The Diagnosis and Management of Traumatic Atlanto-occipital Dislocation Injuries

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RECOMMENDATIONS

Diagnostic

Level I

- Computed tomography (CT) imaging to determine the CCI (condyle-C1 interval) in pediatric patients with potential atlanto-occipital dislocation (AOD) is recommended.

Level III

- If there is clinical or radiographic suspicion of AOD, CT of the craniocervical junction is recommended. The CCI determined on CT has the highest diagnostic sensitivity and specificity for AOD among all radiodiagnostic indicators in pediatric patients. The utility of CCI in adult patients has not been reported.
- A lateral cervical radiograph is recommended for the diagnosis of AOD. If a radiological method for measurement is used to determine AOD on the lateral radiograph, the basion-axial interval-basion dental interval (BAI-BDI) method is recommended. The presence of upper cervical prevertebral soft tissue swelling (STS) on an otherwise non-diagnostic plain cervical radiograph should prompt CT imaging to rule out AOD.

ABBREVIATIONS: AOD, atlanto-occipital dislocation; **BAI-BDI,** basion-axial interval-basion dental interval; **CCI,** condyle-C1 interval; **STS,** soft tissue swelling; **TBI,** traumatic brain injury

Treatment

Level III

- Treatment with internal fixation and fusion using one of a variety of methods is recommended.
- Traction is not recommended in the management of patients with AOD, and is associated with a 10% risk of neurological deterioration.

RATIONALE

Although traumatic atlanto-occipital dislocation (AOD) was perceived to be an uncommon injury resulting in frequent death, improvements in the emergency management of the patient in the field, rapid transport, and better recognition have resulted in more survivors of AOD in the past 2 decades. Infrequent observation of patients with AOD and missed diagnoses may impair outcomes of patients with this unusual injury.[1] An assimilation of the reported experiences of clinicians evaluating and managing AOD in our scientific literature may facilitate development of diagnostic and treatment options for this traumatic disorder. 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.[2] The purpose of the current review is to update the medical evidence on the diagnosis and treatment of AOD since that early publication. Specific questions that were investigated include the sensitivity of plain radiographs, CT, and MRI in the diagnosis of AOD, as well as the safety and efficacy of various treatment modalities for AOD, including no treatment, traction, external immobilization, and internal fixation with fusion.

SEARCH CRITERIA

A National Library of Medicine computerized literature search of publications from 1966 to 2011 was performed using the following headings: "atlanto-occipital joint" and "dislocation." The search was limited to the English language and human studies. An exploded search of these headings led to 522 and 11 257 citations, respectively. A subset of 178 citations contained both headings. The references of the identified articles were reviewed to identify additional case reports. The articles were reviewed using the following criteria for inclusion in diagnosis: human survivors, type of traumatic atlanto-occipital dislocation, and plain radiographic findings. The articles were also reviewed using the following criteria for inclusion in treatment: human survivors, type of traumatic AOD, management, and outcome. The observations from the published reports were combined because the usual methods for analysis were precluded by the infrequent observation of this injury. The type of dislocation was classified according to Traynelis et al[3] into Type I (anterior), Type II (longitudinal), and Type III (posterior) dislocations. Lateral, rotational, and multi-directional dislocations that could not be classified into 1 of these 3 types were considered separately and are notated as "other Type." The duration of follow-up ranged from none reported to 4 years. Of the articles meeting the diagnostic selection criteria reported, 68 articles with 105 patients provided data on 38 Type I, 45 Type II, 4 Type III, and 18 other Types of AOD. Two of these articles[1,4] included 1 patient each from 2 previously published individual case reports.[5,6] Of the articles meeting the treatment selection criteria, 56 articles with 84 patients provided data on 31 Type I, 33 Type II, 4 Type III, and 16 other types of AOD. Two of these articles[1,4] included 1 patient each from 2 previously published individual case reports.[5,6] The information provided by these reports was compiled and scrutinized and make up the basis for this guideline. Summaries of these reports are provided in Evidentiary Table format (Tables 1-2).

SCIENTIFIC FOUNDATION

Diagnosis

A variety of radiographic and descriptive features have been proposed for the diagnosis of AOD (Table 1). Initially, the descriptive measurements were all based on lateral cervical radiographs.[7-12,70] A displacement of more than 10 mm between the basion and dens is considered abnormal by Wholey et al.[7] A ratio of the basion-posterior atlas arch distance divided by the opisthion-anterior atlas arch distance greater than one is considered abnormal by Powers et al.[8] A distance of more than 13 mm between the posterior mandible and anterior atlas, or 20 mm between the posterior mandible and dens are considered abnormal by Dublin et al.[9] Failure of a line from the basion to the axis spinolaminar junction to intersect C2, or a line from the opisthion to the posterior inferior corner of the body of the axis to intersect C1, are considered abnormal by Lee et al.[10] Finally, a displacement of more than 12 mm, or less than minus 4 mm between the basion and posterior C2 line, or a displacement of more than 12 mm from the basion to the dens (2 mm more than the Wholey recommendation) is considered abnormal by Harris et al.[11,12] A comparative study by Lee calculated the sensitivity for the Wholey method of 50%, a 33% sensitivity for the Power's ratio, and a 25% sensitivity for the Dublin method. The authors determined that their X-line method had a sensitivity of 75%,[10] although neither the Power's ratio nor X-line method could be applied in nearly half their patients. A comparative study reported by Harris et al found that the Power's ratio had a sensitivity of 60%, the Lee method a sensitivity of 20%, and the BAI-BDI method a sensitivity of 100% among those patients with AOD on whom the required landmarks could be identified on lateral cervical spine films.[12] Przybylski et al[1] reported failure to diagnose AOD in 2 of 5 patients with the Power's ratio, in 1 of 5 patients with the X-line method, and in 2 of 5 with the BAI-BDI method. No radiographic method reviewed has complete sensitivity. The BAI-BDI method proposed by Harris et al (which incorporates the basion-dens distance described by Wholey) is at present the most reliable means to diagnose AOD on a lateral cervical spine radiograph.[7]

Pang et al proposed the CCI as a sensitive diagnostic measurement of atlantooccipital dislocation as determined on CT imaging. They analyzed and compared CCI from sagittal and coronal reformatted CT images of the craniovertebral junction of 89 children without AOD and 16 children with AOD. They found the CCI to have sensitivity and specificity of 100% compared to "standard" tests on plain films that had sensitivity between 25% and 50% and specificity between 10% and 60%. They concluded that the CCI criterion has the highest diagnostic sensitivity and specificity for AOD among all radiographic methods. Their work provided Class I medical evidence for the diagnosis of AOD among pediatric patients.[13,14]

Horn et al attempted to determine whether magnetic resonance imaging (MRI) findings on short TI inversion recovery (STIR) sequences following acute trauma are predictive of cervical spinal instability. Abnormal soft-tissue (pre-vertebral or para-spinal) findings on MRI were correlated with those identified on CT and plain and dynamic cervical spine x-rays in an attempt to determine cervical stability in 314 trauma patients. They found that MRI is sensitive to soft-tissue injuries of the cervical spine. However, they concluded that when CT and cervical radiographs, including dynamic X-rays, detect no fractures or signs of instability, MRI does not assist in determining cervical stability. In this circumstance they reported that MRI findings may lead to unnecessary testing when not otherwise indicated.[15]

Many of the case reports and case series in the literature do not describe the radiographic/imaging method(s) used to diagnose AOD. Since the most sensitive method to identify AOD on a lateral cervical spine radiograph (the BAI-BDI) was proposed by Harris et al in 1994,[12] this method was not likely used for many of the determinations. When the BAI-BDI was applied retrospectively, a diagnosis was possible on the first lateral radiograph in 53 of 105 patients, (sensitivity = 0.505). Of the 53 patients with AOD in

whom the diagnosis was made on the first lateral radiograph, 4 were not stratified by type. Of the 49 remaining patients, there were 24 Type I, 23 Type II and 2 Type III dislocations. A second, late and consecutive lateral radiograph (11 cases), tomography (1 case), fluoroscopy (2 cases), CT (11 cases), CT in addition to lateral radiographs (3 cases), and MRI (6 cases) were required to establish the diagnosis of AOD in 34 of the 105 patients. Since these data were obtained from case reports and small case series, a determination of the accuracy of plain radiographs to identify AOD compared to patients without AOD could not be performed. As a result, specificity, positive/negative predictive values, and likelihood ratios cannot be discerned from the available literature.

Of the 15 patients in whom the diagnosis was missed on the initial plain radiographs, the initial neurological condition of 3 patients was not described.[16] Of the remaining 12 patients, 4 were neurologically normal (1 Type I, 1 Type III, 2 other type).[17,18] Two of those 4 patients originally reported as normal developed a monoparesis (1 Type 1, 1 other type).[19,20] Neither recovered completely. Eight of the remaining 12 patients had neurologic abnormalities from the outset, 5 of whom worsened. Four of the 5 transiently worsened, including 1 Type I injury patient with quadriparesis and Cranial Nerve IX, X, and XII palsies[21] who improved but was spastic at last follow-up. One patient with a Type I injury developed a hemiparesis that recovered.[22] One Type I injury patient who developed quadriparesis was hemiparetic at follow-up.[23] One lateral AOD patient with paraparesis and toricollis reportedly recovered at last follow-up.[24] One patient (Type I) with a monoparesis initially experienced permanent worsening and was quadriplegic at follow-up.[25]

The presence or absence of soft tissue swelling was described in half of the patients in whom plain spine films were obtained. The sensitivity of the presence of STS when AOD was confirmed by other radiographic means was determined to be 0.69 or 69% (43 of 62 cases). Although plain radiographs do not consistently and reliably identify AOD, the index of suspicion for the potential of AOD may be increased with the identification of prevertebral STS.

Acute craniocervical CT imaging was performed in 62 of the 105 patients with AOD. However, for 23 of the 62 patients studied by CT, the authors did not report whether AOD was diagnosed using this modality. The diagnosis of AOD was reportedly made by CT in 39 of 62 patients (sensitivity = 0.63). No CT abnormalities were reported in 15 of 62 patients. Twenty-eight patients with AOD studied with CT had hemorrhages (21 had craniocervical junction subarachnoid hemorrhage, 2 had paravertebral hemorrhage, 1 had a subdural hemorrhage, 2 had intraparenchymal cord hemorrhages, and 2 had spinal cord contusions). Craniocervical MRI was performed in 34 of 105 patients with AOD. The MRI findings were not reported for 4 of the 34 patients studied. The diagnosis of AOD could be made in 21 of 30 cases studied with MRI (sensitivity = 0.7, or 70%).

In summary, the diagnosis of AOD is often missed on spine plain radiographs, (sensitivity = 0.505), particularly in the circumstance of non-longitudinal AOD (non-Type II). Additional imaging of the craniovertebral junction with either CT or MRI is recommended in patients suspected of having AOD. Other imaging methods such as fluoroscopy, tomography, and myelography have been reported to confirm the diagnosis of AOD, particularly in the older literature, but accuracy data is not available nor can it be calculated. Neurological abnormalities including lower cranial nerve paresis (particularly cranial nerves VI, X, and XII), monoparesis, hemiparesis, quadriparesis, respiratory dysfunction including apnea, and complete high cervical cord motor deficits in the setting of normal plain spinal radiographs should prompt additional imaging with CT or MRI. The presence of prevertebral STS on plain radiographs and subarachnoid hemorrhage on CT at the craniovertebral junction should prompt consideration of the diagnosis of AOD. The CCI determined from CT images has the highest diagnostic specificity and sensitivity among imaging diagnostic criteria for AOD and should be employed when attempting to make a diagnosis of AOD.

Treatment

Various treatment including rigid immobilization and internal surgical fixation and fusion have been described in the treatment of AOD (Table 2). Of 84 patients in whom treatment data are reported, 13 did not receive initial treatment for AOD.[4,19,20,23-30,80] Six of 13 had Type I, 2 had Type II injuries and 5 had other type injuries. At last follow-up in this group of untreated patients, 2 died, 2 improved neurologically, 4 had unchanged deficits from presentation, and 5 worsened neurologically.[4,19,20,25,26,31] There were 3 untreated AOD patients who presented with quadriplegia.[26,31] One improved to quadriparesis at last follow-up; the 2 other remained quadriplegic. In summary, failure to treat AOD resulted in worsening in 7 of 13 patients (54%).

Of 21 patients with AOD initially treated with traction, 2 worsened transiently and developed worsening quadriparesis and CN VI deficits. Both had resolution of their CN VI deficits but had persistent quadriparesis at last follow-up. One patient had a Type II injury[32] and 1 patient had a rotational other type dislocation.[4] Four patients were initially normal and remained normal at follow-up.[33-36] The remaining 15 patients with AOD treated initially with traction experienced improved neurological function compared to their initial findings at last follow-up. The improvement in neurological function in these patients could not be attributed to the initial period of traction. Ten had Type I injuries, 5 had Type II injuries, 2 had Type III injuries, and 2 had other type dislocations. In total, 1 of 6 patients with Type II injuries and 1 of 3 patients with other type, translational injuries experienced neurological worsening with the use of craniocervical traction. Because the frequency of neurological deterioration with traction in the treatment of AOD is approximately 10%, 10 times higher than that for subaxial injuries, the use of traction is not recommended in patients with AOD.

Of 29 patients initially treated with external immobilization excluding traction, 17 were immobilized in anticipation of internal fixation and fusion and none worsened during the pre-surgical interval (5 Type I, 9 Type II, 3 other type).[1,4,36-48] Of the remaining 12 patients treated with external immobilization alone excluding

TABLE 1. Evidentiary Table: Imaging Diagnosis of Atlanto-Occipital Dislocation

Citation	Evidence Class	AOD Type	Diagnosis Made By	X-ray Findings	CT Findings	MRI Findings
Sweet et al,[51] *JNS: Spine,* 2010	III	II	CT	No mention STS	Basion and dens separation of 21 mm, +Dx	Ligament injury, Dx
Kleweno et al,[43] *Spine,* 2008	III	II	Plain X-ray, CT	BDI, +Dx	SAH, + Dx	SC contusion. + Dx.
Gautschi et al,[39] *Spinal Cord,* 2007	III	I	Plain X-ray	2 cm disarticulation, +Dx	Diagnosis	Complete transection of lower medulla, Dx
Bloom et al,[26] *Emerg Med Australia*, 2007	III	I	Plain X-ray	STS, Powers, +Dx	Anterior paravertebral hematoma	SC transection
Vera et al,[52] *Childs Nerv Syst,* 2007	III	II	CT	No mention STS	O-C1 asymmetry, + Dx	None performed
Pang et al,[14] *Neurosurgery,* 2007	I (for pediatric patients)	Not specified	CT	"standard" tests 25%-50% sensitivity, 10%-60% specificity	CCI 100% sensitivity, 100% specificity, +Dx	Multiple "clues" to injury on MRI, but no comparison to other modalities
McKenna et al,[53] *CJEM,* 2006	III	II	Plain X-ray	STS, BDI, +Dx	Unreported	None performed
Saveika et al,[54] *Am J Phys Med Rehabil,* 2006	III	I/II	CT	No mention STS	+Dx	None performed
Hamai et al,[41] *Spine,* 2006	III	I	Plain X-ray	STS, Powers, +Dx	BDI. + Dx	Ligamentous injury. Dx.
Feiz-Erfan et al,[55] *JNS: Spine,* 2005	III	I/II	CT	STS	+Dx	+Dx
Seibert et al,[47] *Acta Neurochir,* 2005	III	I/II	CT	STS	Distraction, +Dx	Dx
		I/II	Plain X-ray	superior subluxation, +Dx	+Dx	+Dx
Gregg et al,[40] *J Trauma*, 2005	III	I	CT	No mention STS	Anterior translation, +Dx	None performed
van de Pol et al,[48] *Spine,* 2005	III	I	Plain X-ray, CT	BDI, Powers+, +Dx	Posterior fossa hematoma, +Dx	Brainstem contusion. Ligamentous injury, +Dx
Payer et al,[45] *Neurosurg,* 2005	III	II	CT	No mention STS	CCI, + Dx	No SCI
Salinsky et al,[46] *Pediatr Neurosurg,* 2005	III	II	Plain X-ray, CT	BDI, +Dx	+Dx	Near total SC transection, +Dx
Gonzalez et al,[56] *JNS: Spine,* 2004	III	II	CT	STS	Widening space, + Dx	None performed
Labler et al,[29] *Eur Spine J,* 2004	III	II	CT	No mention STS	Widening space. + Dx	Ligament injury, Dx
		I	Plain X-ray	Powers, +Dx		Epidural
		II	MRI			Traumatic lesions
Brinkman et al,[57] *Am J Roentgenol,* 2003	III	II	Plain X-ray	BDI, +Dx	Unreported	None performed
Rose et al,[58] *Am J Surg,* 2003	III	II	Plain X-ray	BDI, +Dx	Hematoma	None performed
Bani et al,[37] *Spine,* 2003	III	I	Plain X-ray	Powers, +Dx	Normal	Normal
		II	Plain X-ray	Powers, +Dx	SAH	Medullary contusion, +Dx
Tomasini et al,[59] *Am J Emerg Med,* 2002	III	II	Clinical	Powers normal	Normal	Ischemia
		I	Plain X-ray	Downward displacement, +Dx	+Dx	None performed
Grabb et al,[60] *Pediatr Radiol,* 1999	III	I	Plain X-ray	STS, Powers, +Dx	Unreported	Part tear tectorial
		II	Plain X-ray	STS, Powers, +Dx	None performed	Tear Post. AOL
		II	MRI	STS, Powers	None performed	Part tear tectorial, +Dx
Naso et al,[61] *Neurosurg,* 1997	III	I/II	Plain X-ray	No mention STS, +Dx	Unreported	Delayed study

(Continues)

TABLE 1. ***Continued***

Citation	Evidence Class	AOD Type	Diagnosis Made By	X-ray Findings	CT Findings	MRI Findings
Sponseller et al,[20] *Spine,* 1997	III	I	Plain X-ray (missed)	No mention STS	None performed	None performed
		II	Plain X-ray	No mention STS, +Dx	Unreported	Brainstem contusion
Przybylski et al,[1] *Spine,* 1996	III	I	MRI	Powers/BDI/Xline	SAH, −Dx	BS contusion, +Dx
Pang et al,[5] *Neurosurg,* 1980	III	II	Plain X-ray (missed)	Power/BDI/Xline	SAH, +Dx	BS contusion, +Dx
		II	2nd plain X-ray	Power/BDI-,Xline, +Dx	SAH, +Dx	None performed
		I/Lateral	Plain X-ray (missed)	Power/BDI/Xline	Normal, Head only	None performed
		I/Lateral	Plain X-ray (missed)	Power/BDI/Xline	SAH, +Dx	None performed
Yamaguchi et al,[62] *Neurol Med Chir (Tokyo),* 1996	III	I	Plain X-ray	No mention STS, +Dx	SAH,+ tomo	BS Contusion,+Dx
Guigui et al,[63] *Eur Spine J,* 1995	III	I	Plain X-ray	STS, +Dx	+Dx	None performed
Ahuja et al,[16] *Surg Neurol,* 1994	III	I	Fluoroscopy	STS,Powers	SAH, unknown	None performed
		II	5 Plain X-ray (3 missed)	STS,Powers, +Dx	None performed	None performed
		II		STS,Powers	SAH, +Dx	None performed
		II		STS,Powers	SAH, unknown	None performed
		I/II		STS,Powers	None performed	None performed
		I/II		STS,Powers	SAH, +Dx	None performed
Donahue et al,[38] *Pediatr Neurosurg,* 1994	III	I	Plain X-ray	STS, +Dx	None performed	None performed
		II	Plain X-ray	STS, 5 mm distract, +Dx	None performed	None performed
		II	Plain X-ray	STS, +Dx	None performed	None performed
		II	Plain X-ray	6 mm distract, +Dx	Intracerebral bleed	None performed
Palmer et al,[32] *J Trauma,* 1994	III	II	CT	No mention STS	Unreported, +Dx	CordContusion,+Dx
Dickman et al,[4] *J Spinal Disord,* 1993	III	II	Plain X-ray	15 mm distraction, +Dx	None performed	None performed
Papadopoulos et al,[6] *Neurosurg,* 1991	III	Rotatory	CT	STS	+ Dx	None performed
		Rotatory	MRI	STS	No blood, −Dx	Epidural, +Dx
		II/Rotatory	2nd Plain X-ray	STS, +Dx	+Dx	Epidural, +Dx
Harmanli et al,[64] *Surg Neurol,* 1993	III	II	Plain X-ray	No mention STS, +Dx	None performed	−Dx
Hosono et al,[22] *Spine,* 1993	III	I	Plain X-ray (missed)	STS	Edema, head only	Delayed study
Matava et al,[65] *Spine,* 1993	III	II	Plain X-ray	STS, +Dx	Delayed study	None performed
		II	Plain X-ray	No mention STS, +Dx	None, +DX	None performed
		II	Plain X-ray	No mention STS, +Dx	SAH, +DX	BS Contusion
Nischal et al,[44] *Br J Neurosurg,* 1993	III	II	Plain X-ray	STS, +Dx	BS contusion, +Dx	None performed
		II	Plain X-ray	STS, +Dx	−Dx	None performed
Bundschuh et al,[66] *Spine,* 1992	III	I	Plain X-ray	STS, +Dx	SAH, +Dx	SAH, + Dx
		I	Plain X-ray	STS, Power/Xline, +Dx	SAH	−Dx

(Continues)

TABLE 1. *Continued*

Citation	Evidence Class	AOD Type	Diagnosis Made By	X-ray Findings	CT Findings	MRI Findings
Farley et al,[67] *Spine,* 1992	III	I	Plain X-ray	STS, Powers, +Dx	None performed	Cord contusion
Belzberg et al,[68] *J Neurosurg,* 1991	III	II	2nd Plain X-ray	STS, +Dx	SAH, +Dx	None performed
Hladky et al,[69] *Neurochirurgie,* 1991	III	II	MRI	No mention STS	Contusion,head only	+ Dx
		II	MRI	No STS	Normal, Head only	+ Dx
Lee et al,[36] *J Trauma,* 1991	III	II	Plain X-ray	STS, +Dx	SAH, +Dx	None performed
		I/Rotatory	Plain X-ray	STS, +Dx	+ Dx	None performed
Maves et al,[70] *Pediatr Radiol,* 1991	III	II	Plain X-ray	No mention STS, +Dx	None performed	None performed
		II	Plain X-ray	No mention STS	None performed	None performed
		III	Plain X-ray	No mention STS, +Dx	None performed	None performed
Montane et al,[71] *Spine,* 1991	III	I	Plain X-ray	STS, +Dx	None performed	None performed
		II	2nd Plain X-ray	STS, +Dx	None performed	None performed
		II	2nd Plain X-ray	No STS, +Dx	None performed	None performed
DiBenedetto et al,[21] *Spine*, 1990	III	I	Plain X-ray (missed)	STS	ICH, +DX	None performed
Jones et al,[72] *Am J Neuroradiol,* 1990	III	I	Plain X-ray	No mention STS, +Dx	+DX	Premedullary edema
Colnet et al,[27] *Neurochirurgie,* 1989	III	Lat/rotatory	Tomography	Late study	SAH, +Dx	Delayed study
Jevtich,[18] *Spine,* 1989	III	Lateral	Plain X-ray (missed)	No mention STS	Delayed study	None performed
Hummel et al,[73] *Unfallchirurgie,* 1988	III	I	2nd Plain X-ray	No mention STS, +Dx	Subdural, Head only	None performed
Zampella et al,[31] *Neurosurg,* 1988	III	II	Plain X-ray	No mention STS, +Dx	SAH, Head only	Delayed study
Georgopoulos et al,[28] *J Bone Joint Surg Am,* 1987	III	I	Cineradiography	No mention STS	Delayed study	None performed
Bools et al,[34] *Am J Neuroradiol,* 1986	III	I	Plain X-ray	STS, +Dx	SAH, +DX	None performed
		III	2nd Plain X-ray	No mention STS, +Dx	None performed	None performed
Collalto et al,[19] *J Bone Joint Surg Am,* 1986	III	I/lateral	Plain X-ray (missed)	No STS	SAH, Head only	Delayed study
Putnam et al,[74] *J Am Osteopath Assoc,* 1986	III	I	Plain X-ray	STS, Powers, +Dx	SAH, +Dx	None performed
Ramsay et al,[23] *Injury,* 1986	III	I	Plain X-ray (missed)	No mention STS	None performed	None performed
Roy-Camille et al,[30] *Rev Chir Orthop Reparatrice Appar Mot,* 1986	III	I	Late Plain X-ray	No mention STS, +Dx	Delayed study	None performed
		I	Plain X-ray	STS, +Dx	None performed	None performed
Zigler et al,[75] *Spine*, 1986	III	I	Plain X-ray	No mention STS, +Dx	None performed	None performed
Watridge et al,[24] *Neurosurg,* 1985	III	Lateral	Plain X-ray (missed)	No STS	Delayed study	None performed

(Continues)

TABLE 1. ***Continued***

Citation	Evidence Class	AOD Type	Diagnosis Made By	X-ray Findings	CT Findings	MRI Findings
Banna et al,[33] *J Bone Joint Surg Am,* 1983	III	Rotatory	Plain X-ray	No mention STS, +Dx	+ Dx	None performed
Kaufman et al,[42] *Am J Neuroradiol,* 1982	III	II	Plain X-ray	STS, +Dx	None performed	None performed
		II	Plain X-ray	STS, +Dx	None performed	None performed
Woodring et al,[25] *Am J Roentgenol,* 1981	III	I	Plain X-ray	No mention STS, +Dx	None performed	None performed
		I	Plain X-ray (missed)	STS	None performed	None performed
Powers et al,[8] *Neurosurg,* 1979	III	I	Plain X-ray	Late study, +Dx	None performed	None performed
		II	2nd Plain X-ray	No mention STS, +Dx	None performed	None performed
Rockswold et al,[76] *Minn Med,* 1979	III	II	Plain X-ray	No mention STS, +Dx	None performed	None performed
Eismont et al,[17] *J Bone Joint Surg Am,* 1978	III	III	Plain X-ray (missed)	No mention STS	None performed	None performed
Fruin et al,[77] *J Neurosurg,* 1977	III	I	Plain X-ray	No mention STS, +Dx	None performed	None performed
Page et al,[78] *J Neurosurg,* 1973	III	I	Plain X-ray	STS, +Dx	None performed	None performed
Evarts,[79] *J Bone Joint Surg Am,* 1970	III	I	Plain X-ray	No mention STS, +Dx	None performed	None performed
Gabrielsen et al,[80] *Am J Roentgenol Radium Ther Nucl Med,* 1966	III	I	2nd Plain X-ray	STS, +Dx	None performed	None performed
Farthing,[35] *NC Med J*, 1948	III	III	Plain X-ray	No mention STS, +Dx	None performed	None performed

One patient was eliminated because the plain radiograph interpretation was not reported. Ferrara (1).
Two articles (11 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopolous (2/3), Hladky (1/3), Naso (1/2), Sun (6/6).
One article (5 patients) was eliminated because individual patient data was not reported. Bulas (5/5).
AOD, atlanto-occipital dislocation; MRI, magnetic resonance imaging; CT, computed tomography; STS, soft tissue swelling; BDI, basion-dental interval; SAH, subarachnoid hemorrhage; Dx, diagnosis; BS, brainstem; ICH, intracerebral hemorrhage; AOL, atlanto-occipital ligament.

TABLE 2. Evidentiary Table: Treatment of Atlanto-Occipital Dislocation

Citation	Evidence Class	AOD Type	Initial Exam	Treatment	Outcome
Sweet et al,[51] *JNS: Spine,* 2010	III	II	Quadriparesis, CN6	Fusion	Quadriparesis
Kleweno et al,[43] *Spine,* 2008	III	II	ASIA A	Halo, fusion	ASIA A
Bloom et al,[26] *Emerg Med Australia,* 2007	III	I	Quadriplegia	None	Quadriplegia
Gautschi et al,[39] *Spinal Cord,* 2007	III	I	Quadriplegia	Collar + fusion	Quadriplegia
Pang et al,[14] *Neurosurgery,* 2007	III	Not specified	12/15 ASIA A-C quad, 3/15 ASIA D	Halo, Fusion	10/15 ASIA D & E, 2/15 ASIA C, 3/15 ASIA A (2 late death), 15/15 radiographic fusion
Saveika et al,[54] *Am J Phys Med Rehabil,* 2006	III	I/II	Unreported	Fusion	Tetraplegia
McKenna et al,[53] *CJEM,* 2006	III	II	Unreported	Fusion	Gradual improvement
Hamai et al,[41] *Spine,* 2006	III	II	Quadriparesis	Halo, Fusion	Quadriparesis
Salinsky et al,[46] *Pediatr Neurosurg,* 2005	III	II	Quadriplegia	Halo, Fusion	Quadriplegia
van de Pol et al,[48] *Spine,* 2005	III	I	Unreported	Halo, Fusion	Wheelchair. Legs spasticity
Payer et al,[45] *Neurosurg,* 2005	III	II	Quadriparesis	Brace + Fusion	Full recovery 12 mos
Feiz-Erfan et al,[55] *JNS: Spine,* 2005	III	I/II	Normal	Fusion	Normal
Seibert et al,[47] *Acta Neurochir,* 2005	III	I/II	Normal	Fusion + collar	Normal
Gregg et al,[40] *J Trauma,* 2005	III	I	Quadriplegia	Fusion + halo	Quadriplegia
Gonzalez et al,[56] *JNS: Spine,* 2004	III	II	Unreported	Fusion	Normal
Labler et al, *Eur Spine J,* 2004[29]	III	I	Normal	Fusion	Normal
		II	tetraparesis	Supportive	Death
		II	tetraparesis	Fusion	Normal
Bani et al,[37] *Spine,* 2003	III	I	Unreported	Halo + Fusion	Wheelchair dependant
Govender et al,[49] *J Bone Joint Surg Br,* 2003	III	III	Hemiparesis	Fusion	Normal
		I	CN VI/IX/X/XII	Fusion	Normal
		II	Quadriparesis, CN6	Fusion	Spasticity of lower limbs
		Other	Normal	Halo body jacket	Normal
Naso et al,[61] *Neurosurg,* 1997	III	Mixed I/II	Quadriplegia	Supportive	Death 5 weeks
Sponseller et al,[20] *Spine,* 1997	III	I	Normal	None (neuro worse), Traction, Fusion + Brace	Spastic, CN X
		II	Normal	Brace failed (6 weeks), Fusion	Normal
Przybylsk et al,[1] *Spine,* 1996	III	I	Quadriplegia	Collar + Fusion	Quadriplegia
Pang et al,[5] *Neurosurg,* 1980	III	II	Quadriplegia	Halo failed (22 weeks), Fusion	Quadriplegia
		II	Normal	Fusion + Collar	CN X
		Mixed I/ Lateral	Hemiplegia	Collar + Fusion	Monoparesis
		Mixed I/ Lateral	Quadriparesis, CN VI/VII/XII	Fusion + Collar	CN XII
Yamaguchi et al,[62] *Neurol Med Chir (Tokyo),* 1996	III	I	Quadriplegia, CN X/XI/XII	Brace failed (10 weeks), Fusion	Quadriplegia, CN X/XI/XII
Guigui et al,[63] *Eur Spine J,* 1995	III	I	Normal	Fusion + Brace	Normal

(Continues)

TABLE 2. ***Continued***

Citation	Evidence Class	AOD Type	Initial Exam	Treatment	Outcome
Donahue et al,[38] *Pediatr Neurosurg,* 1994	III	I	Hemiparesis	Halo distracted (temporary neurological worsening), Fusion	Hyperreflexic
		II	CNVI	Halo + Fusion	Normal
		II	Quadriplegia, CNVII/X	Collar/Traction + Fusion	Quadriparesis, CNVII/X
		II	Quadriparesis, CN III/VII	Fusion	Quadriparesis
Palmer et al,[32] *J Trauma,* 1994	III	II	Quadriparesis,CNVI	Traction (neurologically worse), Brace + Fusion	Quadriparesis
Dickman et al,[4] *J Spinal Disord,* 1993	III	II	Quadriplegia, CN IX/X	Brace	Unchanged (sepsis death at 3 months)
Papadopoulos et al,[6] *Neurosurg,* 1991	III	Rotatory	Quadriparesis,CNVI	Traction (neurologically worse), Fusion + Halo	Quadriparesis
		Rotatory	CNVI	None (neurologically worse), Fusion + Halo	Hemiparesis
		Mixed II/ Rotatory	Hemiparesis, CNIII III/VI	Halo + Fusion	Normal
Harmanli et al,[64] *Surg Neurol,* 1993	III	II	Hemiparesis, CNIII	Fusion + Brace	Normal
Hosono et al,[22] *Spine,* 1993	III	I	Hemiparesis	Brace(neurologically worse), Fusion + Brace	Normal
Matava et al,[65] *Spine,* 1993	III	II	Hemiplegia, CNVI.XII	Fusion + Brace	Spastic, CN VI
		II	Hemiparesis, CN VI	Fusion + Brace	Normal
		II	CN VI/IX/X	Fusion + Brace	Spastic
Nischal et al,[44] *Br J Neurosurg,* 1993	III	II	Quadriparesis, CN III/VI/IX/X	Brace + Fusion	Hemiparesis, CN III/VI/IX/X
		II	Quadriplegia, CN IX, X	Brace + Fusion	Hemiparesis
Bundschuh et al,[66] *Spine,* 1992	III	I	Quadriparesis CN VI/IX/X/XII	Traction + Fusion	CN VI/XII
Farley et al,[67] *Spine,* 1992	III	I	Quadriplegia, CN X	Traction + Brace	Quadriplegia
Belzberg et al,[68] *JNS,* 1991	III	II	Quadriparesis, CN VI/IX/X	Traction + Brace + Fusion	Monoparesis, CN VI
Lee et al,[36] *J Trauma,* 1991	III	II	Normal	Traction + Fusion	Normal
		Mixed I/Rot	CN VI	Brace + Fusion	CN6
Montane et al,[71] *Spine,* 1991	III	I	Hemiparesis	Fusion + Brace	Spastic
		II	Quadriparesis	Traction, Fusion + Brace	Normal
		II	Quadriplegia	Fusion + Brace	Quadriplegia
DiBenedetto et al,[21] *Spine,* 1990	III	I	Quadriparesis, CN IX/X/XII	Collar (neurologically worse, 6 weeks), Fusion + Brace	Spasticity
Colnet et al,[27] *Neurochirurgie,* 1989	III	Mixed lat/ rotatory	Hemiplegia, CN VI/IX/X	None (neurologically worse), Traction + Shunt + Decompression	Hemiparesis
Jevtich,[18] *Spine*, 1989	III	Lateral	Normal	Traction + Brace	Normal
Hummel et al,[73] *Unfallchirurgie,*1988	III	I	Hemiparesis	Fusion + Brace	Normal
Zampella et al,[31] *Neurosurg,* 1988	III	II	Quadriplegia, CN V-XII	None	Quadriplegia, CN VI
Georgopoulos et al,[28] *J Bone Joint Surg Am,* 1987	III	I	Normal	None (neurologically worse), Fusion + Brace	Normal
Bools et al,[34] *Am J Neuroradiol,* 1986	III	III	Normal	Traction, Fusion + Brace	Normal
Collalto et al,[19] *J Bone Joint Surg Am,* 1986	III	Mixed I/lateral	Normal	None (neurologically worse), Fusion + Brace	Monoparesis

(Continues)

TABLE 2. ***Continued***

Citation	Evidence Class	AOD Type	Initial Exam	Treatment	Outcome
Putnam et al,[74] *J Am Osteopath Assoc,* 1986	III	I	Quadriplegia, CN V	Brace	Death (sepsis 8 months)
Ramsay et al,[23] *Injury,* 1986	III	I	Quadriparesis	None (neurologically worse), Traction + Brace	Hemiplegia
Roy-Camille et al,[30] *Rev Chir Orthop Reparatrice Appar Mot,* 1986	III	I	CN VI, XI	None, Brace failed (3 months), Traction + Fusion	CN VI
		I	Quadriplegia, CN VI/IX-XII	Traction + Fusion	Quadriplegia
Zigler et al,[75] *Spine,* 1986	III	I	Quadriplegia, CN XI	Traction + Brace + Fusion	Quadriplegia
Watridge et al,[24] *Neurosurg,* 1985	III	Lateral	Paraparesis	None (neurologically worse), Traction + Fusion + Decompress + Brace	Normal
Banna et al,[33] *J Bone Joint Surg Am,* 1983	III	Rotatory	Normal	Traction (2 weeks)	Normal
Kaufman et al,[42] *Am J Neuroradiol,* 1982	III	II	Quadriplegia	Brace + Fusion	Quadriparesis, CN IX/X
		II	Monoparesis	Brace	Normal
Woodring et al,[25] *Am J Roentgenol,* 1981	III	I	Hemiparesis, CN VI	Traction	CN6
		I	Monoparesis	None (neurologically worse), Traction + Fusion	Quadriplegia
Powers et al,[8] *Neurosurg,* 1979	III	I	Hemiparesis, CN VI	Traction + Brace	Hemiparesis
		II	Hemiparesis, CN VII	Traction + Brace	Normal
Rockswold et al,[76] *Minn Med,* 1979	III	II	Hemiparesis, CN VI	Traction, Brace + Fusion	Ambulates
Eismont et al,[17] *J Bone Joint Surg Am,* 1978	III	III	Normal	Collar (neuro worse) Fusion + Brace	Normal
Fruin et al,[77] *Neurosurg,* 1977	III	I	Hemiparesis, CN VI/IX-XII	Traction + Fusion	CN VI/XI
Page et al,[78] *JNS,* 1973	III	I	Quadriplegia, CN X/XII	Traction, Brace failed + (5 mo), Fusion	Quadriparesis, CN X
Evarts,[79] *J Bone Joint Surg Am,* 1970	III	I	Hemiparesis, CN VI/IX/X/XII	Traction, Brace + Fusion	CN VI
Gabrielsen et al,[80] *Am J Roentgenol Radium Ther Neucl Med,* 1966	III	I	Hyperreflexia,,CN VI	Traction, Brace failed (3 mo), Fusion	Numb scalp
Farthing et al,[35] *NC Med J,* 1948	III	III	Normal	Traction + Brace	Normal

Three articles (15 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopolous (2/3), Bulas (5/5), Naso (1/2), Sun (6/6).
Two articles (8 patients) were eliminated because the initial exam was not reported. Grabb (3), Ahuja (5).
Two articles (6 patients) were eliminated because the treatment was not reported. Maves (3), Hladky (3).
One article (2 patients) was eliminated because the outcome was not reported. Jones (1), Bools (1/2).
CN, cranial nerve.

traction, 4 worsened transiently (3 Type I, 1 Type II).[17,21,22,38,49] All 4 of these patients subsequently underwent craniocervical fixation and fusion. Of the remaining 8 patients managed with external immobilization alone, 3 were unstable after 6 to 22 weeks of immobilization (1 Type I, 2 Type II). Of these 3 patients with persistent instability despite external immobilization, 2 presented with quadriplegia and 1 was neurologically normal. All 3 underwent internal fixation and fusion without change in their neurological condition at last follow-up. Only 5 patients with AOD described in the literature were successfully treated with external immobilization alone (1 Type I, 2 Type II, 2 other type dislocations). Since 7 of 12 (58%) patients managed with external immobilization either deteriorated neurologically or failed to achieve craniocervical stability without surgical internal fixation and fusion, treatment of AOD with external immobilization alone should be considered with caution.

There is only initial neurological examination data reported on 79 of 83 patients in the surgical treatment group. Of those 79 patients in whom the neurological exam at admission could be discerned, 16 (20.3%) were reportedly normal (4 Type I, 3 Type II, 3 Type III, 6 other type), 6 (7.6%) had cranial nerves deficits only, 14 (17.7%) had a hemiparesis, 2 had a monoparesis (2.5%), 1 (1.25%) had paraparesis, 16 were reported as quadriparetic (20.2%), 3 (3.8%) had hemiplegia, 1 (1.25%) had hyperreflexia but no motor deficit, and 20 were quadriplegic (25.3%). Seventeen patients with an initial paresis were reported to have completely recovered at last follow-up. Six patients with plegia improved to paresis. Eight patients with paresis and 13 patients with plegia had stable neurologic examinations at the last reported follow-up (no worse and no better).

Cranial nerve deficits appear to be common with AOD. Thirty-eight patients (48.1%) were reported to have CN deficits at presentation, including CN III (3 patients), CN V (1 patient), CN VI (26 patients), CN VII (5 patients), CN IX (12 patients), CN X (15 patients), CN XI (6 patients), and CN XII (11 patients). Twenty-four patients in whom follow-up was reported had complete resolution of their CN deficits, 9 had partial resolution, 5 had no change in their CN deficits, and 3 patients developed new CN deficits at last follow-up.

Finally, 29 patients described in the literature were treated with planned early craniocervical fusion with internal fixation. Only 1 patient worsened neurologically following surgery. This patient with a Type II injury was normal initially and developed a CN X deficit which persisted at follow-up.[1] All but 3 of the remaining 28 patients were reported to improve neurologically at last follow-up. Six had Type I, 17 had type II, 2 had Type III, and 4 had other type AOD injuries. None of the patients treated with craniocervical fusion and internal fixation were reported to have experienced late instability requiring reoperation or further treatment.

Recently, larger case series focusing on the diagnosis and treatment of AOD have been reported in the literature. Horn et al analyzed clinical and radiological factors that predict outcome and management in 33 patients treated at a single institution. Special attention was given to neurological injury at presentation and imaging factors that dictated/determined treatment. Screening cervical spine radiographs were initially obtained and thin-cut (2.5 mm) CT images were acquired thereafter. In addition, most patients underwent MRI imaging. Five patients with severe traumatic brain injury (TBI) received no treatment and died early in their hospital course. Of the remaining 28 patients, 23 underwent craniocervical fixation with fusion and 5 were treated nonoperatively with an external orthosis. Five other severely injured patients died, all of whom were treated surgically. Two died due to TBI, 3 others due to other multiple organ system injuries and medical co-morbidities. The 5 patients treated nonoperatively were managed in this fashion because they had no abnormalities identified on cervical CT images based on established criteria (Power's ratio, BDI, BAI-BDI, X-line methods), despite the presence of abnormal findings in the occipitoatlantal joints, tectorial membrane, alar ligaments, or cruciate ligaments on MRI. TBI at presentation correlated with a high mortality rate (7 of 33 patients). Five patients died from TBI without treatment. Two additional patients treated surgically died as a result of TBI. The authors concluded that the craniocervical junction in patients with CT-documented AOD is unstable and requires surgical fixation if they survive their initial injuries (particularly traumatic brain injuries) and resuscitation.[50]

Hosalkar et al described 16 pediatric patients with traumatic AOD. Eight of these 16 patients died on admission. Of the remaining 8, all were initially treated with halo immobilization. Three of the remaining 8 died due to severe TBI. Of the 5 surviving patients, 4 underwent craniocervical fixation with fusion and 1 was treated in a Minerva cast orthosis. At last follow-up, 1 patient was neurologically intact, 3 had mild hemi-paresis but were functional, and 1 patient was a ventilator-dependent quadriplegic. The authors concluded that early diagnosis, prompt intubation, and early immobilization of the neck and head with respect to the torso appeared to improve survival in young patients who survived their associated brain injuries.

Bellabarba et al analyzed potentially correctable causes of delayed diagnosis of AOD and treatment options in a retrospective evaluation of 17 consecutive AOD patients who survived their injuries. In 13 of their 17 patients, (76%), the diagnosis of AOD was delayed by a mean of 2 days (range 1-15 days). Five (38%) of these 13 patients suffered profound neurological deterioration before AOD was clinically recognized. Surgical stabilization was undertaken in all 17 patients. Only 1 patient deteriorated following surgery. The authors concluded that a delay in the diagnosis of AOD was associated with an increased likelihood of neurological deterioration. Craniocervical instability due to AOD was frequently missed/misdiagnosed with the use of standard lateral radiographs.

SUMMARY

AOD is an uncommon traumatic injury that can be difficult to diagnose and is frequently missed on initial lateral cervical spinal radiographs. AOD is often associated with severe traumatic brain injuries. Patients who survive AOD injuries often have neurological impairment including lower cranial nerve deficits, unilateral or bilateral weakness, or quadriplegia. Nearly 20% of patients with acute traumatic AOD will have a normal neurological examination on presentation. The lack of localizing physical/neurological

examination findings and/or global neurological deficits from severe brain injury may impede/hinder the diagnosis of AOD in patients with normal-appearing initial cervical radiographs. A high index of suspicion must be maintained in order to diagnose AOD. Prevertebral soft tissue swelling on a lateral cervical radiograph or craniocervical subarachnoid hemorrhage on axial CT images have been associated with AOD and should prompt consideration of the diagnosis. Additional imaging including CT and MRI may be required to confirm the diagnosis of AOD if plain radiographs are inadequate. The Condyle-C1 interval as determined on CT imaging has the highest diagnostic sensitivity and sensitivity for AOD among all other radiodiagnostic indicators.

All patients with AOD should be treated. Without treatment, nearly all patients developed neurological worsening, many of whom never fully recover. Treatment of AOD with traction is not recommended. Treatment with external immobilization has been used successfully in selected patients but has a high failure rate. Craniocervical fixation and fusion is recommended for the treatment of patients with acute traumatic AOD.

KEY ISSUES FOR FUTURE INVESTIGATION

Although the use of external immobilization for AOD was often associated with late instability, several patients achieved stability without operative management. The complimentary usage of CT imaging (with 3-dimensional reconstruction images for more precise measurement of the magnitude of displacement) and MRI (for differentiation of partial and complete ligament tears from stretch injuries) may be useful in identifying a subgroup of patients in whom craniocervical stability might be achieved with external immobilization alone. Long-term follow-up of both surgically and non-surgically treated patients with AOD will aid our understanding of the ideal treatment strategy for this unusual and potentially lethal injury.

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