in addition to internal fixation and fusion. Surgery was offered in the setting of craniocervical misalignment in 2 cases, associated C1 and C2 fracture in 1 case,[30] displaced fractures with ligamentous instability in another 3 cases,[10,31] and in 8 patients with bilateral or occipitoatlantal/atlantoaxial joint space widening in association with OCF.[29]

One patient with delayed diplopia had symptom resolution after removal of the fracture fragment,[21] while 1 patient with lower cranial nerve deficits[32] and 1 with diplopia and hemiparesis[23] remained unchanged several days after surgery. One patient who was neurologically intact remained so following occipitocervical fusion.[31] There is no reported follow-up for the remainder of the OCF patients treated surgically.

In summary, 12 of 15 patients who developed delayed symptoms or deficits were not initially treated. Only 3 of these 12 patients were subsequently treated with cervical immobilization. All 3 improved. In comparison, only 3 of 6 patients demonstrated improvement in deficits without treatment. Only 1 patient (OCF Type III) developed a deficit during treatment that persisted (hypoglossal nerve palsy) despite collar use. Only 3 patients underwent surgery for decompression of the brainstem, 1 of whom had immediate and lasting improvement in symptoms post-operatively. Nonoperative treatment with external cervical immobilization is almost always sufficient to promote bony union/healing and recovery or cranial nerve deficit improvement in all types of OCF. Isolated bilateral OCF should prompt consideration of more rigid external immobilization. Patients shown to have unilateral or bilateral OCF associated with occipitoatlantal injury may require surgical stabilization (occipitocervical internal fixation and fusion), or halo-vest immobilization, depending upon the extent of the injury. It is important to note that the presence of OCF should raise the suspicion for the potential of associated atlanto-occipital dislocation; however, either of these 2 traumatic injuries may be present independently.

SUMMARY

OCF is an uncommon injury and requires CT imaging to establish the diagnosis. Patients sustaining high-energy blunt craniocervical trauma, particularly in the setting of loss of consciousness, impaired consciousness, occipitocervical pain or motion impairment, and lower cranial nerve deficits, should undergo CT imaging of the craniocervical junction. Untreated patients with OCF can develop lower cranial nerve deficits that usually recover or improve with external immobilization. Nonsurgical treatment with external cervical immobilization is sufficient in nearly all types of OCF. Bilateral OCF injuries should prompt consideration for more rigid external immobilization in a halo vest device. Surgical treatment (cranio-cervical internal fixation and fusion) may be indicated in patients with OCF who have overt instability, neural compression from displaced fracture fragments, or who have associated occipital-atlantal or atlanto-axial injuries.

TABLE. Evidentiary Table: Occipital Condyle Fractures

Citation	Evidence Class	Age	Sex	Type	Loc	Pain	Plain	CT	MR	Exam	Treatment	Outcome
Aulino et al,[33] *Emergency Radiology,* 2005	III	Avg. 30 y	53 M	I-21	Unrep	Unrep	Unrep	Unrep	Unrep	12%—CN palsies	Unrep	Unrep
			23 F	II-28								
				III-37								
Capuano et al,[27] *Acta Neurochir,* 2004	III	29 y	M	II	Unrep	Unrep	Unrep	B, +	Unrep	CN IX	8 wk collar	2 yr dysphonia
		39 y	M	III	Unrep	Unrep	Unrep	L, +	SAH	CN X-XII	12 wk collar	2 y normal
		17 y	M	III	Unrep	Unrep	Unrep	L, +	Brain injury	CN X-XII	12 wk collar	2 y normal
		14 y	M	III	Unrep	Unrep	Unrep	L, +	Extradural blood	CN X-XII	12 wk halo	18 mo CN XI
		73 y	F	II	Unrep	Unrep	Unrep	R, +	Unrep	CN IX, X	8 wk collar	2 y normal
		26 y	M	II	Unrep	Unrep	Unrep	R, +	Unrep	Unrep	8 wk collar	18 mo normal
		46 y	M	III	Unrep	Unrep	Unrep	L, +	Unrep	CN X-XII	12 wk collar	18 mo normal
		58 y	F	II	Unrep	Unrep	Unrep	R, +	Unrep	Unrep	8 wk collar	18 mo dysphonia
		32 y	M	I	Unrep	Unrep	Unrep	R, +	Unrep	CN IX, X	12 wk halo	18 mo normal
		35 y	F	III	Unrep	Unrep	Unrep	R, +	Unrep	Unrep	12 wk collar	18 mo normal
Hansonet al,[27] *AJR,* 2002	III	Mean: 33 y	64 M	I-3	Unrep	Unrep	Unrep	Unrep	Nuchal lig edema—73%.	Unrep	I : 2—Fusion/Halo	1 month after injury:
		31 y	F	II-23					Extradura/subdural blood—30%.		II : 2—Fusion/Halo	I: 66% normal
				III-69					Cord edema/ hemorrhage —24%.		3-skull base decomp	II: 52% normal
											III : 18-Fusion/Halo	III: 59% normal
Legroset al,[22] *J Trauma,* 2000	III	71 y	F	III	-	Unrep	Unrep	L, +	Epidural blood	Del CN VI, VII, X	6 wk collar	18 mo CN X
		44 y	M	II	-	Unrep	Unrep	R, +	Normal	Del CN VI, IX-XII	6 wk collar	3 mo CN X
Ideet al,[34] *J Neurosurg,* 1998	III	25 y	M	III	+	+	STS, C1fx	R, +	Tectorial membrane tear	Normal	10 wk collar	10 wk Normal
Demisch S et al,[17] *Clin Neurol Neurosurg,* 1998	III	45 y	F	II	Unrep	Unrep	Unrep	R, +	Fx	Del CN XII	None	1 y imp CN XII
Bloomet al,[2] *Clin Radiol,* 1997	II	21 y	M	III	Unrep	Unrep	STS,C6, C7 Fx	R, +	Unrep	Normal	>8 wk collar	Normal
		36 y	F	III	Unrep	Unrep	Unrep	L, +	Unrep	Normal	>8 wk collar	Pain
		15 y	F	I/I	Unrep	Unrep	Unrep	B, +	Unrep	Q-paresis	>8 wk collar	Imp Q-paresis

(Continues)

TABLE. ***Continued***

Citation	Evidence Class	Age	Sex	Type	Loc	Pain	Plain	CT	MR	Exam	Treatment	Outcome
		45 y	F	III/I	Unrep	Unrep	Unrep	B, +	Unrep	CN XII	>8 wk collar	Pain,CN XII
		22 y	F	II	Unrep	Unrep	Unrep	R, +	Unrep	Normal	>8 wk collar	Unrep
		21 y	M	I	Unrep	Unrep	STS,C1, C2, C5 Fx	R, +	Unrep	Normal	>8 wk collar	Unrep
		41 y	M	I	Unrep	Unrep	Unrep	R, +	Unrep	Normal	>8 wk collar	Normal
		6 y	F	II	Unrep	Unrep	Unrep	L, +	Unrep	Normal	>8 wk collar	Normal
		25 y	F	I	Unrep	Unrep	STS,C2 Fx	L, +	Unrep	Normal	>8 wk collar	Unrep
		20 y	M	I	Unrep	Unrep		R, +	Unrep	P-plegia	>8 wk collar	Unrep
Tuliet al,[10] *Neurosurgery,* 1997	III	64 y	F	III	Unrep	+	STS	R, +	None	Normal	12 wk collar	3 mo Normal
		69 y	F	III	Unrep	-	AAWide	L, +	Fx	M-paresis, CN VII	OC Fusion	Improved
		27 y	M	Old	Unrep	-	Normal	L, +	None	Normal	None	3 y Normal
Cottalorda et al,[35] *J Pediatr Orthop,* 1996	III	15 y	F	I	Unrep	+	Normal	R, +	None	Normal	7 wk Minerva, Traction, collar	4 mo Normal
Lam and Stratford,[36] *Can J Neurol Sci,* 1996	III	20 y	F	III	Unrep	Unrep	Normal	R, +	Contusion	Hpa, CN XII	3 mo Halo	5 y imp CN XII
Urculo et al,[20] *J Neurosurg,* 1996	III	62 y	M	III	Unrep	Unrep	Normal	R, +	Fx	Del CN I	None	6 mo same
Noble and Smoker,[6] *Am J Neuroradiol,* 1996	III	33 y	M	I	Unrep	Unrep	Unrep	?, +	None	Del CN XII	None	Unrep
		26 y	M	I	Unrep	Unrep	Unrep	?, +	None	GCS 15	None	Unrep
		16 y	M	II	Unrep	Unrep	Unrep	?, +	None	GCS 13	None	Unrep
		32 y	M	II	Unrep	Unrep	C2Fx	?, +	None	CN VII,XII	None	Unrep
		53 y	F	II	Unrep	Unrep	Unrep	?, +	None	GCS 8	None	Unrep
		47 y	F	II	Unrep	Unrep	Unrep	?, +	None	GCS 15	None	Unrep
		37 y	M	II	Unrep	Unrep	Unrep	?, +	None	GCS 8	None	Unrep
		11 y	M	II	Unrep	Unrep	Unrep	?, +	None	GCS 13	None	Unrep
		33 y	M	II	Unrep	Unrep	Unrep	?, +	None	GCS 15	None	Unrep
		23 y	M	II	Unrep	Unrep	Unrep	?, +	None	Unrep	Unrep	Unrep
		39 y	M	III	Unrep	Unrep	IIOdFx	?, +	None	CN VII	Halo	Unrep
		88 y	M	III	Unrep	Unrep	C1,II Fx	?, +	None	GCS15	Halo	Unrep
		29 y	M	III	Unrep	Unrep	Unrep	?, +	None	Unrep	Unrep	Unrep
		14 y	F	III	Unrep	Unrep	Unrep	?, +	None	GCS 11	Collar	Unrep
		17 y	F	III	Unrep	Unrep	Unrep	?, +	None	GCS 7	None	Unrep
Castling and Hicks,[15] *Br J Oral Maxillofacial Surg,* 1995	III	21 y	M	II	+	+	Normal	R, +	None	Del CN XII	None	2 y Normal
Emery et al,[37] *Eur Spine J,* 1995	III	26 y	M	III	Unrep	+	Normal	L, +	Fx	Hyperreflexic	Collar	4 mo Normal
Paley and Wood,[19] *Br J Oral Maxillofacial Surg,* 1995	III	21 y	M	III	Unrep	+	Normal	L, +	Normal	Del CN XII	None	6 mo imp CN XII
Stroobants et al,[38] *J Neurosurg,* 1994	III	27 y	M	III	-	+	Normal	R, +	None	Normal	10 wk collar	21 mo Normal
		12 y	F	III	-	+	C1 Fx	L, +	None	Normal	4 wk Minerva	Normal
Wasserbergand Bartlett,[21] *Neuroradiol,* 1994	III	39 y	M	III	+	Unrep	Normal	L, +	None	Del CN XII	None	CN XII

(Continues)

TABLE. ***Continued***

Citation	Evidence Class	Age	Sex	Type	Loc	Pain	Plain	CT	MR	Exam	Treatment	Outcome
		24 y	M	III	+	+	Normal	L, +	None	Del Diplopia	Traction, Decomp	Normal
		16 y	M	III	+	Unrep	Normal	R, +	None	Brain injury	Traction, collar	3 mo CN XII
		34 y	M	III	Unrep	Unrep	Normal	R, +	None	Unrep	Traction, halo	Unrep
Young et al,[39] *Neurosurgery,* 1994	III	26 y	F	III	+	Unrep	Normal	L, +	None	Hpa, CN IX-XII	12 wk halo	14 mo imp IX-XII
		20 y	M	III	+	Unrep	Normal	R, +	None	GCS7	Collar	1 yr Hpa
Mann and Cohen,[40] *Am J Radiol,* 1994	III	23 y	M	III	-	+	Normal	R, +	None	Normal	6 wk collar	Normal
Olsson and Kunz,[13] *Acta Radiologica,* 1994	III	43 y	M	III	Unrep	-	Normal	L, +	None	Normal	Collar	Normal
Sharma et al,[32] *Clin Neurol and Neurosurg,* 1993	III	35 y	M	II	Unrep	Unrep	Normal	L, +	None	CN IX,X	Decompression	3 mo imp IX, X
Massaro and Lanotte,[41] *Injury,* 1993	III	21 y	M	III	Unrep	Unrep	Normal	L, +	None	H-sensory, CN XII	8 wk Minerva	2 yr CN XII
Raila et al,[42] *Skeletal Radiol,* 1993	III	25 y	M	III	+	+	Normal	L, +	None	Normal	6 wk collar	Normal
		67 y	M	III	-	+	C1abnormal	L, +	None	Normal	collar	Normal
Bettini et al,[43] *Skeletal Radiol,* 1993	III	39 y	F	I	Unrep	+	C3 Fx	L, +	None	Normal	Unrep	Unrep
		24 y	M	II	+	Unrep	Normal	R, +	None	Coma	Unrep	Unrep
		21 y	F	III	+	Unrep	Unrep	?, +	Coma	Coma	Unrep	Unrep
		21 y	M	III/III	Unrep	+	Normal	B, +	None	Normal	Unrep	Unrep
Leventhal et al,[44] *Orthopaedics,* 1993	III	42 y	F	II	+	Unrep	Normal	L, +	None	CN VI,VII	3 mo collar	Unrep
		19 y	F	III	+	+	Normal	L, +	None	Normal	Collar	Unknown
		43 y	M	III	Unrep	+	C5 Fx	R, +	None	Normal	3 mo collar	Normal
		17 y	F	II	+	Unrep	L1 Fx	R, +	None	GCS 10	3 mo collar	Normal
		36 y	M	I	+	GCS 8	T1 Fx	R, +	None	GCS 8	3 mo halo	Normal
		17 y	M	I	+	GCS 4	Normal	R, +	None	GCS 4	3 mo collar	Normal
Mody and Morris,[9] *Injury,* 1992	III	21 y	M	III	+	Unrep	STS	L, +	None	Unrep	Traction, 6 wk collar	18 mo Normal
Bozboga et al,[23] *Spine,* 1992	III	34 y	F	III	+	+	Normal	L, +	None	L hpa, diplopia	Late decomp.	4 y Normal
		37 y	M	III	+	Unrep	Unrep	L, +	None	Del vertigo	8 wk collar	3 y Normal
Bridgman and McNab,[25] *Surg Neurol,* 1992	III	32 y	M	III	+	+	Normal	L, +	None	Del CN X-XII	Collar	1 y imp X-XII
Wani et al,[12] *J Trauma,* 1991	III	67 y	M	II	+	Unrep	+ cond Fx	L, None	None	CN IX-XII	None	CN IX-XII
Wessels,[45] *S Afr J Surg,* 1990	III	26 y	M	III	+	+	Unrep	R, +	None	CN VII	Collar	6 wk imp
		7 mo	M	II	+	Unrep	Unrep	L, +	None	V,VII	Collar	4 mo VII-XII
		27 y	M	II	+	Unrep	Unrep	R, +	None	VII	Collar	6 wk imp
Mariani,[8] *Ann Emerg Med,* 1990	III	30 y	M	III	+	-	STS	R, -	None	Normal	8 wk collar	Normal
Joneset al,[11] *Am J Neuroradiol,* 1990	III	43 y	M	III/III	+	Unrep	+ con Fx	B, +	Contusion	Q-plegia	OCF	4 wk Q-plegia
Desai et al,[24] *J Trauma,* 1990	III	33 y	M	III	-	+	Normal	L, +	None	6	Collar	4 mo normal

(Continues)

TABLE. ***Continued***

Citation	Evidence Class	Age	Sex	Type	Loc	Pain	Plain	CT	MR	Exam	Treatment	Outcome
Valaskatzis and Hammer,[46] *S African Med J,* 1990	III	19 y	M	III	+	+	Normal	R, +	None	Normal	6 wk collar	Normal
Orbay et al,[18] *Surg Neurol,* 1989	III	37 y	M	III	Unrep	+	Normal	L, + (tomo -)	None	Del CN XII	None	15 mo, CN XII
Savolaine et al,[47] *J Orthop Trauma,* 1989	III	71 y	F	III	+	+	Normal	R, +	None	Hplegia, CN VI	Traction, Halo	LM paresis
Anderson and Montessano,[4] *Spine,* 1988	III	3 y	M	I	+	Unrep	Normal	R, +	None	Uncon	Soft	24 mo normal
		18 y	F	III	+	Unrep	Normal	?, +	None	Unrep	Minerva	36 mo
		22 y	M	III	+	Unrep	Normal	R,Tomo	None	Uncon	Halo	12 mo normal
		23 y	M	III	+	Unrep	Normal	L, +	None	Uncon	Collar	Death
		25 y	M	III	+	Unrep	Normal	? Tomo	None	Unrep	Minerva	17 mo
		37 y	M	II	+	Unrep	Normal	L, +	None	Uncon	Collar	12 mo normal
Curri et al,[48] *J Neurosurg Sci,* 1988	III	16 y	F	III′	+	Unrep	Normal	R, +	None	Decerebrate	Collar	6 mo Unrep
Hashimoto et al,[49] *Neurosurgery,* 1988	III	71 y	M	II	-	Unrep	Normal	L, +	None	CN IX,X,XI,XII	None	6 mo CN IX,X, XI,XII
Deeb et al,[16] *J Computed Tomography,* 1988	III	25 y	F	II	Unrep	Unrep	Normal	Del L,+	None	12	None	Unrep
		66 y	F	Old	Unrep	+	None	Del L,+	Fx	Normal	None	Unrep
Spencer et al,[50] *Neurosurgery,* 1984	III	19 y	M	I	+	GCS8	Normal	L, +	None	GCS 8	Collar, Halo	BCN IX,X
Goldstein et al,[51] *Surg Neurol,* 1982	III	24 y	F	III	Unrep	+	C5-6 sublux	L,Tomo	None	Normal	2 mo collar	Normal
Harding-Smith et al,[7] *J Bone Joint Surg,* 1981	III	18 y	M	III	+	Unrep	STS	R,Tomo	None	Uncon	Collar	16 mo Normal
Bolender et al,[14] *Am J Radiol,* 1978	III	23 y	M	III	Unrep	Unrep	Normal	R,Tomo	None	CN IX,X	None	Unrep
		22 y	M	II	Unrep	Unrep	Normal	R,Tomo	None	Del VI,IX,X	None	Unrep

CT, computed tomography; MRI, magnetic resonance imaging; Unrep, unreported; Del, delayed; + done or positive; -, not done or negative; Fx, fracture; Imp, improvement; STS, soft tissue swelling; C, cervical; AA, atlantoaxial; Q quadric-; P, para; M, mono-; OC, occipital condyle; CN, cranial nerve; GSC, Glasgow Coma Scale; Od, odontoid; L, left; R, right; B, bilateral; tomo, tomography; Tr, traction; Uncon, unconfirmed; Hpa, hemiparesis; Cond, condylar; slx, subluxation.

KEY ISSUES FOR FUTURE INVESTIGATION

CT imaging with 3-dimensional reconstruction is essential for the diagnosis of OCF. MRI is useful for the diagnosis of ligamentous and other soft tissue injuries, including injuries of the spinal cord. Because OCF injuries remain relatively infrequent, cooperative retrospective collection of CT, MRI, and treatment and outcome data in patients with OCF is recommended.

Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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