Cardiopulmonary Medicine

Review Article

Chris Moors*, BSc and Claire Stapleton, PhD

Signs and symptoms of vertebrobasilar insufficiency secondary to atherosclerosis: a systematic review

https://doi.org/10.1515/jom-2024-0203
Received September 22, 2024; accepted March 4, 2025;
published online March 28, 2025

Abstract

Context: Clinicians face a difficult challenge in identifying vertebrobasilar insufficiency (VBI) resulting from atherosclerosis. VBI is a term utilized to describe a reduction in blood flow to the vertebral and basilar arteries that supply the posterior cerebral system. For musculoskeletal clinicians, diagnostic differentiation of VBI is essential, because its presence directly impacts the clinical use of manual treatment interventions. Clinical guidelines provide a set of cardinal symptoms (inclusive of Coman's 5D's) in which VBI may manifest, the accuracy of which is under contestation because literature provides evidence suggesting a wider set of symptoms.

Objectives: The objectives of this study were to gather all relevant literature reporting features of VBI pertaining to atherosclerosis, with the aim to help provide evidence that may guide clinical practice in the use of manual therapy interventions and to raise awareness of the manifestations that VBI may present.

Methods: Six databases were searched from inception to September 2024 (Allied and Alternative Medicine Database [AMED], AgeLine, SPORTDiscus, Medical Literature Analysis and Retrieval System Online [MEDLINE], Cochrane, and Cumulative Index of Nursing and Allied Health (CINAHL Plus). Articles were screened in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) standards, The included articles required a diagnosis of VBI through clinical examination with radiological evidence of atherosclerotic lesions, without evidence of existing or previous neurological infarcts, concomitant arterial pathology, or any other form of pathological mechanism. Primary data were extracted utilizing a template, and the methodological quality was assessed utilizing the Joanna Briggs Institute critical appraisal tool. Findings were summarized utilizing a narrative synthesis and a table of descriptive statistics.

Results: Two hundred and eighty-three papers were identified, and 15 were included (93 cases, 50M/43F, age 64 years old ± 9 standard deviation [SD] yrs). Vertigo was the most common reported symptom, within a total of 37 different symptoms reported either in isolation or combination. Symptoms inclusive to Coman's 5D's accounted for 22 % of reported features.

Conclusions: Vertigo is the most common symptom (27.7 %) of VBI induced by atherosclerosis. However, there is not sufficient data to make concrete conclusions, although results do instill doubt over the sole use of Coman's 5D's in clinical practice. Prospective observational studies with standardized data extraction for VBI symptoms and their pattern of behavior are warranted.

Keywords: atherosclerosis; dizziness; symptoms; vertebrobasilar insufficiency; vertigo

Vertebrobasilar insufficiency (VBI) is a term that currently sits under the wider umbrella of 'cervical arterial insufficiency or dysfunction' and is defined as transitory ischemia of the vertebrobasilar circulation [1]. Initially, features of VBI were reported in early postmortem studies by Kubik and Adams [2], later to which the term 'vertebral basilar insufficiency' was introduced describing progressive occlusion of the basilar and distal vertebral vessels [3]. Reductions in hemodynamic flow through the vertebrobasilar (posterior) circulation can cause hypoxic damage to neurological, cardiorespiratory and vestibular systems within the brainstem and cerebellum. Many pathologies can result in VBI, most commonly atherosclerotic arterial stenosis [4], arterial dissection, hypoplasia, thromboembolism and mechanical compression [5].

Signs and symptoms of transient VBI are collectively diverse, yet evidence suggests that vertigo, termed as a subform of dizziness, is the most frequent clinical symptom of posterior circulation ischemia [6–8]. It is important to note

***Corresponding author: Chris Moors**, BSc, Keele University, Keele, Newcastle, Staffordshire, ST5 5BG, UK, E-mail: christopher.moors@nhs.net
Claire Stapleton, PhD, Keele University, Keele, Newcastle, Staffordshire, UK

that across wider literature, the terms dizziness and vertigo are utilized synonymously, potentially creating a degree of inaccuracy within the research. Ishiyama and Ishiyama [9] highlighted the importance of a clinician's ability to evaluate presenting forms of dizziness and to distinguish between its constituent types, vertigo and light-headedness. To help clarify this separation, definitions have been suggested. Typically *light-headedness* presents as an impending fainting sensation [9], this description of dizziness is non-specific and excludes senses of movement by the external environment. The term *vertigo*, conversely, indicates rotational movement of the external environment and is usually abrupt and spontaneous [9].

Contrary to reports from Heyman et al. [10] and Luxon [11], vertigo and dizziness have been reported to occur in the absence of other neurological signs [12] with isolated episodes of vertigo presenting as the initial feature of VBI in 19 % and 48 % of cases [6, 13]. When vertigo is present with other neurological symptoms, diagnosis of VBI is usually obvious, whereas, when vertigo occurs in isolation it becomes difficult to differentiate from other benign disorders involving the inner ear [6], i.e., vestibular disorders such as Ménière's disease, labyrinthitis and benign paroxysmal positional vertigo (BPPV).

Other common symptoms reported secondary to vertebrobasilar ischemia include auditory and visual disturbances, ataxia, dysphagia, hemiparesis, cardiorespiratory abnormalities [14] and severe cases can lead to stroke or death. Seminal work by Kubik and Adams [2] presented 18 case reports describing neurological infarcts through thromboembolic pathology and alluded to preceding transient symptoms due to basilar artery atherosclerosis. Similar work by Grad and Baloh [6] described posterior circulatory ischemia in two separate forms (transient episodes of VBI), or secondly as neurological infarcts, with VBI having potential to advance into brainstem infarction. For patients presenting with transient VBI symptoms, cerebrovascular causes of posterior ischemia should be considered, particularly in those with determined vascular risk factors; this may allow early medical optimization and thus reduce the risk of progression to neurological infarcts.

VBI features induced by atherosclerosis have the potential to mimic or co-exist with those of other pathologies, such as the aforementioned vestibular disorders, or cervical arterial dissection (CAD), the latter requiring emergency medical referral. Neck or facial pain alongside headaches (occipital/fronto-temporal) are typical initial signs indicative of CAD, which is the leading cause of stroke in healthy young patients [15–17]. Previous literature initially failed to differentiate between VBI induced by atherosclerosis and arterial dissection, leading to a lack of clarity within the evidence. Although attention into the risk of VBI in patients presenting with neck pain has been paid [18–21], differential diagnoses remains a difficult task. By focusing upon features of atherosclerotic-induced VBI, further evidence to assist in distinguishing between VBI resulting from atherosclerosis and arterial dissection is provided in this review.

Significantly for manual therapists, although features of atherosclerotic VBI in isolation do not masquerade as musculoskeletal pain, it can co-exist with musculoskeletal cervical spine pain and dysfunction. Completing a clear subjective assessment and gaining an accurate history of events is essential to effectively assess for any vascular compromise that occurs with and without additional mechanical compression from movements of the cervical spine. Existing evidence evaluating the efficacy of cervical functional positional tests historically utilized during physical examination to assess the vertebral arteries, no longer supports their use in practice [22]. The presence of VBI secondary to atherosclerosis raises precautions toward the use of cervical manual treatment interventions. Although attributed risks are low with cervical spine manipulation [23, 24] and adverse effects are rare, obtaining a clinically reasoned understanding of patient presentation is essential for safe manual treatment practice [25, 26].

To aid contemporary therapeutic treatment and manual therapy practice, features of VBI are reported to manifest themselves within a cardinal set of symptoms coined "Coman's 5D's": diplopia, dysarthria, dizziness, dysphagia, and drop attacks [27]. However, academic literature presents a far more varied set of clinical features and characteristics of VBI [28] and questions the accuracy of Coman's cardinal signs in clinical practice. Regardless of previous and current guidelines [29–31] and clinical usage, literature does not support the contention that practitioners can accurately identify those patients at risk of VBI [32]. Despite attempts to build a consistent evidence base and clinical picture of VBI, no consensus has yet been reached. This review systematically collected observational data regarding clinical symptoms reported in primary research to help clarify what constitutes a true feature of VBI induced by atherosclerosis and therefore aid differential diagnosis. Such information may offer further evidence and guidance for manual therapists, who at present face a cloud of uncertainty around the features, screening, and management of VBI.

Methods

Study identification

A literature search was conducted across six electronic databases: Allied and Alternative Medicine Database (AMED),

Table 1: Search strategy, exact terms, and combinations utilized to complete the literature search.

S1	(MeSH or keyword) VBI		
S2	Cervical arterial dysfunction, not CAD		
S3	(MeSH or keyword) vertebral artery		
S4	(MeSH or keyword) basilar artery		
S5	Intracranial circulation	S6	Posterior cerebral circulation
S7	Cerebrovascular disease	S8	Ischemia
S9	(MeSH or keyword) atherosclerosis	S10	Atheroma
S11	Stenosis	S12	Occlusion
S13	(MeSH or keyword) signs and symptoms	S14	Clinical features
S15	Clinical symptoms	S16	Clinical signs
S17	Clinical presentation	S18	Manifestations
S19	Signs	S20	Symptoms
S21	S1 or S2 or S3 or S4 or S5 or S6 or S7		
S22	S8 or S9 or S10 or S11 or S12		
S23	S13 or S14 or S15 or S16 or S17 or S18 or S19 or S20		
S24	S21 and S22 and S23 (*full-text, English-language, peer-reviewed articles*)		

CAD, cervical arterial dissection; MeSH, medical subject heading; VBI, vertebrobasilar insufficiency.

AgeLine, SPORTDiscus, MEDLINE, Cochrane, and Cumulative Index of Nursing and Allied Health (CINAHL) Plus. The search strategy utilized a combination of Medical Subject Headings (MeSH) terms and free-text terms relevant to VBI and atherosclerosis, including cervical arterial dysfunction, vertebral artery, basilar artery, intracranial circulation, posterior cerebral circulation, cerebrovascular disease, ischemia, atheroma, stenosis, occlusion, clinical features/signs/symptoms/presentation, manifestations, signs, and symptoms. The precise search strings utilized are demonstrated in Table 1. The initial search was undertaken in July 2019 by the first author (CM), and the results were reviewed by the second author (CS); no disagreements occurred between the authors. The search was repeated on four separate occasions between August 2019 and September 2024 to obtain any additional published records over that time.

Study selection

The inclusion and exclusion criteria ensured that all features reported were secondary to atherosclerotic stenosis of the vertebrobasilar territory alone (Table 2). Papers reporting a confirmed diagnosis through the use of the gold standard for the detection of posterior circulation disease and vertebral artery stenosis were included. The invasive technique of intra-arterial digital subtraction angiography (DSA) provides the current gold standard [33–36]. Noninvasive methods of transcranial Doppler ultrasound (TCD), magnetic resonance imaging (MRI), and computerized tomography angiography (CTA) [35] were also deemed appropriate to inform the diagnosis. To allow a full scoping review of the current literature, the search was open to all study designs with no exclusions.

Table 2: Study inclusion/exclusion criteria.

Inclusion criteria	Exclusion criteria
Signs/symptoms/features of VBI sufficiently reported in article	Presence of previous/existing neurological infarcts at time of report
VBI clinically diagnosed through clinical examination and VBI clinically diagnosed through radiological imaging	Presence of any concomitant collateral circulation pathology, i.e., internal carotid artery stenosis
Atherosclerotic lesions/stenosis present in vertebrobasilar vessels	Evidence of other pathological mechanism present or suspected, i.e., thrombus/embolism/mechanical compression/dissection/hypoplasia

VBI, vertebrobasilar insufficiency.

Given the relevance to manual-treatment screening and examination, it was vital that the reported symptoms presented prior to permanent neurological damage and occurred in the absence of other pathological mechanisms. Cases reporting collateral circulatory pathology, presence of neurological infarcts (previous or existing) at the time of the reports, or the presence of other pathological causes – ie, thromboembolic source – were excluded. Cases under medical management were included, because they were deemed pathologically consistent and practically relevant.

Screening process

Results from the database search were manually and systematically screened against the inclusion/exclusion criterion; following the exclusion of duplicates, an independent reviewer also verified the study selection. Reference lists of included articles were manually screened against the same criteria; the final selection was analyzed, and relevant data were extracted.

Data extraction and analysis

To ensure consistent and reliable data extraction, a bespoke form was devised. The following information was systematically extracted and tabulated: authors, year of publication, study design, length of study, the number of cases/patients, diagnostic technique, comorbidities and risk

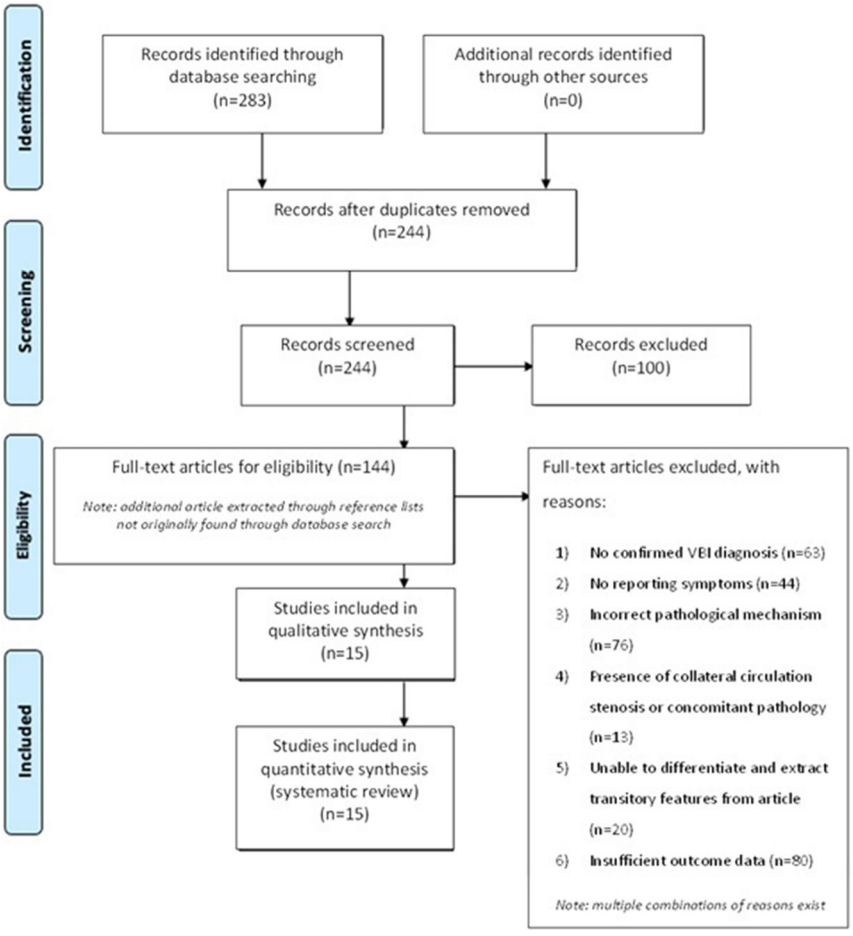

Figure 1: A Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram, showing the systematic research methodology and screening process.

factors, and symptoms and their characteristics (duration/frequency/stenosis rates). A second independent reviewer analyzed a sample of articles against the bespoke extraction form to ensure reliable data gathering. It was agreed by both reviewers that the extraction process was suitable and permitted the capture of all relevant information. A meta-analysis was not performed due to the substantial clinical and methodological heterogeneity of the included studies. A narrative synthesis was performed with results summarized descriptively.

Assessment of methodological quality

The methodological quality of the included studies was evaluated systematically utilizing the appropriate critical appraisal tools [37] based on the article study design, which in this instance were those designed for case controls, case series, and case reports. The Joanna Briggs Institute appraisal tools were utilized to provide a detailed blueprint to follow during the critique, thus allowing the rigorous assessment of the included articles to determine the levels of possible bias in each study and the extent to which these factors were addressed.

Results

Study selection

See Figure 1 for the selection and screening process. Following Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines [38], the database search identified 283 articles. After the removal of duplicate articles (n=33) and excluding articles failing to meet the research criteria plus articles agreed to be unsuitable by independent reviewer (n=1), 15 studies remained, in which 93 individual cases were reported and information was systematically extracted (Table 3). The patient demographics were 50 male and 43 female cases, and the average age was 64 years old (SD=9), ranging from 39 to 81 years old.

Table 3: Information extracted from original studies selected for appraisal.

Author/year/title (chronological)	Study type	Length of study	No. patients/ cases	Method of VBI diagnosis	Risk factors	Signs and symptoms reported	Symptom characteristics (duration/frequency/stenosis % rate)
1) Myers 1977 [39]	Case series	No details provided	8 Cases in total	Four-vessel angiography	No risk factors reported	Drop attacks	Symptom duration/frequency not reported
Reconstruction of vertebral artery stenosis			Case 1 met inclusion criteria Age 63 M				RVA and LVA stenosis on arteriogram No stenosis % rates
2) Grad and Baloh 1989 [6]	Chart review/	1974–1987 records from neuro-otology clinic, retrospective	84 cases in total	Referred due to episodes of vertigo	Risk factors available for select cases, no	Vertigo	(Detailed clinical features listed in text)
Vertigo of vascular origin	Case series	review	42 cases meet the inclusion criteria	Clinical examination and diagnosis of VBI	overall data included	Visual dysfunction	35 cases reported symptoms lasting minutes
			VBI group	CT/MRI when clinically indicated		Drop attacks	4 cases reported seconds
			n=42 Age 68 ± 9 y 17 M 25 F	16 cases angiography utilized		Unsteadiness Extremity weakness Confusion Headache Hearing loss Loss of consciousness Extremity numbness Dysarthria Tinnitus Perioral numbness	5 cases reported hours 26 reports of isolated vertigo, which ranged from 2 days to 1.5 years prior to other neurological symptoms
3) Comerota and Maurer 1992 [40]	Case report	No given length of study or follow-up	2 patients described	Four-vessel angiography	No risk factors reported although history of:	Vertigo only	Symptom frequency not reported in detai
Surgical correction and SPECT imaging of vertebrobasilar insufficiency due to unilateral vertebral artery stenosis			Case 1 met inclusion Age 64 F and 71 F	SPECT scan	Coronary artery bypass, carotid endarterectomy and depression	(Incapacitating)	3-month history of vertigo CT showed isolated RVA origin stenosis
4) Fife et al. 1994 [41]	Case reports	Patients with cerebral angiography at the time of symptoms identified, follow up and medical response recorded (records up to 4 years post initial presentation)	7 Case reports in total	MRA	Case 5:	Episodic dizziness	Variable across Cases 5-7
Isolated dizziness in VBI: clinical features, angiography, and follow-up		See Figure 6	Case 5–7 met inclusion criteria	MRI TCD	HTN Hyperlip	Isolated dizziness Dizziness (occurring with all attacks)	Duration: x1 episode of vertigo and diplopia 3 years prior to presentation

Table 3: (continued)

Author/year/title (chronological)	Study type	Length of study	No. patients/ cases	Method of VBI diagnosis	Risk factors	Signs and symptoms reported	Symptom characteristics (duration/frequency/stenosis % rate)
			Age median 62yo M	Cerebral angiography	Angina	Vertigo	Reports dating back 3months - 4 years
					Case 6/7:	Imbalance	Episodes of dizziness lasting 1min-10 min s
					No reports	Diplopia	Symptoms varying from 1 min–20 min
						Confusion	Frequency:
						Nausea	Between 4 and 7 times daily
						Diffuse headaches	Resolution after 2 weeks reported (Case7)
						Extremity numbness	Increases in frequency and intensity reported
						Transient memory loss	
						Bilateral loss of vision	
						Facial numbness	
5) Kumar et al. 1998 [12]	Case Control study	7 years, retrospective data regarding transient ischemic attacks of the vertebrobasilar system causing dizziness/vertigo and imbalance	27 cases	All at minimum contrast enhanced MRI, some additional imaging, e.g., 4-vessel cerebral angiogram, MRA	HTN 64 %	Vertigo 64 % (Postural-induced imbalance vertigo 50 %)	Symptom duration/frequency not reported
Diagnosis of VBI: time to rethink established dogma?			14 cases met inclusion Age 48-80 7 M 7 F	Investigated vestibular and audiometric data	Heart disease 36 % DM 29 % Smoking 21 % Hyperlip 14 % (n=14)	Imbalance 43 % Hearing loss 29 % Nausea 14 % Syncope 14 % Blurred vision 7 % Tinnitus 7 %	
6) Malek et al. 1999 [42]	Case series	5-year retrospective period from 1994 to 1999, patients included in study presented with posterior-circulation ischemia that failed medical management	21 patients in study	DSA or computational analysis of procedure	HTN	Dizziness	Symptom duration/frequency not reported
Treatment of posterior circulation ischemia with extra cranial percutaneous balloon angioplasty and stent placement			3 cases met inclusion criteria Age 65 F/59 F 60 M		Smoking	Vertigo	Stenosis rates: Preoperative
					Hyperchol	UL extremity numbness	Case 16: LVA 80 %
					Vehicular whiplash	Imbalance	Case 19: RVA 60 %
						Staggering gait	Case 20: RVA 80 %
						Pre-syncope on head turn	Postoperative Case 16/19/20: 0 % residual stenosis
7) Nahser et al. 2000 [43]	Case series	1997-1999 analyzed retrospectively	20 cases in total	Assumed MRI or CT scan	HTN 16/20	Diplopia	Symptom duration/frequency not reported
				DSA or MRA follow-up	Hyperlip 12/20	Hemiparesis	Stenosis rates

Table 3: (continued)

Author/year/title (chronological)	Study type	Length of study	No. patients/ cases	Method of VBI diagnosis	Risk factors	Signs and symptoms reported	Symptom characteristics (duration/frequency/stenosis % rate)
Intracranial vertebrobasilar stenosis: Angioplasty and follow-up		PTA indicated in 18/20 cases due to progression and symptoms of stenosis 2/20 cases asymptomatic	13 cases met inclusion criteria Age 41-74 M and F		Obesity 1/20 DM 2/20 Smoking 11/20 CHD 5/20 Not Case sensitive	Dysarthria Vertigo Hypesthesia Facial paresis	Preoperative: 80–95 % stenosis Postoperative: 0–40 % residual stenosis Follow-up: 0–20 % with 1 case of >90 % stenosis at 12 months Follow-up date varied across cases
8) Rasmussen et al. 2000 [44] Stent-assisted angioplasty of intracranial vertebrobasilar atherosclerosis: an initial experience	Case report (within a Case series)	July 1998 and September 1999, Those patients with VBA stenosis who were unresponsive to medical therapy were put forward for stent-assisted angiography	8 cases in total Case 7 met inclusion criteria Age 75 M	MRI	No reports	−200 episodes of positional transient tetraparesis Facial/perioral numbness Occipital headache Dysarthria Syncope	All experienced over preceding 2 years No symptom duration reported Syncope to presyncope ×3 daily while on medical management Radiographic studies showed preoperative: Occlusion of LVA High-grade stenosis RVA (82 % stenosis) Postoperative: Residual stenosis of RVA (22 % stenosis)
9) Gress et al. 2002 [45] Angioplasty for intracranial symptomatic vertebrobasilar insufficiency	Case series (with two case reports)	13-year retrospective study, those undergoing angioplasty for critical intracranial vertebral or basilar artery stenosis. Clinical features and post-operative complications recorded	25 cases 2 case reports met the inclusion criteria Age 50–87	4-vessel angiogram	Unable to extract risk-factor data relevant to cases 1 and 2	Case 1: R sided weakness/unsteady gait (20 min)/ bilateral visual obscuration and periorbital pain (40 min) Complete R side hemiplegia and dysarthria (20 min)	Case 1: 20–30 min progression leading up to 40 min in duration Case 2:

Table 3: (continued)

Author/year/title (chronological)	Study type	Length of study	No. patients/ cases	Method of VBI diagnosis	Risk factors	Signs and symptoms reported	Symptom characteristics (duration/frequency/stenosis % rate)
						Case 2:	20–30 min in duration, tended to occur after large meals
						Diplopia/Vertigo/ Hemiparesis Weakness either R or L side	No data regarding timeline for which symptoms reoccurred.
10) Lee and Baloh, 2003 [46]	Case report	Approx 1 week of symptoms	1 case	MRA confirmed stenosis	DM	Vertigo	Fluctuating and episodic features
Sudden bilateral simultaneous deafness with vertigo as a sole manifestation of VBI		Single case study presented	Rare presentation	Extensive investigations ruled out all else:	HTN	Vomiting	Initially transient vertigo
			Age 68 F	-Blood tests		Bilateral tinnitus	Worsening to sudden vertigo/vomit/tinnitus
				-Liver function tests		Hearing loss	Bilateral hearing loss
				-Urea nitrogen		Dizziness	Vertigo lasted 2 h, hearing loss persisted
				-ESR/C-reactive, etc			Dizziness and hearing loss on admission
							Stenosis: Severe stenosis of middle third of basilar and blockage of the distal VA
11) Dabus et al. 2006 [47]	Case series	June 1996-Sept 2005 those treated with angioplasty due to failed medical management	25 cases in total	All cases confirmed utilizing DSA	HTN	Dizziness n=3	Symptom duration/frequency not reported
Endovascular treatment of the vertebral artery origin in patients with symptoms of vertebrobasilar ischemia			4 Cases met the inclusion criterion		DM	Ataxia n=1	Stenosis rates:
			Avg. Age 76		Hyperchol	Mental status change n=1	Preoperative: 70–95 % stenosis
			3F, 1M		Not case-sensitive		Postoperative: 0 % residual all cases
							Mean follow-up: 24 months Range: 2–96 months
12) Starke et al. 2009 [48]	Case report	Rare case report of symptomatic bilateral vertebral occlusion presenting as VBI	1 Case report	MRA	No risk factors reported	Dizziness (sitting to standing)	Mild dizziness symptoms initially, progressed to more severe dizziness and other symptoms
Occipital artery-to-posterior inferior cerebellar artery bypass for treatment of bilateral vertebral artery			Age 39 M	Followed by confirmation by non-invasive optimal vessel analysis (NOVA)	No significant medical history	Vertigo Imbalance Diplopia	

Table 3: (continued)

Author/year/title (chronological)	Study type	Length of study	No. patients/ cases	Method of VBI diagnosis	Risk factors	Signs and symptoms reported	Symptom characteristics (duration/frequency/stenosis % rate)
13) Inoue et al. 2012 [49]	Case series (with single case report)	Retrospective March 2008 – Feb 2012 patients treated for infarcts, VBA occlusion 0.8 %	9 cases	MRI/CT/DSA	HTN (9/9)	Dizziness	Symptom duration varied from patient to patient, ranging from 2-10 h
Acute to sub-acute surgical revascularisation for progressing stroke in atherosclerotic vertebrobasilar occlusion			Case 9 met the inclusion criteria	DWI (monitor disease progression)	DM (6/9)	Diplopia	In case description, memory disturbance was reported several days preceding admission.
			Age 56 M	No CT/MRI for hemodynamic data; evidence only for anterior cerebral circulation	Hyperlip (5/9) Smoking (4/9)	Memory disturbance	
14) Britze et al. 2015 [50]	Case series	May 2011-Oct 2011	3 cases in total	CTA	No risk factors reported, history of:	Vertigo with associated:	Symptom duration/frequency not reported
Radial artery bypass for intractable VBI: case series and review of the literature		Rare subset of VBI, those with complete occlusion of VA and who fail medical management.	Case 3 met inclusion	DSA	Coronary artery disease	Weakness/numbness in hands B/L	Symptoms worsened when standing
			Age 73 M	Repeated 3D MRA for follow up	(Severe)	Weakness/numbness both UL and LL	Stenosis rates:
						Ataxia	High-grade stenosis RVA origin
						(Could not ambulate independently)	Moderate stenosis in the midbasilar
							Complete occlusion LVA
15) Cai et al. 2018 [51]	Case series	Retrospective 2009–14, with 6–12 months of follow-up postangioplasty	5 cases	DSA	HTN (5/5)	Vertigo	Duration/intensity not documented
Balloon-expandable stent angioplasty in the treatment of vertebral artery stenosis in the V2 segment			All included	CTA	DM (2/5)	Hemiparesis	Stenosis rates measured utilizing TCD
			Age 54–75 M & F	TCD		Hemiplegia	Preoperative mean (87.0 ± 6.6 %)
				Ruled out any osseous compression at V2 segment, infarct within 4-week data excluded		Dysarthria	Residual mean (12.6 ± 7.8 %)
						Dysphagia	TCD and CTA follow-up at: 1 week/1 month/3 & 6 months
						Dizziness	
						Unsteady gait	

M, male; F, female; DSA, digital subtraction angiography; CT, computed tomography; CTA, computed tomography angiography; ESR, erythrocyte sedimentation rate; TCD, transcranial Doppler; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; DWI, diffusion-weighted imaging; PTA, percutaneous transluminal angioplasty; SPECT, single photon emission computerized tomography; HTN, hypertension; DM, diabetes mellitus; Hyperlip, hyperlipidemia; Hyperchol, hypercholesterolemia; CHD, coronary heart disease; LVA, left vertebral artery; RVA, right vertebral artery; LL, lower limb; UL, upper limb; VBI, vertebrobasilar insufficiency.

Study characteristics

Of the 15 articles, three were published within the last decade and six were published within the last 20 years. The study designs utilized for the manuscripts were case series (n=9), case reports (n=5) and case control studies (n=1). Cases either explored revascularization techniques in those with severe atherosclerotic stenosis who had failed medical intervention or who investigated patients presenting with features of vertigo/dizziness. The cases provide clear symptom reports; however, 10 failed to present sufficient detail regarding symptom duration, frequency, and progression. Case series report information regarding stenosis rates (%) present in the vertebral and basilar arteries provided additional anatomical insight into the atherosclerotic process.

To confirm the clinical diagnosis of VBI, nine different imaging techniques were utilized, and in many cases, multiple methods were utilized. In 55 cases, it was not possible to identify the precise frequency of methods utilized to confirm the diagnosis; thus, these data were not included and have no bearing on the following information. The three commonly utilized diagnostic imaging techniques were MRI (29.8 %), the gold-standard DSA (21.1 %), and CTA (12.3 %); see Table 4. In total, 37 different symptoms were reported; either presenting in isolation or combination. Original symptom reports can be found in Table 3. Vertigo (27.7 %), unsteadiness/incoordination (7.4 %), and diplopia (7.0 %) were the most frequently reported symptoms of VBI. Symptoms associated with 1) vertigo and dizziness (including posture induced vertigo, syncope), 2) visual dysfunction (diplopia, illusions, hallucinations, field defects, complete vision loss and blurred vision) and 3) cognitive dysfunction (confusion, loss of consciousness, memory disturbance, mental status change, dysarthria, dysphagia and drop attacks) accounted for 32.5 % (n=88), 17.7 % (n=48) and 15.5 % (n=42) of reports, respectively (Figure 2).

Table 4: Diagnostic radiographic imaging techniques.

Radiographic imaging technique	No. of cases	Frequency (total n=57) %
Magnetic resonance imaging (MRI)	17	29.8
Digital subtraction angiography (DSA)	12	21.1
Computerized tomography angiography (CTA)	7	12.3
Transcranial doppler ultrasound (TCD)	6	10.5
Magnetic resonance angiography (MRA)	6	10.5
4-Vessel angiography	4	7.0
Cerebral angiography	3	5.3
Diffusion-weighted imaging (DWI)	1	1.8
Single photon emission computerized tomography (SPECT)	1	1.8

The general characteristics of reported symptoms, i.e., duration, frequency and progress over time, range extensively throughout the results with no clear pattern apparent; the duration of symptom episodes ranged from 1 min to 40 min [6, 41, 45], to 2–10 h [49]. The frequency of episodes ranged from one to seven times daily [41]; among cases reporting a 3-month history of VBI episodes [40] to 2–3 year history of preceding symptoms [41, 44]. One study reported 200 fluctuating episodes over a 2-year period [40], while another indicates resolution of symptoms after 14 days from the initial onset [41]. Common risk factors reported by cases (n=67) included hypertension 40.3 %, diabetes mellitus 19.4 %, and smoking 13.4 % (Table 5).

Methodological quality

The included studies demonstrated moderate to good levels of reliability. Levels of research evidence ranged through observational analytic designs and descriptive studies (Levels III-IV) in accordance to The Joanna Briggs Institute Critical Appraisal Tool [37]; limitations inevitably exist across case series/reports. Due to small sample sizes, 7 out of 15 reporting a singular suitable case results (Table 3; Article no. 1, 3, 8, 10, 12–14) lack research power and provide little basis to generalize findings to the wider population. Selection bias also limits the wider application of results because the sample selection limited by the characteristics of each study unavoidably limits the external validity.

All cases utilized standardized and validated methods to confirm VBI; patient demographics and clinical information were sufficiently presented. The inclusion criteria were not explicit across all cases series, as complete inclusion (Table 3; Article no. 6, 7, 9, 13, and 15) and consecutive inclusion (Table 3; Article no. 6, 11, and 13) were reported. In the remaining cases, the inclusion criteria were unclear, reducing the reliability of results.

Those studies observing patients over a follow-up period of time to monitor symptoms or stenosis rates generally demonstrated a large variance in quality; either the protocol was not standardized and inconsistently measured (n=5), the data were not collected for all patients (n=1), and/or the short-term effects were recorded (n=1), potentially damaging the internal validity and reliability of outcomes. Given the primary objectives and outcome measures across cases – ie, stenosis rate, hemodynamic flow, symptom resolution – statistical analysis was not performed in 14 cases. The case control study [12] utilized independent blinded assessment of radiological images to improve internal reliability; outcome measures were standardized across comparison groups. The control group was only utilized as a baseline comparison for

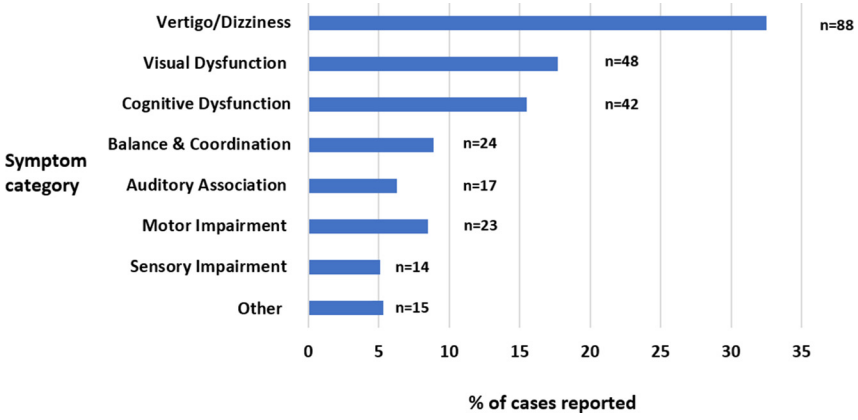

Figure 2: Frequency of symptom of vertebrobasilar insufficiency (VBI) grouped by category, as a percent of the total reported (n=271).

Table 5: Stroke risk factors/comorbidities reported.

Risk factors	No. of cases	Frequency (total n=67) %
Hypertension	27	40.3
Diabetes mellitus	13	19.4
Smoking	9	13.4
Hyperlipidemia/hypercholesterolemia	7	10.4
Coronary heart disease/artery disease	6	9.0
Angina	2	3.0
Alcoholism	1	1.5
Depression	1	1.5
No significant history	1	1.5

Table 6: Coman's 5D's frequency data, reported across 93 cases.

Features of Coman's 5D's	No. of cases	Frequency (n=54) %
Diplopia	17	7.0
Drop attacks	15	4.5
Dysarthria	11	6.2
Dizziness	10	4.1
Dysphagia	1	0.4
Total inclusive of 5Ds only:	54 (from 93 cases)	22.2 %

vestibular testing, and the study was unable to measure perfusion rate.

Discussion

Clinical findings

The aim of this systematic review was to summarize the reported features of atherosclerotic-induced VBI. In turn, this will provide evidence to help clinicians successfully identify atherosclerotic-induced VBI, aid differential diagnosis, and guide the use of manual therapy interventions.

In the literature reviewed, 37 different symptoms were reported. Vertigo was the most common. The symptoms inclusive to Coman's cardinal signs accounted for 22.2 % of all symptoms (Table 6). A large percentage of symptoms reported in the literature are not sufficiently represented through the use of Coman's cardinal signs, perhaps indicating a risk of misidentification of VBI and therefore potential clinical mismanagement. Findings suggest that the current literature does not support the sole use of Coman's cardinal signs in the clinical detection of VBI secondary to atherosclerosis. Further to this, it perhaps suggests a need to re-evaluate the terms constituting Coman's 5D's because these are still inherent within medical and manual treatment (physiotherapy, osteopathy, chiropractic) education and teaching. Results here imply that relying on Coman's 5D's in isolation is not sufficient to reliably raise clinical suspicion and inform the safe use of manual therapy intervention. Musculoskeletal and manual therapists need to place large value upon the ability to take an accurate patient history in order to build a reliable clinical picture in which to draw clinical conclusions.

The commonality of vertigo as a symptom of VBI raises questions as to whether evidence is sufficient to advance the information provided within the Coman's cardinal signs and whether "vertigo" warrants a greater degree of acknowledgement. Having said that, there needs to be an element of caution. Within the literature, there appears to be an inconsistent use of terminology and an unclear distinction between terms associated with vertigo, and operational definitions are rarely provided. Due to the ambiguity of whether reports of dizziness constituted vertigo or lightheadedness, this review kept the terms separate. A lack of precision in research terminology was noted by Kerry and Taylor [52], who further emphasized the importance of differentiating the nature of dizziness in order to establish whether a vascular or nonvascular cause is present.

Misinterpretation across the literature may lead to incorrect representation and carryover of inaccurate information, potentially compromising the validity of this review.

The high incidence of vertigo may reflect the selection bias present within the studies; combined with the retrospective nature and study heterogeneity present, this suggests that caution should be made when drawing conclusions. The case series described by Grad and Baloh [6] accounted for 62 % of vertigo reports, thus possessing far more weighting on results comparatively to individual cases. Excluding all cases from Grad and Baloh [6] makes little change in the overall results; the comparative margins between symptom frequencies are altered, yet vertigo remains the most common symptom.

Vertigo as a feature of VBI

Four cases reported isolated episodes of vertigo and dizziness secondary to atherosclerotic stenosis in the posterior cerebral circulation (Table 3; Article no. 2–5). Previous understanding [10, 11, 52] suggested that vertigo should not be attributed to VBI unless it is in the presence of other neurological signs in the brainstem. However, results of this review in addition to previous reports [13, 53] provide contradicting evidence that episodes of isolated vertigo can be featured in VBI induced by posterior circulation ischemia.

Risk factors of VBI

Diabetes and smoking were identified as common risk factors, with hypertension (40.3 %) being the most frequent [54]. Coronary artery disease and hyperlipidemia were also noted, which also increases the risk of VBI [54]. The results of this study resembles reports in the 'New England Posterior Circulation Registry' outlined by Ishiyama and Ishiyama [9]. However, a volume of data remains unaccounted for, as the information was either not reported or could not be extracted accurately in 49 % of the cases. Nevertheless, a strong link between risk factors and vertebrobasilar ischemia exists and assists in identifying those at risk of VBI through vascular compromise.

Research restrictions and limitations

Volumes of literature exist containing information that could better inform results and improve study reliability. Regrettably, in 20 of the studies reviewed, transient VBI reports were grouped with neurological infarct data that could not be extracted and were therefore excluded from this paper. Cases investigating arterial revascularization techniques lacked detail describing symptom pattern and timelines preceding the intervention. Given the objectives of such studies, the lack of information was understandable; however, a potential opportunity to build the clinical picture of VBI may have been missed. Symptom duration is particularly useful regarding dizziness and vertigo, because duration can help determine whether symptoms are caused by transient ischemia or a benign inner ear disorder [41].

Cases in this review were subject to selection bias due to their retrospective design, ultimately affecting the review in many ways. Although information generally was systematically retrieved, missing or incomplete information was a common issue. Results highlight the sporadic and inconsistent pattern of VBI features and potential progression, and the small number of accurate reports limits the reliability and external validity of this information. Studies included patients who typically failed medical management and possessed high vertebrobasilar stenosis rates. Thus, there is a gap in the literature regarding those with mild or moderate arterial stenosis who are not under medical management. Studies reported presenting episodes of VBI across a time span of 3 months to 3 years, indicating that features of VBI may occur in earlier stages of vertebrobasilar atherosclerosis. Information on this patient population in which VBI has not yet been identified is very relevant. Selection bias exists toward patients specifically presenting with vertigo; thus, the conclusions drawn may not be reliably applicable to those other cases in which vertigo is not a prominent feature.

Recommendations for future research and practice

Resolution toward distinguishing the features of vertigo and other forms of dizziness is warranted, and future research should provide clear definitions alongside the reports. The need for clarity is outlined and exemplification is provided in this review and in previous studies [9, 52, 53]. Considering that vertigo as a singular phenomenon separate from dizziness is perhaps justified; however, given the common practical use of Coman's cardinal signs, it may be more pragmatic to distinguish the terms through clinical education and emphasize the distinct characteristics of each. Additionally, improving the knowledge of known arterial presentations and the diverse features of VBI could strengthen the clinician's differential diagnostic skills, a notion also proposed by Kerry et al. [28].

To help develop a stronger evidence base, future clinical case studies reporting VBI should present greater levels of relevant clinical data and clearly differentiate the symptoms relative to transient ischemia, neurological infarcts, and arterial dissection. Reports should collect accurate information pertaining to the pattern, duration, and timeline of VBI symptoms. Such information would greatly benefit differential diagnostic processes. The future use of cohort study designs (prospective observational studies) tailored toward the retrieval of symptom pattern data may permit complete data collection; the need for larger sample sizes and the risk of attrition bias would need to be considered to avoid compromising the validity of the potential results.

Conclusions

Vertigo was reported as the most common symptom of VBI caused by atherosclerosis. This systematic review identified the need for further observational studies detailing the features and characteristics of patients with VBI. There is now insufficient data regarding VBI resulting from atherosclerotic stenosis, so accurate and reliable conclusions cannot be drawn. Current evidence presents an extremely diverse and multifactorial clinical picture of VBI, which cannot be clinically represented through Coman's 5D's alone. Academic and clinical attention needs to be paid to the correct use and distinction of the terms "dizziness" and "vertigo." The separation of these features within VBI and Coman's signs is justified, although results of this study are not sufficient to accurately inform contemporary clinical guidelines. This review raises awareness for musculoskeletal clinicians about how symptoms of VBI secondary to atherosclerosis manifest, providing aid in the diagnostic differentiation between that and features of vestibular disorders and CAD. The results of this study highlight common risk factors of VBI that should be considered in the clinical decision-making process. If present, this should lead to increased clinical suspicion and caution when considering manual therapy techniques as an intervention. Atherosclerosis rates increase with age, although it is evident that multiple non–age-related cardiovascular factors – ie, smoking, alcohol intake, and obesity – also need to be considered to inform clinical decisions and differential diagnoses, prior to the use of manual treatment techniques.

Research ethics: The Keele University Research Ethics Committee (Institutional Review Board) deemed the study exempt from review.
Informed consent: Not applicable.
Author contributions: All authors have accepted responsibility for the entire content of this manuscript and approved its submission.
Use of Large Language Models, AI and Machine Learning Tools: None declared.
Conflict of interest: None declared.
Research funding: None declared.
Data availability: Not applicable.

References

1. Special report from the National Institute of Neurological Disorders and Stroke. Classification of cerebrovascular diseases III. Stroke 1990;21:637–76.
2. Kubik CS, Adams R. Occlusion of the basilar artery: a clinical and pathological study. Brain 1946;69:74–121.
3. Millikan CH, Siekert RG. Studies in cerebrovascular disease. I. The syndrome of intermittent insufficiency of the basilar arterial system. Proc Staff Meet Mayo Clin 1955;30:61–8.
4. Moubayed SP, Saliba I. Vertebrobasilar insufficiency presenting as isolated positional vertigo or dizziness: a double-blind retrospective cohort study. Laryngoscope 2009;119:2071–6.
5. Kuether TA, Nesbit GM, Clark WM, Barnwell SL. Rotational vertebral artery occlusion: a mechanism of vertebrobasilar insufficiency. Neurosurgery 1997;41:427–33.
6. Grad A, Baloh RW. Vertigo of vascular origin: clinical and electronystagmographic features in 84 cases. Arch Neurol 1989;46: 281–4.
7. Searles DE, Pazdera L, Korbel E, Vysata O, Caplan LR. Symptoms and signs of posterior circulation ischemia in the New England medical centre posterior circulation registry. Arch Neurol 2012;69:346–51.
8. Schneider PA, Rossman ME, Bernstein EF, Ringlestein EB, Torem S, Otis S. Non-invasive evaluation of vertebrobasilar insufficiency. JUM 1991;10:373–9.
9. Ishiyama G, Ishiyama A. Vertebrobasilar infarcts and ischemia. Otolaryngol Clin 2011;44:415–36.
10. Heyman A, Leviton A, Millikan CH, Nefzger D, Ostfeld AM, Sahs AL, et al. Transient focal cerebral ischemia: epidemiological and clinical aspects. Stroke 1974;5:277–87.
11. Luxon LM. Signs and symptoms of vertebrobasilar insufficiency. In: Hofferberth B, Brune GG, Sitzer G, Weger H-D, editors. Vascular brain stem diseases. Basel: Karger; 1990:93–111 pp.
12. Kumar A, Mafee M, Dobben G, Whipple M, Pieri A. Diagnosis of vertebrobasilar insufficiency: time to rethink established dogma? Ear Nose Throat J 1998;77:966–74.
13. Williams D, Wilson TG. The diagnosis of the major and minor syndromes of basilar insufficiency. Brain 1962;85:741–74.
14. Mitchell J. The vertebral artery: a review of anatomical, histopathological and functional factors influencing blood flow to the hind brain. Physiother Theory Pract 2005;21:23–6.
15. Park KW, Park JS, Hwang SC, Im SB, Shin WH, Kim BT. Vertebral artery dissection: natural history, clinical features and therapeutic considerations. The Korean Neuro Society 2008;44:109–15.
16. Thomas LC, Rivett DA, Attia JR, Parsons M, Levi C. Risk factors and clinical features of craniocervical arterial dissection. Man Ther 2011;16: 351–6.

17. Saeed AB, Shuaib A, Al-Sulaiti G, Emery D. Verterbral artery dissection: warning symptoms, clinical features and prognosis in 26 patients. Can J Neurol Sci 2000;27:292–6.
18. Di Fabio RP. Manipulation of the cervical spine: risks and benefits. Phys Ther 1999;79:50–65.
19. Haldeman S, Kohlbeck FJ, McGregor M. Risk factors and precipitating neck movements causing vertebrobasilar artery dissection after cervical trauma and spinal manipulation. Spine 1999;24:785–94.
20. Haldeman S, Kohlbeck FJ, McGregor M. Unpredictability of cerebrovascular ischemia associated with cervical spine manipulation therapy: a review of sixty four cases after cervical spine manipulation. Spine 2002;27:49–55.
21. Hurwitz EL, Aker PD, Adams AH, Meeker WC, Shekelle PG. Manipulation and mobilization of the cervical spine; a systematic review of the literature. Spine 1996;21:1746–60.
22. Hutting N, Kerry R, Coppieters MW, Scholten-Peeters GGM. Considerations to improve the safety of cervical spine manual therapy. Musculoskelet Sci Pract 2018;33:41–5.
23. Carey P. A report on the occurrence of cerebral vascular accidents in chiropractic practice. J Can Chiropr Assoc 1993;34:104–6.
24. Haldeman S, Carey P, Townsend M, Papadopoulos C. Arterial dissections following cervical manipulation: the chiropractic experience. CMAJ (Can Med Assoc J) 2001;165:905–6.
25. Puentedura EJ, March J, Anders J, Perez A, Landers MR, Wallmann HW, et al. Safety of cervical spine manipulation: are adverse events preventable and are manipulations being performed appropriately? A review of 134 case reports. J Man Manip Ther 2012;20:66–74.
26. Rushton A, Rivett D, Carlesso L, Flynn T, Hing W, Kerry R. International framework for examination of the cervical region for potential of cervical arterial dysfunction prior to orthopaedic manual therapy intervention. J Manual Therapy 2014;19:222–8.
27. Coman WB. Dizziness related to ENT conditions. In: Grieve GP, editor. Modern manual therapy of the vertebral Column. Edinburgh, UK: Churchill Livingstone; 1986.
28. Kerry R, Taylor AJ, Mitchell J, McCarthy C, Brew J. Manual therapy and cervical dysfunction, direction for the future: a clinical perspective. J Manual and Manipulative therapy 2008;16:39–48.
29. Australian Physiotherapy, Association (APA) Guidelines, Thomas L, Shirley D, Rivett D. Clinical guide to safe manual therapy practice in the cervical spine. 2017. https://www.physiotherapy.asn.au/cervicalspine.
30. Rushton A, Carlesso LC, Flynn T, Hing WA, Rubinstein SM, Vogel S, et al. Position statement: international framework for examination of the cervical region for potential of vascular pathologies of the neck prior to musculoskeletal intervention: international IFOMPT cervical framework. J Orthop Sports Phys Ther. 2023;53:7–22.
31. Magarey ME, Coughlan B, Rebbeck T. Clinical guidelines for pre-manipulative procedures for the cervical spine. Melbourne, Australia: Australian Physiotherapy Association; 2000.
32. Childs JD, Flynn TW, Fritz JM, Piva SR, Whitman JM, Wainner RS, et al. Screening for vertebrobasilar insufficiency in patients with neck pain: manual therapy decision-making in the presence of uncertainty. J Ortho Sports Phys 2005;35:300–6.
33. Bhadelia RA, Bengoa F, Gesner L, Patel SK, Uzun G, Wolpert SM, et al. Efficacy of MR angiography in the detection and characterization of occlusive disease in the vertebrobasilar system. JCAT 2001;25:458–65.
34. Caplan LR, Wityk RJ, Pazdera L, Chang HM, Pessin MS, DeWitt LD. New England medical center posterior circulation stroke registry II. Vascular lesions. J Clin Neurol 2005;1:31.
35. Cloud GC, Markus HS. Diagnosis and management of vertebral artery stenosis. QJM; International J Med 2003;96:27–34.
36. Khan S, Rich P, Clifton A, Markus HS. Noninvasive detection of vertebral artery stenosis. Stroke 2009;40:3499–503.
37. Briggs J. The Joanna Briggs institute critical appraisal tool. University of Adelaide; 2017. [Online]. [20/11/2019]. Available from: https://jbi.global/critical-appraisal-tools.
38. Moher D, Liberati A, Tetzlaff J, Altman DG, PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ 2009;339:b2535.
39. Myers KA. Reconstruction of vertebral artery stenosis. Aust N Z J Surg 1977;47:41–8.
40. Comerota AJ, Maurer AH. Surgical correction and SPECT imaging of vertebrobasilar insufficiency due to unilateral vertebral artery stenosis. Stroke 1992;23:602–6.
41. Fife TD, Baloh RW, Duckwiler GR. Isolated dizziness in vertebrobasilar insufficiency: clinical features, angiography, and follow up. J Stroke & CVD 1994;4:4–12.
42. Malek AM, Higashida RT, Phatouros CC, Lempert TE, Meyers PM, Gress DR, et al. Treatment of posterior circulation ischemia with extracranial percutaneous balloon angioplasty and stent placement. Stroke 1999;30:2073–85.
43. Nahser HC, Henkes H, Weber W, Berg-Dammer E, Yousry TA, Kühne D. Intracranial vertebrobasilar stenosis: angioplasty and follow up. AJNR (Am J Neuroradiol) 2000;21:1293–301.
44. Rasmussen PA, Perl II J, Barr JD, Markarian GZ, Katzan I, Sila C, et al. Stent-assisted angioplasty if intracranial vertebrobasilar atherosclerosis: an initial experience. J Neurosurg 2000;92:771–8.
45. Gress DR, Smith WS, Dowd CF, Halbach VV, Finley RJ, Higashida RT. Angioplasty for intracranial symptomatic vertebrobasilar ischemia. Neurosurgery 2002;51:23–9.
46. Lee H, Yi HA, Baloh RW. Sudden bilateral simultaneous deafness with vertigo as a sole manifestation of vertebrobasilar insufficiency. JNNP 2003;74:539–41.
47. Dabus G, Gerstle RJ, Derdeyn CP, Cross III DT, Moran CJ. Endovascular treatment of the vertebral artery origin in patients with symptoms of vertebrobasilar ischemia. J Intervent Neuroradiol 2006;48:917–23.
48. Starke RM, Chwajol M, Lefton D, Sen C, Berenstein A, Langer DJ. Occipital artery-to-posterior inferior cerebellar artery bypass for treatment of bilateral vertebral artery occlusion: the role of quantitative magnetic resonance angiography non-invasive optimal vessel analysis, a technical case report. J Neurosurg 2009;64:E779–81.
49. Inoue T, Tamura A, Tsutumi K, Saito I, Saito N. Acute to subacute surgical revascularisation for progressing stroke in atherosclerotic vertebrobasilar occlusion. Acta Neurochir 2012;154:1455–61.
50. Britze GW, Agarwal V, Mihlon F, Ramanathan D, Agrawal A, Nimjee SM, et al. Radial artery bypass for intractable vertebrobasilar insufficiency: case series and review of the literature. J World Neurosurg 2015;85:106–13.
51. Cai X, Wei Y, Ren S, Wu Z, Peng X, Huang Y, et al. Balloon-expendable stent angioplasty in the treatment of vertebral artery stenosis in the V2 segment. Videosurg Other Minimal Invasive Tech 2018;13:227–32.
52. Kerry R, Taylor AJ. Cervical arterial dysfunction: knowledge and reasoning for manual physical therapists. JOSPT 2009;39:378–87.
53. Savitz SI, Caplan LR. Vertebrobasilar disease. N Engl J Med 2005;352:2618–26.
54. Lima Neto AC, Bittar R, Gattas GS, Bor-Seng-Shu E, Oliveir ML, Monsanto RDC, et al. Pathophysiology and diagnosis of vertebrobasilar insufficiency: a review of the literature. Int Arch Otorhinolaryngol 2017;21:302–7.