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Review Article

Bow Hunter's syndrome surgical approach and outcome: Two new cases and literature review

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ABSTRACT

Background: Bow Hunter's syndrome (BHS) is a rare entity known as rotational vertebral artery occlusion syndrome. Classically, it presents with nausea, vertigo, and dizziness elicited by extension or rotation of the neck. There are several management approach modalities, including surgical and nonsurgical alternatives.

Methods: We conducted an electronic database search on PubMed and Scopus. The search was performed on February 18, 2024, using a combination of keywords related to Bow Hunter Syndrome regarding management. From the latter query, 97 results followed, from which we included 76 and excluded 21 due to the information being irrelevant to our study and non-retrievable publications.

Results: A total of 121 patients were retrieved. The mean age of presentation was 50 years, with a female-to-male ratio of 3:1. There were 108 adult cases, and only 13 were pediatric and adolescents. Symptoms were elicited by right rotation (46%). The most affected levels were C1-C2 (44%). The anterior approach was the most common (40%) and had a better outcome (84%), followed by the posterior (30%), which had more cases with partial recovery (19% vs. 16%).

Conclusion: BHS management is still challenging as there are many factors that we must consider when deciding on the approach. There is inconclusive evidence on the proper management of these patients. Although the suggestions found in our review and our experience are valuable, no definitive management ensures a good quality of life and outcome for these patients. Further research is needed on this topic.

Keywords: Artery, Bow, Insufficiency, Management, Occlusion, Vertebral

INTRODUCTION

Bow hunter's syndrome (BHS), also recognized as rotational vertebral artery (VA) occlusion syndrome, denotes the mechanical obstruction or narrowing of the VA when the head and neck are rotated or extended. This uncommon clinical condition may result in symptomatic ischemia or infarction within the vertebrobasilar artery system.[29,30,63]

Typically, the underlying mechanism involves dynamic narrowing or occlusion of the VA due to mechanical pressure exerted by a bony structure.[19] Common causes include osteophytes, herniated discs, spondylosis, tendinous bands, or tumors.[10]

This study aims to review the literature of cases reported from 1988 to 2024 according to our specific search strategy aiming to elucidate surgical management evidence within this entity, in addition to two new cases from our own experience, providing a comprehensive review of their symptoms, surgical approaches, outcomes, and etiologies of their dynamic occlusion, and reviewing clinical and anatomical correlations of VA occlusion (VAO) in these patients.

MATERIALS AND METHODS

A comprehensive search of the literature was conducted using electronic databases including PubMed and Scopus. The search was performed on February 18, 2024 using a combination of keywords related to Bow Hunter Syndrome. The search query used on PubMed included the following boolean operators and keywords as follow: ("bow hunter's syndrome"[All Fields] OR "bow hunter's stroke"[All Fields] OR "rotational vertebrobasilar insufficiency"[All Fields] OR "dynamic vertebrobasilar insufficiency"[All Fields]) AND ("Choice Behavior"[MeSH Terms] OR "Disease Management"[MeSH Terms] OR "surgical procedures, operative"[MeSH Terms]). From the latter query 97 results followed from which we included 76 and excluded 21 due to the information being irrelevant for our study and non-retrievable publications.

RESULTS

A total of 121 patients were retrieved from the results of our search. The mean age of presentation was 50 ± 18 years, with a female-to-male ratio of 3:1. There were six pediatric cases.[11,12,46,56] Seven adolescent cases and 108 adult cases were retrieved [Table 1].[3,11,12,22,56,57]

Rotational vertebrobasilar insufficiency is defined by reversible symptoms when the neck is rotated. The most common direction for neck rotation that elicited symptoms was to the right (46%), followed by the left (38%), and 12 were bilateral (14%). The symptoms most frequently reported were vertigo, syncope, nausea, dizziness, and visual disturbances. The most common etiology in all the cases was osteophytes, as in our two presented cases, followed by idiopathic causes. Osteophytes were considered as any anatomical bony anomaly that could affect VA flow [Table 2].

The most common surgical approach was anterior (42%), followed by posterior (30%). We conducted a posterior approach and decompression with remarkable results in the two cases that we presented. About 95% of the cases underwent a surgical procedure, with decompression most commonly performed (81%). The most frequently affected level was C1-C2 (44%), following C5-C6 (25%) in frequency of occurrence. The latter can correlate with atlantoaxial affection being this entity's most common presentation type (51%). Most patients experienced recovery, with 18 patients showing partial recovery as defined by the presence of symptoms postoperatively.

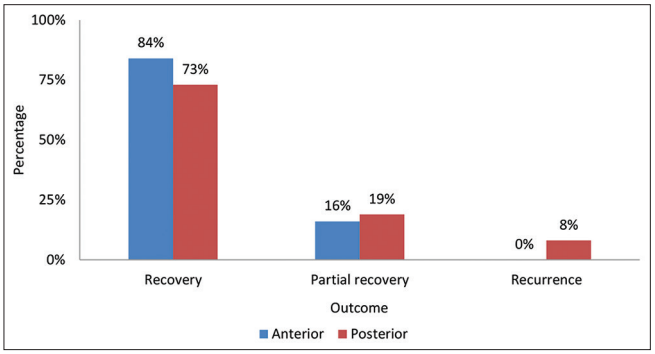
BHS remains a rare clinical condition without clear evidence of a superior treatment method, but as we revised, we could imply that in most of the cases in which an anterior approach was conducted, a better outcome resulted (84% anterior vs. 73% posterior). Partial recovery cases had a higher occurrence in posterior approaches (19% posterior vs. 16% anterior), and only 8% of cases presented recurrence, all of which a posterior approach was performed [Graph 1].

In our two cases, the first patient presented 2 weeks postoperatively with marked improvement in dysmetria and no gait disturbance. However, a discrete positive Romberg and left horizontal nystagmus persisted at the 6-month follow-up. In the second case, physical examination was unremarkable at 6 months follow-up.

CASE PRESENTATION

Case 1

A 24-year-old man presented with a 9-month history of recurring episodes of hemiparesis, right hemihypoesthesia,



Graph 1: Approach and outcome comparison.

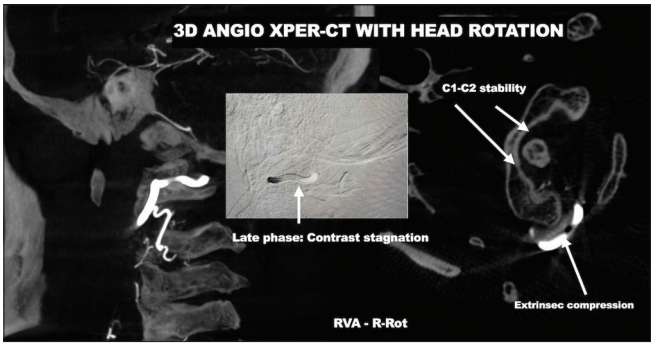


Figure 1: 3D XPER-computed tomography angiogram, right head rotation. The lateral view confirms contrast stagnation in the late phases at the C1 vertebral groove. Axial view confirms the atlantoaxial joint is stable. RVA: Right vertebral artery, R-Rot: Right head rotation.

Table 1: Summary of 121 revised cases of bow hunter syndrome.

Author and Year	Case No	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Renthlei *et al.*, 2023[46]	1	M, Pediatric	R, Altered sensorium and tremor	Not mentioned	Atlantoaxial dislocation	Conservative and fusion after 8 months	Recovery
Spence *et al.*, 2023[52]	2	F, 42	R, Neck discomfort, headache and vertigo	C5	Thyroid cartilage	Surgical decompression	Not specified
Regenhardt *et al.*, 2022[45]	3	M, 58	R, Dizziness, bilateral hearing loss	C5-C6	Osteophyte. Hypoplastic LVA	Conservative	Not specified
Judy and Theodore, 2021[27]	4	M, 60	Light-headedness, dizziness, and posterior headache.	C4-C5	Osteophyte	Surgical Anterior decompression	Recovery
Dohzono *et al.*, 2020[18]	5	F, 59	Bi, Incomplete left hemiplegia	C3-C4	Bony ankylosis	Surgical posterior fusion	Recovery.
Luzzi *et al.*, 2021[34]	6	F, 42	L, Dizziness, double vision, arms paresthesia	C5	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	7	F, 66	L, Drop attacks (atonic seizure)	C3	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	8	F, 67	L, Drop attacks, vertigo	C5	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	9	M, 66	L, Drop attacks	C6	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	10	M, 67	L, Drop attacks, arms and legs paresthesias	C3-C4	Osteophyte, disc herniation	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	11	M, 32	R, Vertigo paresthesia	C5-C6	Congenital bony anomaly	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	12	M, 61	R, Double vision, dizziness	C4	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	13	M, 63	L, Drop attacks	C5	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	14	M, 74	L, Stroke, drop attacks	C5	Osteophyte	Surgical anterior decompression	Recovery. Persistence of mild ataxia.
Luzzi *et al.*, 2021	15	M, 78	R, Drop attacks, arms paresthesia	C5	Osteophyte	Surgical anterior decompression	Recovery
Luzzi *et al.*, 2021	16	M, 80	L, Stroke, drop attacks	C5	Osteophyte	Surgical anterior decompression	Recovery. Swallowing and dysphagia caused by stroke.
Luzzi *et al.*, 2021	17	M, 38	L, Vertigo	C6	Trauma	Surgical anterior decompression with fusion	Recovery
Golomb *et al.*, 2020[22]	18	M, 12	L, anisocoria	C2	Bony compression/ instability	Surgical posterior fusion	Recovery
Bando *et al.*, 2020[3]	19	F, 13	R, Visual disturbance, hypoesthesia, left side paralysis	C1-C2	Atlantoaxial dislocation (C1 aplasia and instability)	Posterior fusion	Slight hypoesthesia of the left hand

(*Contd...*)

Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Cornelius *et al.*, 2019[12]	20	M, 8	R, Ataxia, vertigo, nausea, hemiparesis, gait disturbance, bilateral pyramidal tract signs.	C1, C2	Bony malformations and fibrous band	Surgical anterior decompression	Residual hemiparesis.
Cornelius *et al.*, 2019	21	M, 9	L, Vertigo, nausea, nystagmus, torticollis	C0-C1	Bony compression malformation	Surgical posterior bilateral fusion C0-C2	Recovery
Cornelius *et al.*, 2019	22	M, 16	Bi, Neck pain, and loss of consciousness.	C2	Bilateral bony stenosis and fibrous band on the left side.	Anterior bilateral decompression	Recovery
Cornelius *et al.*, 2019	23	M, 42	R, Vertigo, during head extension, loss of consciousness.	C1	Fibrous band at sulcus of atlas	Left anterolateral decompression	Recovery
Cornelius *et al.*, 2019	24	M, 46	Neck pain, visual impairment, dysphasia, dizziness, dysphagia,	C1-C2	Fibrous band	Left anterolateral decompression	Recovery, residual psychosomatic symptoms.
Cornelius *et al.*, 2019	25	M, 29	L, Vertigo, and loss of consciousness.	Atlas	Fibrous band	Left anterolateral decompression	Recovery
Cornelius *et al.*, 2019	26	M, 24	R, Vertigo, nausea, and visual disturbances	C1-C2	Fibrous band	Right anterolateral decompression	Recovery
Cornelius *et al.*, 2019	27	M, 21	L, Vertigo during extreme head rotation.	C2	Bony stenosis and fibrous band	Left anterolateral decompression	Recovery
Cornelius *et al.*, 2018[13]	28	M, 54	L, Syncope and blurry vision	C6-C7	Osteophyte (transverse foramen)	Surgical anterior decompression	Recovery
Ng *et al.*, 2018[41]	29	M, 70	L, syncope	C3-C4	Osteophyte	Surgical anterior fusion	Recovery
Schunemann *et al.*, 2018[48]	30	M, 60	L, dizziness	C2	Idiopathic	Surgical anterior fusion	Recovery
Takeshima *et al.*, 2018[56]	31	M, 65	DS, vertigo	C1	Idiopathic	Surgical posterior atlantoaxial fixation	Recovery
Takeshima *et al.*, 2018	32	M, 34	NDS, vertigo	C1	Os odontoideum	Surgical posterior atlantoaxial fixation	Recovery
Takeshima *et al.*, 2018	33	M, 7	rotational	C2	Atlantoaxial subluxation	Surgical posterior atlantoaxial fixation	Partial recovery (mRS 3;1)
Takeshima *et al.*, 2018	34	M, 22	R, Vertigo, visual field defect	C1	Idiopathic	Surgical posterior atlantoaxial fixation	Partial recovery mRS (2;1)

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Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Takeshima *et al.*, 2018	35	M, 52	DS, Diplopia, vertigo	C2	Rheumatoid arthritis	Surgical posterior atlantoaxial fixation	mRS 5;4
Takeshima *et al.*, 2018	36	M, 60	DS, Syncope	C3-C4	Spondylosis	Conservative external fixation	Recovery
Takeshima *et al.*, 2018	37	M, 16	NDS, Diplopia, vertigo.	C1	Idiopathic	Conservative external fixation	Recovery
Takeshima *et al.*, 2018	38	F, 18	NDS, Vertigo	C2	Idiopathic	Surgical posterior atlantoaxial fixation	Recovery
Berti *et al.*, 2018[4]	39	M, 56	"Hot flash" sensation, nausea, emesis, diplopia, and dysarthria	C4-C5	Fibrous band	Endovascular plugs	Recovery
Bergl, 2017[5]	40	M, 62	L, Dizziness, tinnitus, headache	C6	Idiopathic	Posterior fixation and fusion	Recovery
Strickland *et al.*, 2017[53]	41	F, 73	R, Vertigo, numbness, tingling	C1-C2	Osteophyte	Posterior decompression	Recovery
Motiei-Langroudi *et al.*, 2017[37]	42	M, 61	L, Lightheadedness, and facial numbness	V1 segment	Tortuous V1 segment	Self-expanding biliary stent placement	Recovery
Lu *et al.*, 2017[33]	43	M, 71	Bi, Chronic vertigo, headaches, tremors, and irregular respiration	C4-C5	Idiopathic	Decompression and anterior fusión	Recovery
Buch *et al.*, 2017[7]	44	M, 38	R, Dizziness, syncope	C1	Persistent first intersegmental artery	Posterior decompression	Recovery
Chaudhry *et al.*, 2016[8]	45	F, 56	R, Syncope	C5-C6	Fibrous band	Anterior decompression	Recovery
Chaudhry *et al.*, 2016	46	M, 73	L, Lightheadedness, and vertigo	C4-C6	Osteophyte	Decompression and anterior fusion	Recovery
Takekawa *et al.*, 2015[55]	47	F, 23	Bi, Three episodes of recurrent ischemic stroke, dysesthesia	C1-C2	Not specified	Not mentioned	Not mentioned
Ravindra *et al.*, 2015[44]	48	M, 66	Bi, Episodes of syncope, and loss of consciousness	C1	Extradural vertebral artery compression	Posterior decompression	Recovery
Ravindra *et al.*, 2015	49	F, 53	R, Headaches, forgetfulness	PICA	Extradural dynamic compression of PICA	Medical	Not mentioned
Ravindra *et al.*, 2015	50	F, 37	Bi, Neck pain	PICA	Extradural dynamic compression of PICA	Decompression	Not mentioned
Healy *et al.*, 2015[23]	51	M, 58	Bi, Cervicalgia, presyncope, vertigo, and sensation of "impending doom."	C1-C2 and C4-C5	Osteophyte	Posterior fusion and decompression	Recovery

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Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Takeshima *et al.*, 2014[57]	52	F, 18	Bilateral headache, nausea, and cerebellar ataxia	C2 (atlantoaxial level)	Bony malformation	Posterior fusion	Recovery
Dargon *et al.*, 2013[16]	53	M, 53	R, photopsia, syncope	C2-C5	Idiopathic	Anterior decompression	Recovery
Buchanan *et al.*, 2014[6]	54	M, 52	L, Dizziness, bilateral upper extremity weakness	C3-C4	Osteophyte	Anterior decompression	Recovery
Safain *et al.*, 2014[47]	55	F, 37	Vertigo, tightness, headache, dimming of entire visual fields	Occiput-C1 and C2-C3	Instability at the occipitocervical junction	Fusion	Recovery
Anaizi *et al.*, 2014[2]	56	F, 68	R, Disorientation, loss of balance, loss of consciousness	C1	Osteophyte	Decompression	Recovery
Ding *et al.*, 2013[17]	57	F, 43	L, Pre-syncopal and syncopal episodes	C4-C5	Osteophyte	Decompression	Recovery
Nguyen *et al.*, 2015[42]	58	M, 52	R, Presyncope	C6-C7	Idiopathic	Anterior decompression	Recovery
Hong *et al.*, 2023[25]	59	M, 29	Recurrent vertigo and syncope	C6	Tubercule	Decompression	Re-operation and recovery after 2nd decompression
Vates *et al.*, 2002[59]	60	M, 56	L, Dizziness, and syncope	C4-C5	Laterally herniated intervertebral disc	Anterior decompression	Recovery
Fleming *et al.*, 2013[20]	61	M, 54	Bi, Diplopia, tinnitus, headache	C4-C5	Spondylosis	Surgical anterior decompression with fusion	Recovery
Darkhabani *et al.*, 2012[15]	62	M, 66	Vertigo, Ataxia	C2-C6	Idiopathic	Endovascular Stenting	Recovery
Darkhabani *et al.*, 2012	63	M,55	Vertigo, Cefalea, Tinnitus, Diplopia	C2-C6	Idiopathic	Endovascular Stenting	Recovery
Darkhabani *et al.*, 2012	64	M,85	Vertigo, syncope	C2-C6	Idiopathic	Endovascular Stenting	Recovery
Darkhabani *et al.*, 2012	65	M, 70	Confusion, Diplopia	C2-C6	Idiopathic	Endovascular Stenting	Recovery
Kan *et al.*, 2018[28]	66	M, 41	R, Bradycardia, nausea, vomiting, vertigo	N/A	Trauma	PICA-PICA bypass	Recovery
Cornelius *et al.*, 2012[11]	67	M, 8	R, Ataxia, vertigo, nausea, hemiparesis, gait disturbance	C1-C2	Bony compression	Left Anterolateral Decompression	Recovery
Cornelius *et al.*, 2012	68	M, 9	L, Vertigo, nausea, nystagmus, torticollis	C0-C1	Bony compression	Posterior Decompression and Fusion	Recovery
Cornelius *et al.*, 2012	69	M, 16	Bi, Neck pain, Syncope	C2	Bony compression	Anterior Decompression and Fusion	Recovery
Cornelius *et al.*, 2012	70	M, 42	R, Vertigo, Syncope	Sulcus of Atlas	Fibrous band at sulcus of atlas	Anterior Decompression	Less Vertigo, No Syncope
Cornelius *et al.*, 2012	71	F, 46	R, Neck pain, visual impairment, dysphasia, dizziness, dysphagia	C1-C2	Fibrous bands C1-C2	Anterior Decompression	No syncope, residual psychosomatic symptoms

(*Contd...*)

Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Yoshimura *et al.*, 2011[64]	72	M, 64	R, Dizziness, lipothymia, syncope	Bilateral VA occlusion at the left C3-C4 and right C1-C2	Instability at C3-C4	Anterior decompression and fusion	Recovery
Lee *et al.*, 2011[32]	73	M, 50	R, Syncope, neck pain, dizziness	C7	Bony compression	Decompression	Recovery
Lee *et al.*, 2011	74	F, 28	R, Dizziness, ataxia, diminished vision	C7	Hypertrophic transverse process	Decompression	Recovery
Sugiu *et al.*, 2009[54]	75	M, 56	L, Vertigo, syncope	C1-C2	VA stenosis	Endovascular	Recovery
Ho *et al.*, 2008[24]	76	M, 34	R, Dizziness, near-syncope	C4-C5, C5-C6 right, C1-C2 left	Idiopathic	Anterior decompression and fusion	Recovery
Tsutsumi *et al.*, 2008[58]	77	M, 59	L, Lipothyimia, syncope	C5-C6, C6-C7	Instability at C5-C6, C6-C7	Anterior Decompression and fusion	Recovery
Kim *et al.*, 2008[31]	78	M, 60	L, Dizziness	C2	Bony compression	Anterior decompression	Recovery
Whitmore *et al.*, 2007[62]	79	M, 57	R, Syncope, occipital headache	C1-C2	Bony compression	Decompression	Recovery
Velat *et al.*, 2006[60]	80	M, 58	L, Dizziness, vertigo, lipothymia	C3-C4, C4-C5	Osteophyte formation	Anterior decompression	Recovery
Netuka *et al.*, 2005 [39]	81	M, 54	R, Headache, vertigo, nausea	C1-C2	Not mentioned	Anterior decompression	Recovery
Seki *et al.*, 2001[49]	82	M, 47	R, Syncope	C2	Idiopathic	Anterior decompression	Recovery
Shimizu *et al.*, 1999[50]	83	M, 53	L, Vertigo, syncope	C2	VA course variant	Anterior decompression	Recovery
Matsuyama *et al.*, 1997[35]	84	M, 57	Vertigo	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	85	F, 63	Dizziness	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	86	M, 65	Syncope	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	87	M, 59	Facial numbness	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	88	M, 61	Vertigo	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	89	F, 53	Vertigo	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	90	M, 73	Syncope	C1-C2	Not mentioned	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	91	F, 75	Vertigo	C1-C2	Not specified	Posterior fusion	Recovery
Matsuyama *et al.*, 1997	92	F, 56	Syncope	C1	Not mentioned	Posterior decompression	Recurrence
Matsuyama *et al.*, 1997	93	M, 53	Dizziness	C1-C2	Not mentioned	Posterior decompression	Recovery
Matsuyama *et al.*, 1997	94	F, 55	Syncope	C1	Not mentioned	Posterior decompression	Recovery
Matsuyama *et al.*, 1997	95	M, 64	Dizziness	C1-C2	Not mentioned	Posterior decompression	Recovery
Matsuyama *et al.*, 1997	96	M, 51	Dizziness	C1	Not mentioned	Posterior decompression	Cerebellar infarction

(*Contd...*)

Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Matsuyama *et al.*, 1997	97	M, 58	Vertigo	C1	Not mentioned	Posterior decompression	Recovery
Matsuyama *et al.*, 1997	98	F, 57	Vertigo	C1-C2	Not mentioned	Posterior decompression	Recurrence
Matsuyama *et al.*, 1997	99	F, 71	Vertigo	C1-C2	Not mentioned	Posterior decompression	Asymptomatic
Matsuyama *et al.*, 1997	100	M, 59	Syncope	C1	Not mentioned	Posterior decompression	Asymptomatic
Nagasawa and Ohtsuki, 1991[38]	101	M, 32	Syncope	C2	Tumor	Lateral decompression	Recovery
Shimizu *et al.*, 1988[51]	102	M, 37	Dizziness	C1	Fibrous bands C1-C2	Lateral decompression	Recovery
Algattas *et al.*, 2024[1]	103	M, 69	R, Presyncope, dizziness	C3-C4	Uncovertebral joint hypertrophy	Anterior decompression and fusion	Recovery
Vilela *et al.*, 2005[61]	104	62	L, Dizziness, syncope	C6-C7	Compression	Lateral decompression	Mild dizziness
Vilela *et al.*, 2005	105	M, 52	R, Syncope, blurred vision, dizziness	C3-C4	Not specified	Partial lateral decompression	Recovery
Vilela *et al.*, 2005	106	65	R, Syncope, nausea, vomiting, dimming vision	C1	Foraminal stenosis	Posterior decompression	Mild dizziness
Vilela *et al.*, 2005	107	76	Bi, Tinnitus, near syncope, disequilibrium, generalized weakness	C1-C2	Instability	Posterior decompression (using sublaminar wires)	Mild neck pain
Vilela *et al.*, 2005	108	M, 60	Bi, Syncope	C1-C2	Osteophytes	Partial C2 corpectomy (decompression)	Occasional dizziness with head turning
Vilela *et al.*, 2005	109	54	R, Vertigo, nausea and vomiting	C5-C6	Not specified	Anterior decompression	Recovery
Vilela *et al.*, 2005	110	F, 71	L, Near syncope, occipital neuralgia	C1-C2	Osteophytes	Decompression	Recovery
Vilela *et al.*, 2005	111	68	R, Syncope, blackouts, dysarthria, dimming vision	C6	Osteophytes	Anterior decompression	Recovery
Vilela *et al.*, 2005	112	54	R, Syncope	C3-C4	Osteophytes	Anterior decompression	92mo Recovery
Vilela *et al.*, 2005	113	58	L, Dizziness, and syncope	C3-C4, C4-C5, C5-C6	Osteophytes	Anterior decompression	Occasional dizziness when head turned to the left; no syncope
Chen *et al.*, 2000[9]	114	Not mentioned	R, Dizziness, and vertigo	C4	Not mentioned	Decompression	Recovery
Jost and Dailey, 2015[26]	115	M, 55	Syncope, vision loss	C6-C7	Foraminal disc herniation	Anterior decompression and fusion	Not mentioned
Jost and Dailey, 2015	116	F, 47	Dizziness, disturbance of vision	C5-C6	Spondylosis and disc herniation	Anterior decompression and fusion	Neck pain and stiffness

(*Contd...*)

Table 1: (*Continued*).

Author and Year	No.	Sex/age	Side/presentation	Level	Etiology	Definitive approach	Outcomes
Montano *et al.*, 2021[36]	117	F, 79	L, lightheadedness, tinnitus, darkening of vision	C4-C5	Bony malformation	Anterior decompression and fusion C4-C5	Recovery
Kantak *et al.*, 2021[29]	118	M, 37	R, Blurry vision, diminished visual field, lightheadedness and headaches	Atlantoaxial	Trauma (facet joint subluxation)	Posterior fusion	Recovery
Lee *et al.*, 2011[32]	119	M, 50	L, Syncope, dizziness	C7	Bony compression (uncinate process)	Anterior decompression	Recovery
Lee *et al.*, 2011	120	F, 28	R, Dizziness, ataxia, diminished vision, right arm numbness with adduction	C7	Hypertrophic transverse process	Anterior decompression	Recovery
Dabus *et al.*, 2008[14]	121	M, 41	R, Dizziness, right headedness	C1-C2	Thyroid cartilage	Conservative	Conservative

L: Left, R: Right, M: Male, F: Female, Bi: Bilateral, DS: Dominant side, NDS: Non-dominant side, PICA: Posterior inferior cerebellar artery, mRS: Modified Rankin Scale

dysarthria, and aphasia. In addition, he reported experiencing diplopia, nausea, and occasional dizziness, particularly when rotating or turning his head. Single-photon-emission computed tomography (CT) showed cerebellar hypoperfusion.

With the latter clinical presentation, a dynamic arterial compression was suspected. A 2digital subtraction angiogram (2DDSA) and an XPERCT reconstruction revealed contrast stagnation at the vertebral groove of C1 during head rotation, confirming right VA V3 bony stenosis [Figure 1].

The surgical intervention involved the execution of a decompression procedure, which included a suboccipital craniectomy with right lateral extension for exploration and decompression of the foramen magnum and the right VA. Subsequently, resection of C1 right hemi-arch and C2 cervical hemilaminectomy with foraminotomy was performed, respectively, to decompress the right VA, aided by microscope assistance and intraoperative neurophysiological monitoring.

Technical description

Surgical "hockey stick incision" site marking from C2 medial line to occipital protuberance and right mastoidal apophysis was performed. The occipital bone and C1 tubercle were identified, and the superficial and deep muscular planes were dissected. The right C1 transverse process and suboccipital triangle were identified, followed by blunt dissection. Using a surgical microscope (ZEISS Kinevo 900, Carl Zeiss, Germany), the suboccipital triangle's muscle was released,

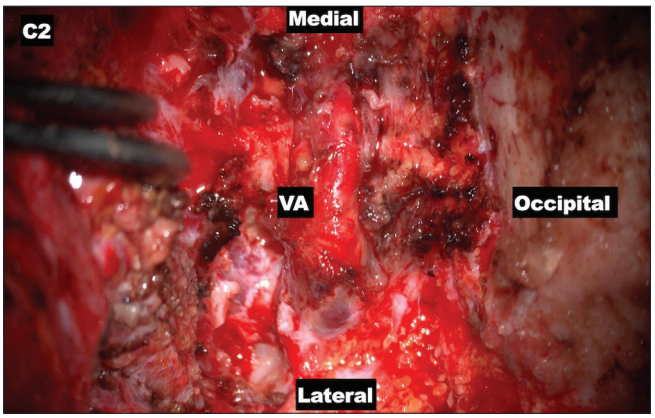


Figure 2: Microsurgical view after vertebral artery decompression. A right hemilaminectomy of C1 and the lateral superior portion of the transverse foramen was opened to allow VA foramen decompression. VA: Vertebral artery.

exposing the right posterior hemi-arch of C1 and its right transverse process. Subsequently, using an NSK (Primado2, NSK, Japan) drilling device with a 1 and 2 mm diamond drill, a transverse foraminal opening and resection of C1 right hemi-arch were performed [Figure 2]. An intravenous injection of fluorescein was administered (0.4 cc sodic Fluorescein + 20 cc saline bolus), and flow through the right VA was observed. The entire vertical and horizontal course of the right VA was freed. A new injection of fluorescein was performed, confirming improved flow through the right

Table 2: Patient clinical characteristics.

Variable	Value (%)
Total number of patients	121
The mean age of presentation	50
Sex M/F	
M	75% (81/108)
F	25% (27/108)
Approach anterior versus posterior	
Anterior	42% (51/121)
Posterior	30% (37/121)
Surgical versus conservative	
Surgical	95% (115/121)
Conservative	4% (5/121)
Fixation versus decompression	
Fixation	39% (39/110)
Decompression	81% (81/110)
Outcome	
Recovery	77.5% (93/120)
Partial recovery	15% (18/120)
Recurrence	5% (6/120)
Not specified	2.5% (3/120)
Affected level (frequency of occurrence)	
C0	2%
C1-C2	44%
C3-C4	16%
C5-C6	25%
C7	13%
Etiology	
Osteophyte	21%
Idiopathic	12%
Fibrous band	10%
Bony compression	9%
Type	
Atlantoaxial	61/118 (51%)
Subaxial	48/118 (8%)
Mixed	9/118 (41%)

M: Male, F: Female

VA. Suboccipital craniotomy was performed, identifying the trajectory directed toward the foramen magnum. Irrigation with 400 mcg of nimodipine with saline solution was done.

Two weeks postoperatively, the patient presented marked dismetry improvement and no gait disturbance. Discrete positive Romberg and left horizontal nystagmus were noted. At 6 months' follow-up, the patient persists with the left horizontal nystagmus with the rest of the examination resulting unremarkable.

Case 2

This 57-year-old woman presents with a 4-year history characterized by episodes of dizziness, fainting spells, and nausea on rotating her head to both sides, particularly after a vehicle collision. She also experienced vomiting, pallor, and predominantly left-sided tinnitus. Her current medications include rivaroxaban, captopril, and celecoxib. She has been evaluated at her health service institution, where she was diagnosed with cerebral venous thrombosis based on findings from diagnostic cerebral angiography.

During the neurological examination, discrete bilateral papilledema was noted. Fainting and dizziness occurred following extraocular movements and upward deviation of the gaze. In addition, fainting was observed after rotational head movements and left jugular compression. The remainder of the examination was unremarkable.

Cerebral diagnostic angiography performed by another operator reported a left transverse sinus venous thrombosis. Stenosis of the middle and distal third of the right VA was reported; however, the static nature of this study warrants further complementary imaging. However, after reviewing her 2DDSA, we considered that there was no venous thrombosis and repeated a 2D-DSA comparing VA flow with a cephalic neutral and rotational position. A right VAO was observed during the cephalic left rotation. A 3D XPERCT angiogram confirmed stenosis at the beginning of the horizontal pathway in C1-C2. Left VA was non-dominant, with significant hemodynamic repercussions during left rotation [Figure 3].

Technical description

The occipital bone, right mastoid process, C1 tubercle, and C2 spinous process are identified, followed by blunt dissection. Under microscopic view, lateral 1/3 hemifacetectomy at C2 is performed using an NSK (Primado2, NSK, Japan) drilling device with a 1 and 2-mm diamond drill. With the assistance of microsurgical instruments and MIS Kerrison rongeurs, the lateral wall and roof of the vertebral foramen were opened. Dissection and cutting with Takayama microscissors of the fibrotic band compressing the VA at its exit from the right vertebral foramen are performed. The artery is freed from all bony and soft-tissue contact [Figure 4]. An IV injection of sodic fluorescein (0.4 cc sodic fluorescein + 20 cc saline bolus) was administered, and adequate flow through the right VA was observed using a YELLOW 560 filter. The procedure was concluded. On postoperative day 1, a CT-angiography (CTA) scan was performed with cephalic left rotation, which showed correct decompression and flow through the right VA.

At 6 months follow-up, the patient presented significant improvement with an unremarkable physical examination.

DISCUSSION

Overview of BHS

BHS is a rare entity associated with vertebrobasilar insufficiency due to mechanical stenosis or transient dynamic occlusion of a dominant VA.[41] Compression may occur frequently on

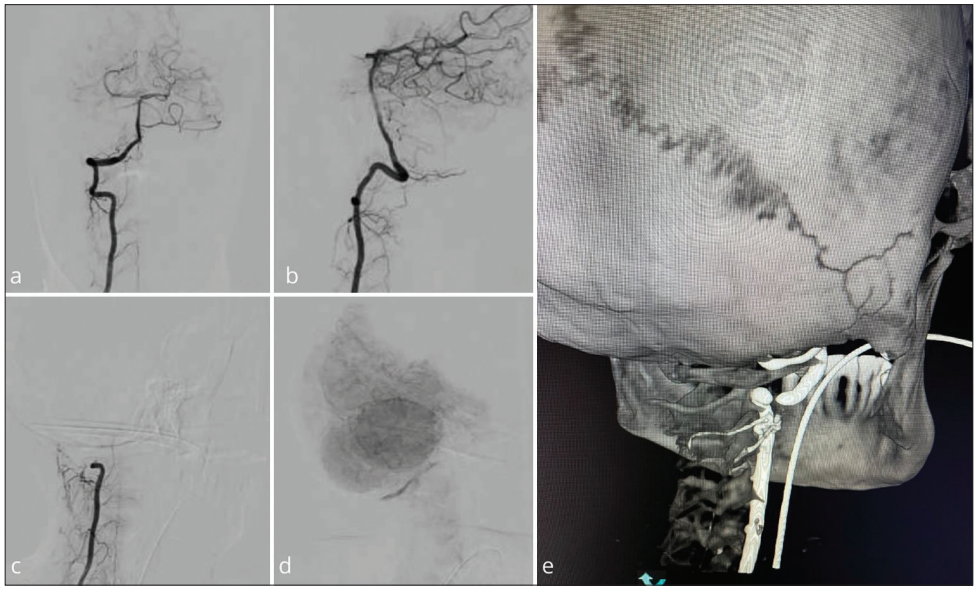


Figure 3: 2D DSA angiogram, right vertebral artery (VA); (a and b) AP and lateral view during the arterial phase with a neutral position. (c) Right VA injection, arterial phase. (d) Capillary phase left VA. (e) XPER computed tomography angiogram with head rotation.

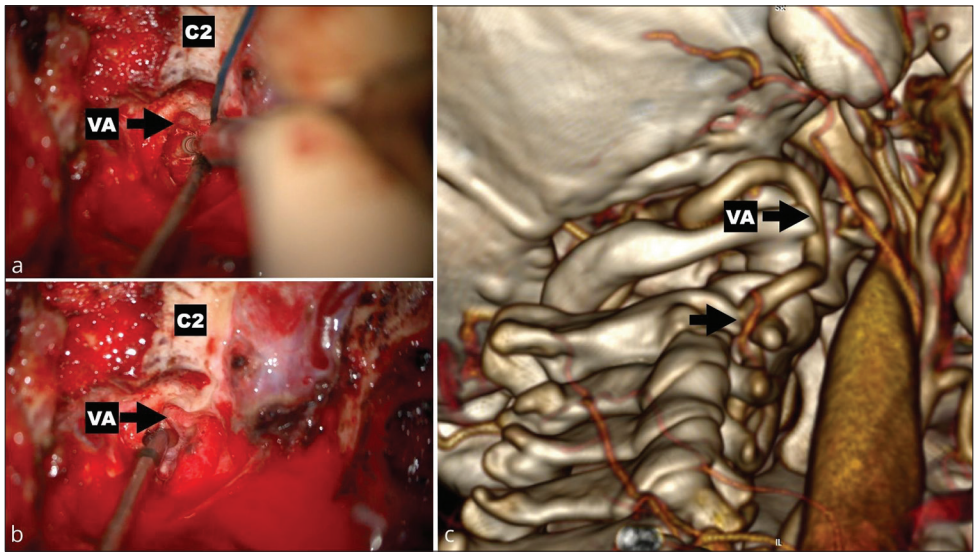


Figure 4: (a and b) Microsurgical view during vertebral artery (VA) liberation. (c) Microsurgical view after VA liberation. Postoperative angio computed tomography with left head rotation showed the decompression of the VA in C1 and C2 and the opening of the vertebral foramina.

V2–V3 VA segments, which correlate with the most common presentation, atlantoaxial (C1–C2), as seen in our revision. Nevertheless, there is evidence that the most common presentation in pediatric patients is at the atlantooccipital level.[43] Some pathoanatomic elements are necessary for the condition to become symptomatic: Dominant VA compression (osteophyte, disc material or mechanical instability, fibrous bands); causes may be traumatic, degenerative, or a connective tissue disorder. Other elements may be a contralateral hypoplastic or aplastic VA, a posterior inferior cerebellar artery (PICA) ending contralateral VA and insufficient flow through posterior circulation leading to insufficient collateral flow.[13]

In various cases of BHS, collateral perfusion allows sufficient flow through the contralateral VA and posterior communicating artery, compensating primary VAO. Nevertheless, there are cases in which hypoplastic arteries or PICA cause hemodynamic insufficiency, and other causes could be due to thromboembolic stroke due to repetitive microtrauma. There are three types of BHS: atlantoaxial,

subaxial, and mixed-type BHS; atlantoaxial is the most common. Symptoms vary, but most of the time, it presents with vertigo, visual disturbance, syncope, and cerebellar stroke. Typically, there is a long delay between symptom onset and diagnosis.[12]

Current treatment approaches and diagnosis modalities

Dynamic angiography remains the gold standard of diagnosis for BHS.[5,12] We must emphasize the importance of diagnosing this condition using the gold standard modality, as standard techniques may overlook certain anomalous bony structures, potentially leading to further complications. Even a posterior approach could ignore these structures. Posterior or anterior approaches with fusion may be needed in patients with cervical instability, always considering the limited mobility that this may cause.[12] Capturing any perivascular bone or soft-tissue anomalies as the etiologies for BHS requires using cone-beam CT, a diagnostic tool that provides for three-dimensional viewing of the vasculature and soft tissue.[33]

There are three aspects to consider when doing an imaging study in patients with positional VAO (PVAO): identify the occurrence and location of temporary VAO, the pattern of cerebral ischemia, and the underlying disorder or etiology. One of the drawbacks of this method is that head rotation carries a potential risk of stroke recurrence, especially in the case of embolism. Two aspects must be considered before using this imaging technique: identifying the pattern of cerebral ischemia and checking for spinal instability.[56]

At present, there are three treatment approaches: conservative treatment, which involves avoiding specific neck movements; surgical treatment of the cervical spine, which includes cervical spine fusion and fixation or decompressive surgery alone; and endovascular stenting.[5]

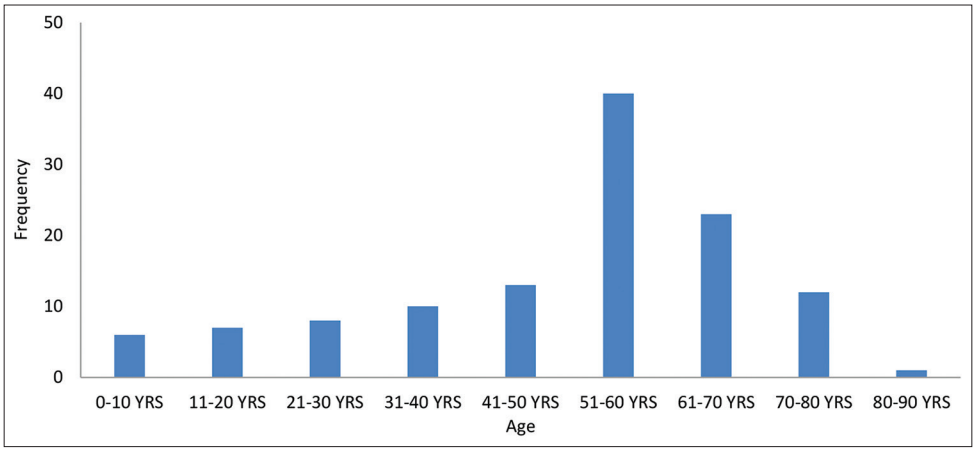
Efficacy of treatment modalities

It must be considered that there is no gold standard for the treatment of BHS. The literature describes various treatment methods, including conservative, endovascular, and surgical approaches. There is limited evidence supporting endovascular treatment for this entity. Conservative modalities tend to fail and have a high rate of conversion to surgical decompression.[40]

Based on this review, there are different perspectives and opinions regarding the optimal management of this condition, considering lower recurrence rates, complications, and quality of life for the patient. In our experience, we have reported two cases where we performed a posterior approach with decompression, and neither case resulted in an embolic stroke. We have found that some authors recommend a fusion approach almost regardless of the patient's presentation; however, we believe that it is essential to individualize patient management and perform the bony decompression, maintaining as much as possible the cervical spine joints, with the objective of preserving the stability of the spine.

Considering that most patients in our review are adults between 50 and 60 years of age [Graph 2], a fusion surgery could be well-tolerated, always individualizing the patient's conditions. The treatment approach depends always on the patient's anatomy, the pathology, and the etiology of the occlusion. It must be encouraged that every patient be given individualized, case-by-case management on an individual basis. The anterior approach could be safer compared to the posterior if transverse foramen decompression is not needed.[48]

On the other hand, young patients or children benefit significantly from avoiding unnecessary fusion, thus improving their quality of life. Fusion should be considered a last resort. Conservative management with a collar should



Graph 2: Common age of presentation. YRS: Years.

always be considered a temporary measure, as it could be hazardous. Endovascular management should not be considered an alternative for treating this condition at present due to the questionable effectiveness of its results since the VA compression is extrinsic, and bony structures tend to have a higher force than an endoluminal stent. Some advantages of endovascular stent placement include the minimally invasive nature of the procedures and neck motion preservation.[56]

There is a controversy between arterial decompressive surgery and spinal fusion surgery for treating patients with spinal instability. Drawbacks are plenty, but head rotation restriction and VA reocclusion on fusion and decompression surgery, respectively, are primarily essential trends. Takeshima *et al*. denote that their case series have favorable long-term outcomes following spinal fusion, noting that most cases are embolic.[56] This could be considered a condition where the patient could benefit from fusion. Furthermore, it states that spinal fusion is a better approach than arterial decompression for the treatment of PVAO because decompression surgery would scatter new thrombi or aggravate arterial dissection. Hence, in the case of recurrent stroke, fusion surgery would be the best alternative because it would ensure VA healing, limiting stroke recurrence [Table 3].

Table 3: Merits and drawbacks of surgical decompression or fusion approaches.

Approach	Merits	Drawbacks
Fixation	Low recurrence risk More reliable in the long term Limit stroke recurrence (in the long term) Vertebral artery healing	Limited range of motion Risk of vascular damage during procedure (leading to stroke) Affected quality of life
Decompression	Diminished vertebral artery injury risk No head movement limitation Improved quality of life	Risk of recurrence Risk of stroke (due to preserved mobility)
Conservative	Quality of life is relatively unaffected	Temporary measure Risk of stroke Limited range of motion

It is critical to avoid VA injury during surgical fixation as this could lead to stroke. For example, there is a 0.3–2% of the risk of VA injury during C2 pedicle screw fixation. Excluding anterior corpectomies, 53.1% of VA injuries occurred during the posterior approach, while only 24.3% occurred during the anterior approach. Fusion rates are similar between single-level anterior and posterior approaches.[48]

According to Cornelius *et al*., an anterolateral approach used for a vascular decompression on the V3 VA segment is a go-to approach with optimal outcomes and efficacy according to its patient population, in which the outcome was remarkable; this is comparable to our results. In this review, the author recommends identifying the VA during the procedure through micro-Doppler imaging, indocyanine green angiography, CTA, or DSA.[12,13] We also recommend using an IV injection of 0.4 cc of fluorescein as a practical and well-known technique to ensure adequate blood flow, as used in one of our cases.

When comparing decompression of the VA with posterior fixation surgery, decompression is a valuable approach for symptom management. However, there is a risk of symptom recurrence beyond 3 months postoperatively, as well as a risk of cerebral infarction.[21] On the other hand, posterior fixation surgery limits the range of motion but does affect activities of daily living due to the range of motion; the recurrence risk is very low. Based on the latter, in 2010, Fujimoto concluded that compared to exclusive decompression, the posterior fixation approach is safer and more reliable as a decompression surgical treatment. Head rotation is limited to 50–70% and is reported not to inhibit patients' activities of daily living.[21] Several complementary techniques, such as dynamic intraoperative catheter angiography, indocyanine green videography, and Doppler ultrasonography, contribute to the postoperative outcome. However, in 2014, Zaidi *et al*., supported by the work done by Shimizu *et al*., in 1988, recommended the approach of posterior decompression without fusion of the C1-C2 level.[51,65]

The choice of surgical technique approach depends on the site of compression, VA segment, and spinal instability. In the case of V2-V3 segment compression, the best alternative would be anterolateral. On the other hand, a posterolateral approach would be preferred in the case of a V3 segment compression.[13] An anterior approach is the preferred choice in atlantoaxial junction-level occlusions. However, these cases are extremely rare. Spinal segment stability is also critical for surgical modality as unstable V2–V3 segments require anterior discectomy and fusion, and in the case of V3, screw/rod system stabilization is the preferred choice. Correct diagnosis is critical because this leads to an optimal surgical technique selection and, later, to an excellent prognosis. Instability could be defined with a dynamic X-ray, and stabilization should be performed only if it is present. Some authors consider arterial decompression with fusion as overtreatment, and we emphasize the importance of directed treatment modalities. Stabilization stand-alone surgery has also reported good outcomes and the advantage is the diminished VA injury risk. The drawback is the limitation of head movement.[13] Other authors recommend an anterior decompression approach when the obstruction is located at

C5–C7 due to the more naturally accessed artery.[65]

Strickland *et al*. describe how numerous studies have been published demonstrating no objective difference following a decompression with or without fusion.[53]

Outcomes

Based on this review, we believe that there is no gold-standard approach for treating this condition. However, the outcome will always depend on correctly matching the patient with an optimal management approach. For a better outcome, several factors should be considered: the occurrence of stroke, initial neurological condition, age, comorbidities, occupation, and level of activity. We must consider intraoperative fluorescein, as it provides certainty of reperfusion during the procedure.

Preventing recurrent strokes in patients with PVAO should be a top priority for the long term. It is essential to differentiate that head rotation can be minimized on a subliminal level in patients with hemodynamic stroke. In contrast, in patients with embolic PVAO, thrombus scattering cannot be controlled, and vertebrobasilar strokes often result in permanent neurological deficits.[56]

CONCLUSION

BHS remains a rare entity where we can rely on our diagnosis with the gold standard modality being clear. However, the true challenge lies in finding the correct approach depending on the case and how we individualize it. Advantages and disadvantages depend on the chosen approach, and it is crucial to consider factors such as age, neurological presentation, comorbidities, VA level of involvement, risk of thrombosis or cerebral infarction, and cervical instability, among others. In addition to the above, weighing the risks and benefits for the patient is essential.

There is still a wide field of research needed to define evidence-based clinical recommendations for the proper treatment of this condition. Current recommendations are guided by experience, as demonstrated in our case and review. While these suggestions may be helpful, there is still much uncertainty about the best management approach to ensure patients' good outcomes and quality of life.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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