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Release, reduction, and fixation of one-stage posterior approach for basilar invagination with irreducible atlantoaxial dislocation

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ABSTRACT

Purpose: We evaluate the efficacy, safety and indications of single stage posterior release, reduction, and fixation of basilar invagination (BI) with irreducible atlantoaxial dislocation (IAAD).

Materials and methods: Seventeen patients with BI and IAAD consecutively underwent one-stage release, reduction, and fixation by a posterior approach from July 2000 to June 2015 were followed up for at least 12 months. There were 8 males. Mean age was 56 35.2 ± 13.8 years (range 12–56). The clinical symptoms and signs of the patients were recorded. Pre- and postoperative imaging examinations were performed. Neurological function was assessed using the Japanese Orthopedic Association (JOA) and Ranawat scores.

Results: Average follow-up time was 47.4 months (12–97 months). The JOA score increased from preoperative 4–10 (8.06 ± 2.52) to postoperative 13–16 (15.20 ± 0.62). The preoperative Chamberlain line, McRae line, Wackenheim line, atlantodens interval, and cervico medullary angle were 12.52 ± 5.17 mm, 6.59 ± 3.04 mm, 6.96 ± 4.32 mm, 9.88 ± 1.93 mm, and $115.35 \pm 12.40^{\circ}$, respectively. The postoperative values were 2.0 ± 3.67 mm, -3.06 ± 1.85 mm, -1.76 ± 2.88 mm, 1.17 ± 1.18 mm, and $136.76 \pm 11.44^{\circ}$, respectively. **Conclusion:** One-stage release, reduction, and fixation for patients with BI and IAAD through a posterior approach is safe and efficient.

ARTICLE HISTORY

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KEYWORDS

Basilar invagination; irreducible atlantoaxial dislocation; posterior approach; release and reduction and fixation; one stage

Introduction

Basilar invagination (BI) is an anomaly of the craniovertebral junction (CVJ) and presents a series of symptoms and signs caused by compression to the cervicomedullary junction (CMJ), brainstem, spinal cord, and cerebellum. The compression is due to the intrusion of the odontoid process into the foramen magnum and cranial cavity. BI is often associated with other anomalies in the CVJ, such as Chiari malformation, syringomyelia, and occipitalization of the atlas.^{1–3}

According to the presence or lack of atlantoaxial dislocation (AAD), BI is categorized as dislocation type (type I, unstable type or slope-odontoid) and non-dislocation type (type II, stable or atlanto-occipital). This classification reflects the characteristics of BI and provides a basis for treatment.^{3,4} Dislocation-type BI can be primary or secondary and the former is the most frequent; this type is characterized by atlantoaxial horizontal and/or vertical instability or dislocation. The odontoid process shifts into the foramen magnum and cranial cavity posterosuperiorly and over the Chamberlain line (CL), McRae line (ML), and Wackenheim line (WL). This shift results in progressive CMJ compression. Non-dislocation-type BI is mainly caused by secondary factors (osteoarthritis, osteogenesis imperfecta, rickets, osteomalacia, rheumatoid arthritis or hyperparathyroidism etc.) and is characterized by the upward movement and entire

invagination of the skull base and atlantoaxial vertebra around the foramen magnum. The odontoid process is above the CL and under the ML and WL. This BI type does not combine with AAD and seldom requires surgery because of the lack of clinical symptoms and signs.

AAD is classified as reducible AAD (RAAD), irreducible AAD (IAAD), and fixed or bony AAD (FAAD or BAAD, respectively).5-7 RAAD can be reduced under dynamic X-ray or skull traction, which only requires posterior fixation and fusion to maintain reduction. FAAD requires local decompression or decompression and stability because bone fusion cannot be reduced. The definition of IAAD remains unclear, and this type is mainly determined based on whether the dislocation can be reduced through skull traction preoperatively or under general anesthesia. Wang et al.⁶ believed that RAAD could be completely reduced and that IAAD could be incompletely reduced under traction. Yin et al.⁵ speculated that IAAD is AAD that could not be reduced under skull or occipitocervical bidirectional traction. At present, most authors believe that IAAD cannot be significantly reduced using large-weight skull traction preoperatively or under general anesthesia and requires the release of the atlantoaxial joint to be reduced. Therefore, IAAD may include AAD with from no to incomplete reduction under large-weight skull traction. Furthermore, the treatment of BI with IAAD remains

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Table 1. Presenting symptoms and signs in 17 patients with BI and IAAD.

	J	
Symptoms and signs	No. of	patients (%)
Head and neck pain	15	(88.2%)
Restricted neck movement	13	(76.5%)
Brevicollis or lower hairline	9	(52.9%)
Torticollis	12	(70.6%)
Numbness of limbs	14	(82.4%)
Quadriparesis	13	(76.5%)
Tendon hyperreflexia	16	(94.1%)
Ataxia	13	(76.5%)

Note. One patient may have more than 1 clinical feature.

controversial: whether or not anterior or posterior release or combined anterior release with posterior fixation and fusion is the most suitable. In summary, although the diagnosis and treatment of RAAD and FAAD are clear, those of IAAD have not been elucidated yet. In recent years, the advances in surgical techniques and the development of internal fixation devices have led surgeons to investigate direct posterior distraction reduction and fixation for dislocation-type BI.^{1,8-15} Several researchers believe that the technique does not need preoperative or intraoperative traction to determine whether AAD is reduced or not and that IAAD could be transferred to RAAD using certain intraoperative manipulations and directly exerting stress on the atlantoaxial joint.^{1,9,12,14} Hence, simple posterior surgery could be performed directly for BI with IAAD, and few patients may need anterior or anterior combined with posterior approach for surgery.

This study aims to (1) evaluate the feasibility, safety, and efficacy of the simple posterior approach for release, reduction, and fixation for BI with IAAD and describe IAAD; (2) clarify the treatment strategy of BI with IAAD and indications of the simple posterior approach for release, reduction, and fixation for BI with IAAD; and (3) investigate the key techniquesand advantages of the one-stage approach for the treatment of BI with IAAD.

Materials and methods

Inclusion criteria

The inclusion criteria were: (1) For BI, the odontoid process 5 mm higher than the CL or lower than the ML and WL. Or (2) For IAAD, the atlantodens interval (ADI) exceeds 3 mm in adults or 5 mm in children, AAD is observed in the lateral mass joint sagittal reconstruction of computed tomography (CT), and no significant change in AAD is observed under anesthesia or preoperative large-weight skull traction (1/6-1/5) of the body weight (BW)). And (3) a one-stage posterior approach was performed for release, reduction, and fixation for BI with IAAD.

follow-up.

final

Associated imaging evaluation of preoperation and

Table 2.

Exclusion criteria

The exclusion criteria were: (1) occipitocervical abnormalities caused by tumor, tuberculosis, or inflammatory diseases; (2) clinical symptoms and signs inconsistent with imaging findings; and (3) presence of surgical contraindications.

Clinical data

From July 2000 to June 2015, 52 cases with BI were treated in the First Affiliated Hospital of Xinjiang Medical University. Thirty-seven of these cases had AAD. Seventeen patients with BI and IAAD were included in the study based on the inclusion and

			J	CL (mm)	WL	WL (mm)	ML	ML (mm)	ADI	ADI (mm)	Ū	CMA (°)	Cage (mm)		Follow-up	
Patient No.age (Patient No.age (years)Sex Associated radiologicalanomalies	logicalanomalies	Pre	Pos	Pre	Pos	Pre	Pos	Pre	Pos	Pre	Pos	Left	Right (months)	(months) Fuse or not
1 31	F AAD, C1A, KFS, DAOJ, Tor	OJ, Tor	7	-	2	-	4	-	10	0	118	130	1		97	Yes
2 50	M AAD, C1A		8	8	7	-	7	-	10	4	112	142	11	11	90	Yes
3 13	M AAD, KFS, Os		8	-	Ś	-2	4	-2	12	2	128	139		1	79	Yes
4 53	F AAD, C1A		6	2	Ś	~ -	2	-4	11	2	124	145	9	7	43	Yes
5 27	M AAD, C1A, KFS,C4–5 and (-5 and C6-7fusion1	4	-	7	-2	12	2	7	-	132	157	12	1	35	Yes
6 39	F AAD, C1A	,	7	6	11	-2	12	5	10	ε	122	140	6	10	23	Yes
7 33	M AAD, C1A, KFS	,	9	5	6	-	14	-	12	2	116	132	10	10	25	Yes
8 35	F AAD, C1A, KFS, CM		0	2	ŝ	-5	S	-2	6	0	107	143	8	6	17	Yes
9 44	M AAD, C1A	,	8	6	S	-2	12	2	10	-	110	116	12	12	15	Yes
10 14	M AAD, C1A, KFS		5	~ -	4	-6	2	-5	7	0	126	143		1	13	Yes
11 34	F AAD, C1A,Syr	,	8	0	10	-2	11	-	10	-	115	138	12	12	12	Yes
12 49	F AAD, C1A, Syr		9	0	11	-2	-	-5	13	0	134	152	I	I	19	Yes
13 38	M AAD, C1A, KFS, CM		8	0	9	-5	5	-4	8	0	100	134	12	12	89	Yes
14 25	M AAD, C1A		5	~ -	Ś	-6	2	-5	10	-	97	129	I	Ι	90	Yes
15 56	F AAD, C1A, CM	,	0	0	11	-2	9	-4	7	0	125	141	7	8	69	Yes
16 46	F AAD, C1A, KFS	,	9	0	S	-4	6	~ -	6	-	94	133	10	11	78	Yes
17 28	M AAD, C1A	,	8	2	6	-6	12	~ -	13	2	101	111	12	12	13	Yes
Mean±SD			2.52 ± 5.17 2.0 ±		$3.67 \ 6.59 \pm 3.04$		5 7.00 ± 4.3	32−1.76±2.	.88 9.88 ± 1.	93 1.17±1.	18115.35 ± 12	3.06 ± 1.85 $7.00 \pm 4.32 - 1.76 \pm 2.88$ 9.88 ± 1.93 $1.17 \pm 1.18115.35 \pm 12.40136.76 \pm 11.44$	44			
	<i>T</i> -value			10.52		13.84		13.14		19.62		8.97				
	<i>p</i> -value			< 0.01		< 0.01		< 0.01		< 0.01		< 0.01				
CL: Chamberlair malformation; S	CL: Chamberlain line; ML: McRae line; ML: Wackenheim line; ADI: atlantodens interval; CMA: cervicomedullary angle; M: male; F: female; AAD: atlantoaxial dislocation; C1a: C1 arch assimilated with occiput; CM: Chiari malformation; Syr: syringomyelia; KFS: Klippel-Feil syndrome with C2–C3 segmentation defect; OS: Osodontoideum; DAOJ: dysplasiaof atlantooccipital joint; Tor: torticollis;JOA: Japanese Orthopedic Association score; pre:	Wackenheim line; el-Feil syndrome v	ADI: atla vith C2–0	ntodens inte 23 segmenta	erval; CMA: tion defect	cervicomedu ; Os: Osodoni	llary angle; :oideum; D,	M: male; F: AOJ: dysplas	: female; AA iaof atlanto	D: atlantoax occipital joir	cial dislocatio nt; Tor: tortico	interval; CMA: cervicomedullary angle; M: male; F: female; AAD: atlantoaxial dislocation; C1A: C1 arch assimilated with occiput; CM: Chiari intation defect; 0s: 0sodontoideum; DAOJ: dysplasiaof atlantooccipital joint; Tor: torticollis;JOA: Japanese Orthopedic Association score; pre-	n assimila ese Ortho	ted with	n occiput ssociatior	CM: Chiari score; pre:

before surgery; post: after surgery

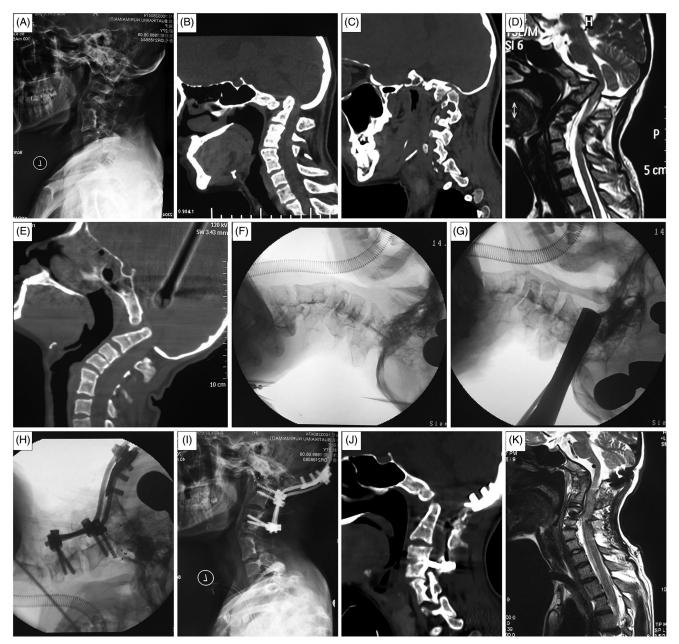


Figure 1. (A) 27-years-old female with BI,AAD and Klippel-Feil syndrome was treated by posterior atlantoaxial joint release, lever and distraction reduction, occipitocervical fixation. (A) Lateral X-ray showed BI; (B) CT median sagittal reconstruction presented AAD, assimilation of the atlas, and C2–C3, C4-5, and C6-7 fusion; (C) CT lateral sagittal reconstruction presented significant atlantoaxial joint dislocation; (D) Preoperative MRI median sagittal T2W1 showed odontoid process intrusion into the foramen magnum and compression to CMJ; (E) CT median sagittal reconstruction showed the partial reduction of the odontoid after a large weight of the skull traction (1/4BW); (F) Intraoperative fluoroscopy presented AAD partial reduction under anesthesia and large weight skull traction (1/3BW); (G,H) Intraoperative atlantoaxial joint release, lever reduction, implantation of 12 mm cage with autogenous bone into atlantoaxial facet joint and occipitocervical fixation was performed; (I) Postoperative lateral X-rays of the cervical spine showed good internal fixation position; (J) Postoperative CT median sagittal reconstruction showed odontoid process migration outside from the foramen magnum and full reduction of AAD; (K) Postoperative MRI median sagittal T2W1 showed the elimination of CMJ compression.

exclusion criteria. There were 8 males. Ages were from 12 years to 56 years $(35.2 \pm 13.8 \text{ years})$. The course of the disease was 3–36 months $(11.5 \pm 3.6 \text{ months})$. Two patients also had trauma. The clinical presentations are summarized in Table 1. Cervical cord function was assessed using the Japanese Orthopedic Association (JOA) score. The Ranawat grading scale was used to evaluate neurological function: I, no neurological dysfunction; II, subjective weakness, tendon hyperreflexia, and paresthesia; IIIA, objective weakness and pyramid sign, but walking without disability; and IIIB, quadriplegia and no walking. Preoperatively, the JOA score was 4–10 (8.06 ± 2.52) and the Ranawat score was categorized as II in 1 case, IIIA in 12 cases, and IIIB in 4 cases.

Anteroposterior, lateral, dynamic, and mouth-open radiography; CT scans with reconstruction views; and magnetic resonance imaging (MRI) of the CVJ were conducted on all patients preoperatively. CL, ML, WL, ADI, and cervicomedullary angle (CMA) were surveyed, and all measured data satisfied the diagnostic criteria of BI with AAD.^{1,3} The associated radiological presentations are summarized in Table 2.

Preoperative preparation

Under close monitoring, skull traction was performed, and the traction weight was gradually increased. The traction weight of

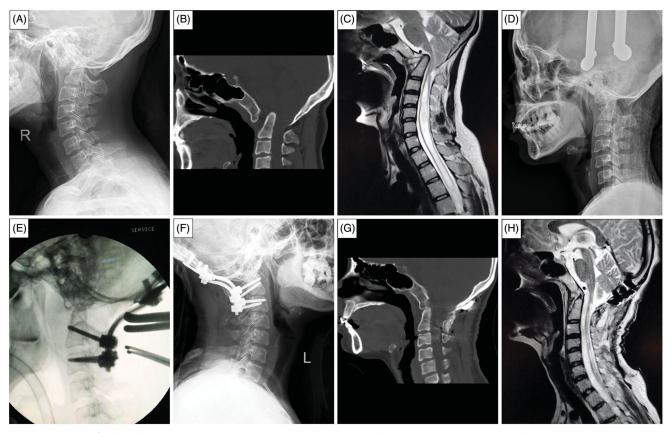


Figure 2. (A) 34-years-old female with BI, AAD and syringomyelia was treated by posterior atlantoaxial joint release, lever and distraction reduction, occipitocervical fixation. (A) Preoperative lateral X-ray showed BI; (B) Preoperative CTmedian sagittal reconstruction presented AAD, assimilation of the atlas; (C) Preoperative MRI median sagittal T2W1 showed odontoid process intrusion into the foramen magnum and compression to CMJ; (D) Preoperativelateral X-ray showed the partial reduction of the odontoid after a large weight of the skull traction (1/6 BW); (E) Intraoperative atlantoaxial joint release, lever reduction, implantation of 9 mm cage with autogenous bone into atlantoaxial facet joint and occipitocervical fixation was performed; (F) Postoperative lateral X-rays of the cervical spine showed good internal fixation position;(G) Postoperative CT median sagittal reconstruction showed odontoid process migration outside from the foramen magnum and full reduction of AAD; (K) Postoperative MRI median sagittal T2W1 showed the elimination of CMJ compression and disappearance of syringomyelia after surgery.

the group reached 1/5-1/6 of the BW. After 3-7 days of traction, the reduction of AAD was evaluated.

Selection of surgical methods

Under general anesthesia the head was placed in hyperextension with skull traction in the supine position. The initial traction weight of 5 kg was gradually increased, the maximum weight did not exceed 1/5 of the patient's weight (the best preoperative traction weight). If signs of nerve lesions emerge, then the traction weight was immediately reduced under intraoperative electrophysiological monitoring of spinal and cranial lesions. Traction was maintained for 30 min. If the dislocation changed significantly, then the patient was diagnosed with BI and RAAD, and posterior surgery was selected.¹³ If the reduction of dislocation is less than 50%, one-stage posterior approach is chosen for simple release, reduction and fixation (Figures 1 and 2). Otherwise, anterior surgery or release reduction was selected.

Once BI with IAAD was determined, the patient was turned over to the prone position; the patient's head was placed in the Mayfield head holder. The operative and postoperative care procedures are illustrated in Figure 1. No neuromonitoring changes were found in the 17 cases during traction and operation.

Follow-up

Cervical anteroposterior, lateral, and dynamic X-ray, MRI, or CT scanning were conducted to evaluate reduction, decompression,

and bone graft and internal fixation within 1–2 weeks and 1, 3, 6, and 12 months after surgery and annually thereafter. Neurological function was also assessed.

Statistical analysis

SPSS19.0 (SPSS, USA) was used for the statistical analysis. Continuous variables (i.e. preoperative and final follow-up radiology results and JOA score) were aggregated as the mean and standard deviation and analyzed using Student'sttest. Categorical variables (i.e. Ranawat score) were aggregated as proportions and analyzed using Fisher's exact test. Significance was set at p < 0.05 for all tests.

Results

The operation time was 90-210 min, with an average of 145 min. The bleeding volume was 150-350 mL, with an average of 175 mL. No intraoperative blood transfusions were needed. All patients were followed up for 12 months to 97 months, with an average of 47.4 months.

Radiographic findings

All patients exhibited good fixation and fusion (fusion rate = 100%) and did not experience screw loosening or implant failure.

Full reduction of AAD and normal alignment of the CVJ and cervical spine were accomplished for all cases. At the final follow-up, the patients showed an average CL of -3-9 mm (2.0±3.67 mm), WL of -1-6 mm (-3.06 ± 1.85 mm), ML of -5-5 mm (-1.76 ± 2.88 mm), ADI of 0-4 mm (1.17 ± 1.18 mm), and CMA of $111^{\circ}-157^{\circ}$ ($136.76^{\circ}\pm11.44^{\circ}$). These parameters were significantly improved at the final follow-up (p < 0.05; Table 2).

Neurologic recovery

Compared with in the preoperative period, the JOA score was 13-16 (15.20 ± 0.62) and the Ranawat score was grade I in 13 cases and grade II in 4 at final follow-up. This neurologic improvement was significant (p < 0.05).

Complication

Two patients had complications: wound infection in one case and CSF leakage in another. Both complications responded to treatment. On the premise of systemic application of sensitive antibiotics, the terminal cistern catheter was used for continuous external drainage for the leak. No complications related to vertebral artery injury were observed.

Discussion

Feasibility, safety, and efficacy of the simple posterior approach for the treatment of BI with IAAD

The application of posterior surgery in the treatment of BI with AAD has prompted us to explore the posterior approach for the treatment of BI with IAADfurther.^{1,5,6,8,13,14,16-26} This study serves as a continuation and has achieved good results (Table 2, Figures 1 and 2), indicating that this technique is viable, safe, and efficient.

What is IAAD?

A large number of studies have shown that accurate assessment of the reducibility of AAD is important in the treatment of BI, and traction may be the most effective assessment method.^{5,6,13,14,16–26} In the past, the reduction of AAD was assessed by preoperative dynamic X-ray or skull traction of conventional weight (1/10 of the BW). Although some authors advocating simple posterior surgery believe that all congenital AAD associated with BI is IAAD and that identifying RAAD or IAAD through traction is unnecessary,^{1,8} a majority of them advocate large-weight skull traction (1/6 of the BW) to assess the reducibility of AAD preoperation or postanesthesia.^{5,6,13,14,16–26} This method was applied in the present work, and only a few patients were subjected to traction at 1/3 of the BW.

However, the determination of IAAD is controversial. Wang *et al.*¹⁶ reported that AAD that can be fully reduced under traction is RAAD; otherwise, it is IAAD. Xia *et al.*¹⁷ stated that IAAD could not be reduced under skull or craniocervical traction. The controversy lies in how partial reduction of AAD is judged as IAAD or RAAD. Peng *et al.*¹⁵ proposed that when the tip of the odontoid process is lower than or close to the WL and ML and the ADI is significantly improved under traction, then AAD is RAAD; otherwise, it is IAAD. We propose that BI with AAD is RAAD if the improvement of ADI and CL, WL, and ML is equal to or greater than 50% under preoperative or

postanesthesia large-weight skull traction. By contrast, if ADI and CL, WL, and ML exhibit less than 50% improvement, then AAD is IAAD. According to this standard, the one-stage posterior approach for the treatment of BI with IAAD was selected in this series.

Treatment strategy of BI with IAAD and indications of onestage posterior approach for BI with IAAD

The treatment of BI with AAD mainly aims to relieve the compression of the CMJ, restore the normal alignment of the CVJ, and reconstruct stability.¹⁸ This objective can be achieved in patients with BI and AAD if alignment can be restored.^{1,3,8,9,12,14,15,18,27} At present, RAAD can be directly treated by posterior reduction, fixation, and fusion, whereas FAAD or BAAD requires the removal of the odontoid process. However, the treatment of IAAD remains controversial.

Achieving decompression by simple resection of the odontoid process and foramen magnum is not only difficult but may also damage the stability of the CVJ and lead to progression of the disease. Although posterior fixation and fusion can be performed, neurological improvement is not ideal because of the lack of full decompression or fusion. Given the comprehensive information on BI, researchers have reached a consensus on using reduction to treat patients with BI and IAAD. At present, treatments of patients with BI and IAAD include anterior, posterior, and anterior combined with posterior approaches.^{1,5,8,12–14,16–19}

The causes of difficult reduction of AAD include narrowed joint space; hyperosteogeny; hyperplasia and contracture of muscles, ligaments, and joint capsule; and anteroinferior tilt of the lateral mass of the axis.^{9,11,28} Anterior and anterior combined with posterior surgery can achieve thorough release and reduction for BI with IAAD through soft tissue release, osteophyte removal, and leverage and present favorable results.^{5,6,13,16,17,19-22,26,29} However, the single anterior approach requires effective implantation and pressure-side fixation of the plate screw;^{5,17,19,29} these processes may increase the risk of infection, dysphagia, fusion failure, and insufficient restoration of the alignment of the CVJ, and even the minimally invasive anterior approach may give the same results.²¹ Since Wang et al.¹⁶ first reported the use of combined anterior and posterior surgery for the treatment of BI with IAAD, authors have confirmed the effectiveness of this technique,^{6,13,16,20,26} particularly for severe and complex BI with IAAD. However, this technique presents several limitations, such as intraoperative changes in position, long operation time, high risk of complication, and increased hospital stay.6,13,16,20,26

Simple posterior surgery for patients with BI and IAAD has achieved favorable results.^{1,8,9,11–15,30} Chandra et al.⁸ used decompression-compression-extension reduction (DCER) for the treatment of BI and obtained 94% complete reduction of AAD and 100% patient satisfaction rate. Thus, DCER can effectively treat patients with BI and AAD. Several authors also reported that posterior reduction techniques can convert IAAD into RAAD using an intraoperative internal fixation device or relative handling on the atlantoaxial joint without traction.^{1,8,12,30} Posterior surgery is an important option in the treatment of BI with IAAD because of its simple and convenient operation and good reduction correction capability from modern surgical techniques and internal fixation devices. However, several authors are concerned about the lack of full reduction of IAAD, failure of internal fixation, and risks of fractures or neurovascular injury.^{1,8,12,16,30} Furthermore, BI with IAAD is common in

congenital dysplasia and is often accompanied by two basic pathological changes, namely, assimilation of the atlas and C2–C3 fusion.^{18,27,31} Both phenomena are not only the latent factors of AAD but also considered important factors of difficult reduction and may lead to progressive atlas anteroinferior displacement, tilt of the atlantoaxial articular facet, and "swan-neck" deformity. Degeneration of the atlantoaxial joint and surrounding tissue also limits reduction.^{9,11,28} Therefore, the more complicated the combined deformity, the more serious the dislocation, and the more severe the degeneration of the atlantoaxial joint, the more difficult is posterior reduction.

For BI with IAAD, another key factor that affects whether simple posterior surgery should be selected or not is the degree of horizontal and vertical dislocations of the atlantoaxial joint. Given that anteroinferior displacement of the atlas results in increased ADI and enlarged vertical atlanto-odontoid distance, ADI and CL, WL, and ML are commonly used to reflect the severity of the horizontal and vertical dislocations of the atlantoaxial joint, toaxial joint, respectively. In this series, ADI was 9.88 mm, CL was 12.52 mm, WL was 6.59 mm and ML was 6.96 mm; these values are similar to those in the literature.^{1,8,12,15,30} CL \leq 12 mm, WL \leq 6 mm, and ML \leq 6 mm served as the evaluation criteria for selecting posterior surgery for BI with IAAD; otherwise, anterior or anterior combined with posterior surgery should be considered.

Considering the present results and those in the literature, the author summarized the indications of simple posterior release, reduction, and internal fixation for the treatment of BI with IAAD: (1) AAD is improved by <50% under preoperative or postanesthesia traction; (2) the odontoid process is above the CL by ≤ 12 mm, WL by ≤ 6 mm, and ML by ≤ 6 mm, and the odontoid is close to the three lines after traction; (3) lack of bony barrier between atlas and odontoid process or anterior tilt malformation of the odontoid process tip; and (4) complicated dysplasia of the upper cervical spine.^{13,18,27}

The effects of patient age; traction time; bone density; and release, reduction, and fixation techniques on the reduction of the posterior approach should also be evaluated. In general, children, adolescents, or young patients are more likely to be reduced, and RAAD is common in these groups.^{13,20} Traction time varies widely, the common duration being 1–2 weeks. We used preoperative large-weight skull traction lasted for 3–7 days combined with postanesthesia traction. Peng *et al.*¹⁵ reported using preoperative large-weight skull traction for 15–30 days (average = 20 days). Lengthening the time of large-weight skull traction may help improve the reduction of IAAD.

Bone strength is also related to reduction and fixation. Many authors report that atlantoaxial distraction and compression reduction can be performed using an internal fixation device; hence, not only bone structures but also internal fixation must bear enormous stress, which easily leads to fracture and internal fixation failure.^{1,8,12,20,30}

Key techniques and advantages of the one-stage posterior approach for BI with IAAD

For BI with IAAD, horizontal and vertical dislocations must be addressed. In contrast to dislocation or spondylolisthesis of other parts, good horizontal reduction can be achieved only if the posterosuperiorly displaced odontoid process is removed from the foramen magnum or if the vertical dislocation of the fused anterior arch of atlas is restored; therefore, the key to the treatment is whether vertical dislocation is thoroughly corrected. Jian *et al.*¹

and Meng *et al.*¹⁴ used occipitocervical fixation devices for longitudinal and horizontal reductions of BI with IAAD by distraction. Chandra *et al.*⁸ reported that 35 cases of BI with IAAD were treated directly through posterior atlantoaxial intervertebral DCER under non-traction. Yin *et al.*¹² adopted a similar technique for the treatment of 174 patients with BI and IAAD and obtained satisfactory results. This series is the same. These findings prove the key role of correcting the vertical dislocation of AAD in the treatment of BI with IAAD.

We consider that multiaxial screw in the axis do not provide strong enough fixation; various stresses are mainly applied to the atlantoaxial joint, which may lead to fracture and internal fixation failure. Full longitudinal reduction is also difficult when the height of the intervertebral implants in the atlantoaxial joint is approximately 5-6 mm⁹. In this series, posterior surgery was selected, and a few key techniques were adopted to overcome these limitations, which included: (1) Sufficient soft tissue release. After the C1-C2 posterior capsular was incised, the detacher was inserted into the atlantoaxial lateral mass joint, which is levered to release toward the anterior and lateral sides until the joint capsule, ligament, and musclecontractures at the front were released. (2) Distraction among the segments. After release, the test mode was used to distract the intervertebral space step by step and obtain a space with height of at least 6 mm (6-12 mm, average $= 9.9 \,\mathrm{mm}$). (3) Atlantoaxial reduction. The atlantoaxial attachment was carefully stressed several times to test intersegmental mobility. Atlantoaxial reduction was observed under intervertebral and non-intervertebral distraction. The rod was pre-bent under the optimal reduction condition. The rod was fixed to multiaxial screws of C2-C3 or C2-C4, the attachment of which was pushed forward to reduce AAD. The proximal end of the rod was pressed into the occipital bone fixation device through the lever effect to complete horizontal and vertical reductions. (4) Structural strut grafting. Structural autologous iliac bone or cages were implanted into the atlantoaxial facet joints to prevent intersegmental collapse, which would result in failure of internal fixation.

Compared with single posterior surgery, our technique can obtain sufficient longitudinal reduction through large-weight skull traction, thorough intersegmental release, and lever distraction. The effect that is close to anterior release and as much reduction as possible should be achieved before fixation. The fixation strength is also limited because of local bone dysplasia, particularly when using atlantoaxial fixation only. Thus, simply using internal fixation for reduction can easily cause fracture or implant failure. After sufficient release and preliminary reduction, occipitocervical fixation not only fixes firmly but also exhibits strong reduction and orthopedic capability to help achieve full reduction (Figures 1 and 2).

Conclusions

Simple posterior release, reduction, and fixation for the treatment of BI with IAAD remains challenging and controversial. Based on careful preoperative assessment and strict indications, the technique is not only feasible and effective but can also achieve full decompression, reduction, and reconstruction of normal alignment and stability. Hence, the technique in an option for the treatment of BI with IAAD.

Disclosure statement

No potential conflict of interest was reported by the authors.

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