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Original Article

Work-up and Management of Asymptomatic Extracranial Traumatic Vertebral Artery Injury

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ABSTRACT: ***Background:*** Non-penetrating head and neck trauma is associated with extracranial traumatic vertebral artery injury (eTVAI) in approximately 1–2% of cases. Most patients are initially asymptomatic but have an increased risk for delayed stroke and mortality. Limited evidence is available to guide the management of asymptomatic eTVAI. As such, we sought to investigate national practice patterns regarding screening, treatment, and follow-up domains. ***Methods:*** A cross-sectional, electronic survey was distributed to members of the Canadian Neurosurgical Society and Canadian Spine Society. We presented two cases of asymptomatic eTVAI, stratified by injury mechanism, fracture type, and angiographic findings. Screening questions were answered prior to presentation of angiographic findings. Survey responses were analyzed using descriptive statistics. ***Results:*** One hundred-eight of 232 (46%) participants, representing 20 academic institutions, completed the survey. Case 1: 78% of respondents would screen for eTVAI with computed topography angiography (CTA) (97%), immediately (88%). The majority of respondents (97%) would treat with aspirin (89%) for 3–6 months (46%). Respondents would follow up clinically (89%) or radiographically (75%), every 1–3 months. Case 2: 73% of respondents would screen with CTA (96%), immediately (88%). Most respondents (94%) would treat with aspirin (50%) for 3–6 months (35%). Thirty-six percent of respondents would utilize endovascular therapy. Respondents would follow up clinically (97%) or radiographically (89%), every 1–3 months. ***Conclusion:*** This survey of Canadian practice patterns highlights consistency in the approach to screening, treatment, and follow-up of asymptomatic eTVAI. These findings are relevant to neurosurgeons, spinal surgeons, stroke neurologists, and neuro-interventionalists.

RÉSUMÉ : Bilan et prise en charge des lésions traumatiques extra-crâniennes asymptomatiques de l'artère vertébrale ***Contexte :*** Les traumatismes non pénétrants de la tête et du cou sont associés à des lésions traumatiques extra-crâniennes de l'artère vertébrale dans environ 1 à 2 % des cas. La plupart des patients sont initialement asymptomatiques mais présentent un risque accru d'AVC tardif et de mortalité. À cet égard, on dispose de peu de données pour guider la prise en charge de ces lésions traumatiques asymptomatique. Nous avons donc cherché à étudier les pratiques nationales en matière de dépistage, de traitement et de suivi de ces lésions. ***Méthodes :*** Dans un premier temps, une enquête électronique transversale a été distribuée aux membres de la Société canadienne de neurochirurgie et de la Société canadienne du rachis. Nous avons à cette occasion présenté deux cas asymptomatiques de lésions traumatiques extra-crâniennes de l'artère vertébrale. Ces deux cas ont été en retour stratifiés en fonction du mécanisme de lésion, du type de fracture et des résultats obtenus à des examens d'angiographie. À noter que les participants ont répondu aux questions de dépistage avant que les résultats des examens d'angiographie leur soient présentés. Enfin, les réponses au questionnaire ont été analysées à l'aide de statistiques descriptives. ***Résultats :*** Au total, 108 participants sur 232 issus de 20 établissements universitaires ont répondu à l'enquête, ce qui représente un taux de participation de 46 %. Pour le premier cas, 78 % des participants ont affirmé qu'ils auraient tenté de dépister des lésions traumatiques extra-crâniennes de l'artère vertébrale, la majorité d'entre eux au moyen d'un examen d'angiographie par tomodensitométrie (97 %). Ajoutons de surcroît que 88 % d'entre eux l'auraient fait sur le champ. La majorité des participants (97 %) ont aussi indiqué qu'ils auraient traité un tel cas avec de l'aspirine (89 %), et ce, pendant 3 à 6 mois (46 %). Enfin, ils auraient effectué un suivi clinique (89 %) ou radiographique (75%) à tous les 1 à 3 mois. Pour le deuxième cas, 73 % des participants auraient tenté de dépister des lésions traumatiques extra-crâniennes de l'artère vertébrale, la majorité au moyen d'un examen d'angiographie par tomodensitométrie (96 %). Ajoutons que 88 % d'entre eux l'auraient fait sur le champ. La plupart des participants (94 %) auraient traité un tel cas avec de l'aspirine (50 %), et ce, pendant 3 à 6 mois (35 %). De plus, 36 % des participants auraient eu recours à un traitement endovasculaire. Ils auraient également effectué un suivi clinique (97 %) ou radiographique (89 %) à tous les 1 à 3 mois.

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Conclusion : Cette enquête sur les modèles canadiens de pratique met en évidence la cohérence des pratiques de dépistage, de traitement et de suivi des lésions traumatiques extra-crâniennes asymptomatiques de l'artère vertébrale. Ces résultats sont donc pertinents pour les neurochirurgiens, les chirurgiens de la colonne vertébrale, les neurologues spécialisés dans les AVC et les neuro-interventionnistes.

Keywords: Neurovascular; Spinal Trauma; Trauma; Vascular; Spine; Neurotrauma; Neurosurgery; Neurosurgery - vascular; Neuroimaging; Interventional neuroradiology

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Introduction

Extracranial traumatic vertebral artery injury (eTVAI) occurs in approximately 1–2% of non-penetrating head and neck traumas.[1–4] Most patients are initially asymptomatic but have an increased risk for delayed stroke and mortality.[4–7] Guidelines for eTVAI are outdated and supported predominantly by Level 3 evidence.[8] Furthermore, limited evidence is available to guide the management of *asymptomatic* patients with eTVAI.[8]

Screening for eTVAI using computed topography angiography (CTA) may be considered in cases of blunt trauma; guidelines for the management of eTVAI recommend the use of screening criteria.[8] The modified Denver and Memphis criteria are screening criteria for blunt cerebrovascular injury (BCVI) in patients with high-risk features such as expanding cervical hematoma, cardiothoracic injuries, focal neurological deficits, concomitant traumatic brain injury, and radiographic findings such as fractures or ischemia (Table 1).[9–12] Despite evidence supporting their utility, these criteria may be underutilized, particularly in asymptomatic patients.[4,9,10,13,14]

Regarding treatment and follow-up, the Cervical Artery Dissection in Stroke Study (CADISS) may guide medical management of patients with symptomatic cerebrovascular dissection; however, the trial included spontaneous vertebral artery injuries.[15] It did not include asymptomatic patients or elaborate on optimal dosages, treatment duration, or choice of antiplatelet or anticoagulation therapy. Guidelines for eTVAI recommend that choice of therapy should be individualized based on the vertebral artery injury, other associated injuries, and potential risk of bleeding.[8] Digital subtraction angiography (DSA) may be considered for the diagnosis of eTVAI in select patients; however, the role of endovascular therapy in eTVAI remains undefined.[8,16–18] Guidelines for eTVAI do not provide a recommendation regarding the use of endovascular therapy as an adjunct to antithrombotic therapy in adult patients to reduce the risk of stroke.[8]

Given the limited evidence available to guide the management of *asymptomatic* eTVAI, the purpose of this study was to investigate Canadian practice patterns reflecting screening, treatment, and follow-up domains. Such findings would be broadly relevant to neurosurgeons, spinal surgeons, stroke neurologists, neurointerventionalists, and trauma specialists managing the work-up and/or treatment of eTVAI. They may also be used to inform a shared decision-making approach with patients and their families.

Methods

Study Design and Target Population

The Canadian Neurosurgery Research Collaborative (CNRC) is a group of resident neurosurgeons seeking to advance the care of

Table 1: Criterion-based screening tools for blunt cerebrovascular injury

Modified Denver criteria[a]	Modified Memphis criteria[b]
Signs or symptoms of BCVI	
• Arterial hemorrhage from neck/mouth/nose	• Basilar skull fracture with involvement of the carotid canal
• Cervical bruit in patient <50 years old	• Basilar skull fracture with involvement of the petrous bone
• Expanding cervical hematoma	• Any cervical spine fracture
• Lateralizing neurological deficit (e.g., TIA, hemiparesis, vertebrobasilar symptoms, and Horner's syndrome)	• Neurological examination not explained by brain imaging
• Neurological deficit incongruous with head CT scan finding	• Horner's syndrome
• Stroke on CT or MRI, head scan	• LeFort II or III fracture pattern
Risk factors for BCVI	• Neck soft tissue injury (seatbelt sign or hanging or hematoma)
High energy transfer mechanism associated with:	• CTA (as a screening criterion)[c]
• Severe facial fracture (LeForte II or III)	
• Mandible fracture	
• Complex skull fracture/basilar skull fracture/occipital condyle fracture	
• GCS <6 with diffuse axonal injury	
• Cervical spine fracture (evidence of subluxation, extension into the transverse foramen, fractures of any part of C1-3, and any body fracture)	
• Near hanging with anoxic brain injury	
• Clothesline type injury or seat belt abrasion with significant swelling, pain, or altered mental status	
• TBI with concomitant thoracic injuries	
• Scalp degloving	
• Thoracic vascular injuries	
• Blunt cardiac rupture	

BCVI = blunt cerebrovascular injury; CT/CTA = computed tomography angiography; GCS = Glasgow Coma Scale; MRI = magnetic resonance imaging; TIA = transient ischemic attack.
[a]Cothren Burlew et al. *J Trauma Acute Care Surg*, **2012**, *72*, 330
[b]Ciapetti et al. *Scand J Trauma Resusc Emerg Med.* **2010**, *18*, 61
[c]Emmett et al. *J Trauma*, 2011, 70, 1058.

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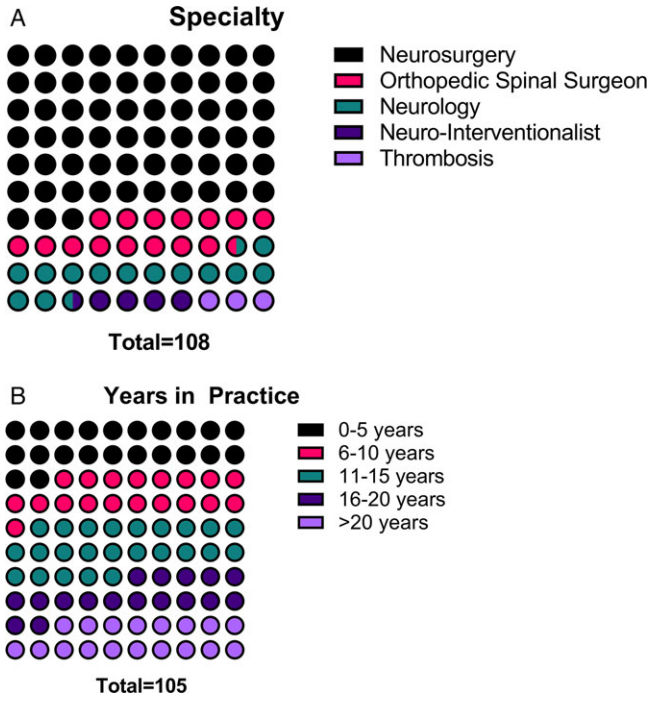

Figure 1: Demographic variables: (a) specialty and (b) years in practice.

patients through collaborative research. Our group conducted a self-administered, cross-sectional survey of Canadian physicians who screen, treat, and/or follow patients with eTVAI. Neurosurgeons, fellowship-trained spinal surgeons, neurologists, and neuro-interventionalists were eligible for study inclusion. We excluded respondents who self-reported they do not work-up or manage patients with eTVAI. This study adheres to the Checklist for Reporting of Survey Studies (CROSS) guidelines (Supplemental Material A).[19]

Survey Development and Testing

A narrative literature review was performed in order to identify eTVAI-related management domains lacking scientific support. MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials were search from inception until September 2020. Free-text words relevant to presentation (asymptomatic and symptomatic), location (extracranial, neck, and cervical), mechanism of injury (non-penetrating, blunt, and traumatic), and vascular injury (vertebral artery and dissection) were included. Abstracts were reviewed by two investigators (MAM and CJT). Management domains lacking scientific support were identified and informed development of a draft 32-question survey (Supplemental Material B). The survey did not involve patients, and case descriptions did not include actual patient data. The survey draft was provided to the CNRC steering committee and was assessed in order to optimize redundancy and improve clarity. Assessment of face and content validity was conducted by fellowship-trained spinal neurosurgeons and stroke neurologists at the coordinating site.

Survey Design

Demographics (subspecialty and years in practice) were recorded. We assessed respondents perceived value of clinical and radiographic symptoms and signs in screening for eTVAI. We inquired about use of criterion-based screening tools (e.g., modified Denver or Memphis criteria).[9,12] Stroke and transient ischemic attack (TIA)-related symptoms were considered separate, as most respondents listed both exclusively. Two case-based scenarios were presented, involving asymptomatic patients with eTVAI, no additional injuries, and conventional arterial anatomy. Response options included "none" and "other" (specified by free text). To avoid ambiguity regarding interpretation of "asymptomatic," we used the term "neurologically intact."

Case 1 was described as a 35-year-old patient with a fall from standing height. Unenhanced CT demonstrated a cervical lateral mass fracture extending into the vertebral foramen. Respondents were asked if they would screen for eTVAI, and if so, their modality of choice. Subsequently, respondents were asked to assume that CTA was obtained and demonstrated nonprogressive eTVAI. Treatment and follow-up questions were then stratified according to: (a) <25% lumen diameter reduction without intimal flap and (b) >25% luminal diameter reduction with raised intimal flap. These cutoffs are in keeping with the Denver Grading Scale for BCVI, including eTVAI.[4,8,20,21]

Case 2 described a 55-year-old patient in a high-speed motor vehicle collision. Unenhanced CT demonstrated an atypical hangman's fracture (unilateral oblique C2 body and contralateral pars fracture) (Supplemental Material B). Respondents were asked if they would screen for eTVAI, and if so, their modality of choice. Respondents were subsequently asked to assume that CTA imaging was obtained and positive for pseudoaneurysm dissection eTVAI.

Survey Administration

Anonymous and voluntary survey responses were collected electronically using Survey Monkey (San Mateo, CA, USA). Participation in the survey was considered as consent for enrollment in the study. No financial incentive was provided. In order to ensure our sample was representative of the study population, we included 150 members of the Canadian Neurosurgical Society (CNSS). We also included 83 orthopedic spine surgeon members of the Canadian Spine Society (CSS). Stroke neurologists and neuro-interventionalists ($n = 64$) who investigate and/or manage asymptomatic eTVAI were approached by residents distributing the survey at individual sites. A follow-up reminder was sent at 5 weeks. If respondents answered more than 80% of the survey, it was considered complete. Respondent demographic data were used to ensure single survey participation.

Statistical Analysis

Responses were analyzed using descriptive statistics. Categorical data were reported as counts and percentages. Results are featured as proportions with 95% confidence intervals.

Results

Thirty-six percent (108 of 297) of clinicians responded, representing 20 academic institutions. The response rate for each question exceeded 80%. Sixty-three percent of respondents were neurosurgeons, 17% stroke neurologists, 16% orthopedic spinal surgeons, and 5% neuro-interventionalists (Figure 1a). Years in practice were evenly distributed (Figure 1b).

Self-Confidence and Evidence-Based Supporting Decision-Making

Fifty-six percent of respondents agree or strongly agree they are confident in their ability to manage asymptomatic eTVAI, whereas 30% were neutral (Figure 2a). Respondents were asked if their

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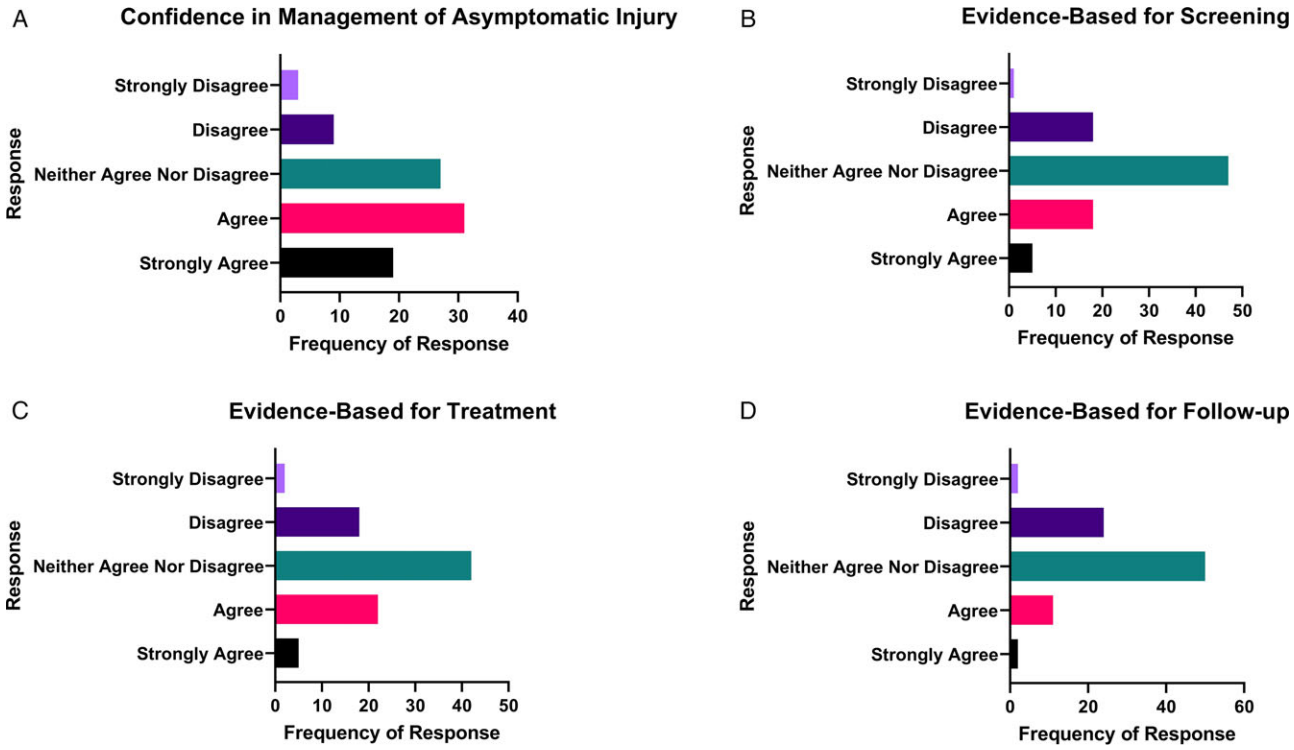

Figure 2: (a) Self-confidence and (b) evidence-based supporting decision-making in eTVAI.

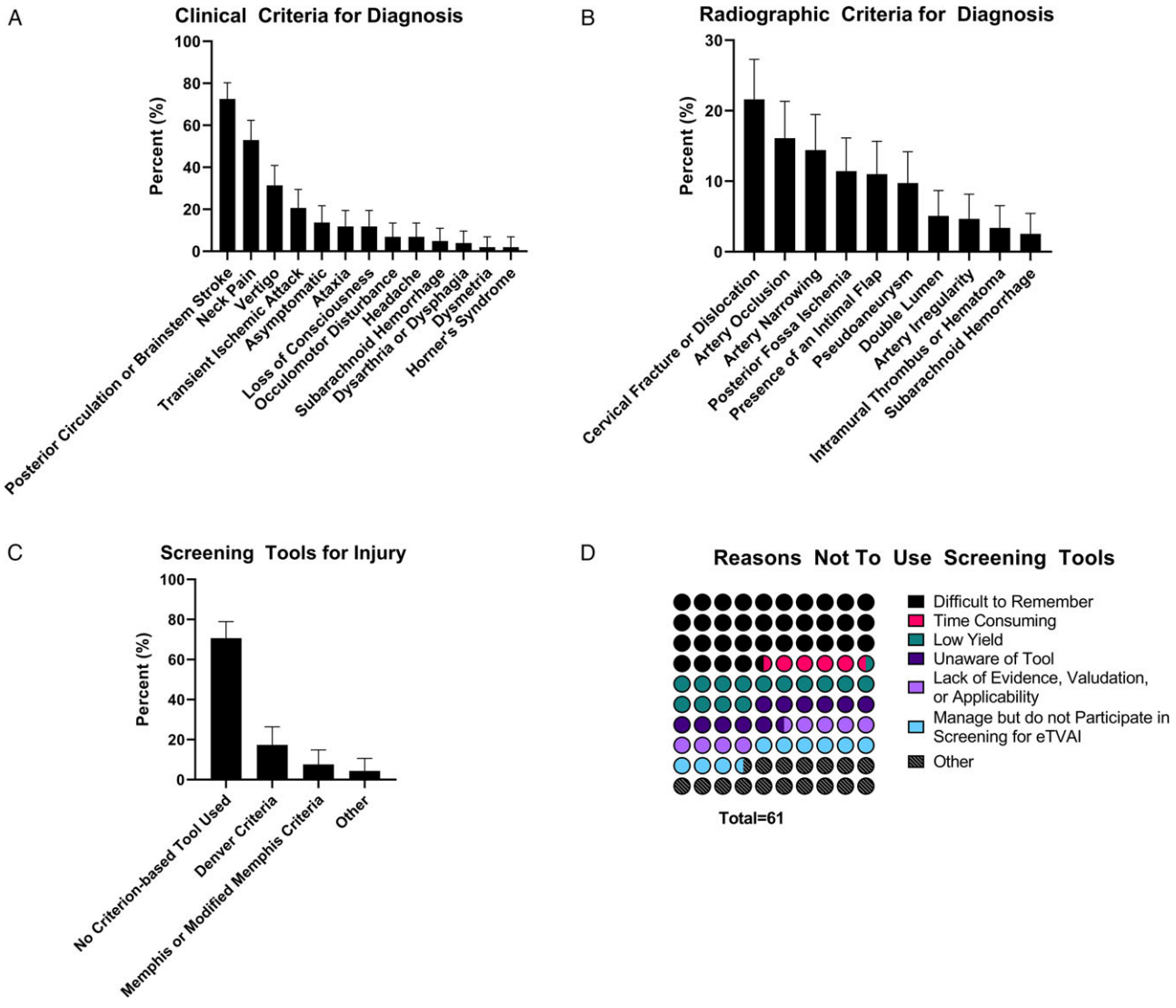

Figure 3: (a) Clinical and (b) radiological signs ranked by respondents according to perceived association with eTVAI; (c) criterion-based screening tools utilized in eTVAI; and (d) reasons for not using screening tools in eTVAI.

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Table 2: Respondents top perceived clinical and radiographic signs of eTVAI

Clinical signs of eTVAI	%	Radiographic signs of eTVAI	*n* (%)
"Posterior circulation or brainstem stroke-related symptoms"	76	Cervical fracture or dislocation	40
Neck pain	56	Vessel luminal narrowing	36
Vertigo or dizziness	24	Vessel occlusion	36
Asymptomatic (no symptoms)	14	Posterior fossa infarction (CT or MRI)	26
Altered level of consciousness	13	Vessel dissection (or double lumen)	24
"Transient ischemic attack-related symptoms"	12	"Pseudoaneurysm"	21
Headache	12	Vessel intimal flap	20
Ataxia	11	Mural thrombus or vessel wall hematoma	17
Traumatic mechanism	9	Abnormality on CT/CTA (NOS)	16
Horner's syndrome	7	Abnormality on MRI/MRA (NOS)	9
Oculomotor dysfunction	6	Subarachnoid hemorrhage	6
Visual disturbance NOS	6	Vessel wall irregularity NOS	6
Nausea/emesis	6	Vessel filling defect	6
Cerebellar signs NOS	5	Contrast extravasation	4
Dysarthria	4	Abnormality on DSA (NOS)	3
Cervical ecchymosis or hematoma	3	"Aneurysm"	2
Tinnitus	1	Distal embolism	1
Cervical bruit	1	"Arterial blush"	1
"Signs of basal skull fracture"	1	"Rat tail sign"	1
Radiculopathy/myelopathy	1	"No finding"	1

CTA = computed tomography angiography; DSA = digital subtraction angiography; MRA = magnetic resonance imaging angiography; NOS = not otherwise specified.

decision-making regarding screening, treatment, and follow-up is evidence-based, as opposed to expert opinion (Figure 2b-d; Supplemental Material C). The majority of respondents neither agree nor disagree regarding screening (53%), treatment (47%), and follow-up (56%).

Clinical and radiological symptoms associated with eTVAI

Respondents reported their perceived top three clinical signs of eTVAI (Figure 3a; Table 2). The three most frequent responses were "posterior circulation/brainstem stroke-related symptoms" (76%), "neck pain" (56%), and vertigo or dizziness (24%).

Respondents reported their perceived top three radiographic signs of eTVAI (Figure 3b; Table 2). The three most frequent responses were "cervical fracture or dislocation" (40%), "vessel luminal narrowing" (36%), and "vessel occlusions" (36%).

Screening criteria

Seventy-one percent of respondents do not use a criterion-based tool to diagnose eTVAI (Figure 3c). Modified Denver and Memphis criteria were used by 17 and 8%, respectively. The three most frequent reasons for not using a criterion-based screening tool included "difficult to remember" (34%), "low yield" (15%), and "not aware of a criterion-based screening tool" (12%) (Figure 3d; Supplemental Material C).

Scenario-Based Questions:

Table 3 presents a summary of respondent's screening, treatment, and follow-up practices for both cases of eTVAI. Expanded data are available in Supplemental Material C.

For Case 1 (i.e., low-energy injury mechanism and uncomplicated fracture pattern), the majority of respondents would screen with CTA (Figure 4a), immediately (Figure 4b). Regardless of luminal diameter reduction of presence of intimal flap, most respondents would start treatment with ASA (Figures 5a and b), continue medical therapy for 3–6 months (Figure 5c), follow-up clinically every 1–3 months (Figures 6a and b), and follow-up radiographically every 1–3 or 3–6 months (Figures 6a and b). The overall duration of clinical and radiographic follow-up varied from 3–6 months to 1–3 years (Figure 7a). A minority of respondents would follow radiologically "until complete regression."

For Case 2 (i.e., high-energy injury mechanism and complicated fracture pattern), the majority of respondents would screen with CTA (Figure 4a), immediately (Figure 4b). Most respondents would treat eTVAI-related pseudoaneurysm dissection with ASA or endovascular surgery (Figure 5e); the duration for medical therapy varied, but 3–6 months was most frequently reported (Figure 5f). Most respondents would follow up clinically every 1–4 weeks or 1–3 months, and radiologically every 1–3 or 3–6 months (Figure 6c). The overall duration of clinical and radiological follow-up ranged from 3–6 months to 1–3 years (Figure 7b). A minority of respondents would follow radiologically "until complete regression."

Discussion

Unrecognized eTVAI is associated with high rates of stroke and mortality.[1,4,21] Comprehensive screening and timely initiation of treatment is associated with increased detection of BCVI and decreased risk of stroke and mortality.[14] Current recommendations for the work-up and management of eTVAI pertain primarily to symptomatic cases.[8] Given the paucity of evidence pertaining to *asymptomatic* eTVAI, the CNRC investigated national practice patterns across 20 academic institutions. We presented two clinical scenarios of blunt trauma resulting in asymptomatic eTVAI, stratified based on trauma mechanism, fracture complexity, and degree of vessel injury. In both cases, the majority of respondents opted to screen for eTVAI with CTA, initiate aspirin therapy for 3–6 months, and follow-up clinically and radiographically within 1–3 months, respectively (Table 3). These findings are relevant to neurosurgeons, spinal surgeons, stroke neurologists, and neurointerventionalists caring for patients with eTVAI. Furthermore, they may inform a shared decision-making approach with patients and their families. A summary of potential management consideration for the screening, treatment, and follow-up of asymptomatic eTVAI is highlighted in Table 4.

Clinical and Radiographic Findings in eTVAI

Patients with eTVAI are often asymptomatic.[4,20] Immediate symptoms may be explained by the high rate of associated injuries (93% of patients).[21] Cervical fractures are prevalent (70–75% of

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Table 3: Clinical case details* and respondents preferred screening, management, and follow-up

	Case 1a	Case 1b	Case 2
Age	35 years	35 years	55 years
Gender	Male	Male	Female
Injury mechanism	Fall from standing height	Fall from standing height	High-speed MVC
Examination	Neurologically intact	Neurologically intact	Neurologically intact
Unenhanced CT description	Cervical lateral mass fracture extending into foramen transversarium	Cervical lateral mass fracture extending into foramen transversarium	Atypical Hangman's fracture (unilateral oblique C2 body/ contralateral pars)
eTVAI CTA description	<25% lumen reduction	>25% lumen reduction with raised intimal flap	Pseudoaneurysm dissection
Summary of respondents choice screening, treatment, and follow-up**			
Screening modality			
CTA	71 (97%)		63 (96%)
MRA	2 (3%)		1 (2%)
DSA	0 (0%)		0 (0%)
Screening timing			
Immediately	68 (88%)		60 (88%)
Other	10 (12%)		8 (12%)
Initiate treatment			
ASA	86 (89%)	78 (76%)	54 (50%)
Clopidogrel	3 (3%)	7 (7%)	6 (6%)
Other	8 (8%)	14 (13%)	19 (18%)
Endovascular surgery	0%	4 (4%)	36 (33%)
Treatment duration			
1–3 months	31 (36%)		17 (28%)
3–6 months	40 (46%)		21 (35%)
Other	16 (18%)		22 (37%)
Clinical F/U			
1–4 weeks	14 (17%)	20 (24%)	21 (25%)
1–3 months	35 (43%)	31 (37%)	32 (38%)
3–6 months	16 (20%)	16 (19%)	12 (14%)
Other	17 (20%)	16 (20%)	19 (23%)
Radiological F/U			
1–4 weeks	7 (10%)	14 (18%)	14 (18%)
1–3 months	27 (39%)	27 (35%)	27 (36%)
3–6 months	27 (39%)	24 (32%)	19 (25%)
Other	8 (12%)	11 (15%)	16 (21%)
Overall F/U			
3–6 months	34 (37%)		25 (29%)
7–12 months	14 (15%)		14 (16%)
1–3 years	11 (12%)		19 (22%)
"Until complete regression"	12 (13%)		12 (14%)
Other	22 (27%) %		16 (19%)

CTA = computed tomography angiography; F/U = follow-up; MVC = motor vehicle collision.
*For complete response data, including expanded "other" categories, please refer to tables found in Supplemental material C.
**(*n*, %)

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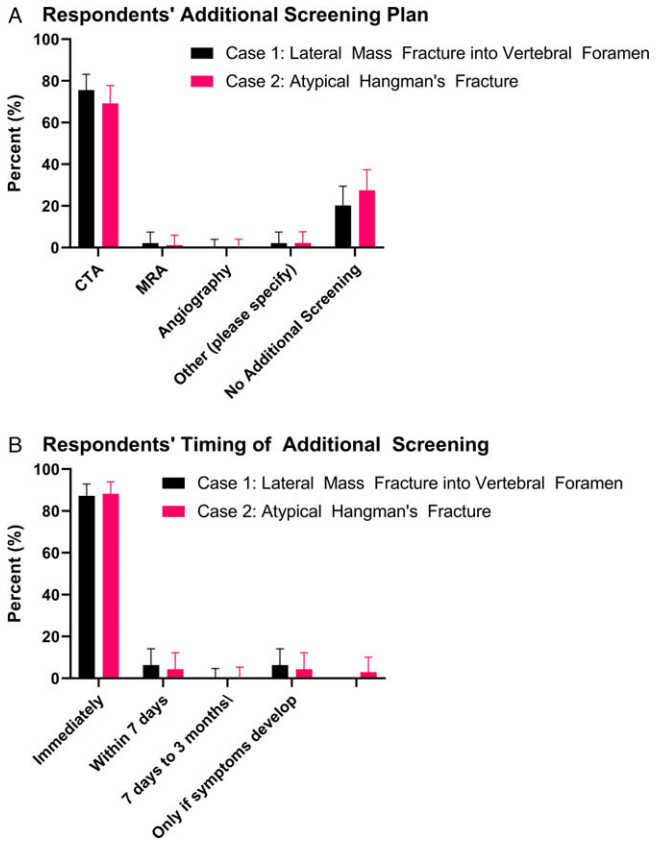

Figure 4: Respondents (a) screening plan and (b) timing of additional screening for Cases 1 and 2.

patients).[1,4,20] Delayed symptoms typically develop within 10–72 hours[10,20,22,23] and may be explained by infarction secondary to thromboembolism, or vessel luminal narrowing that progresses to occlusion.[20] Vessel occlusion may not manifest symptoms in patients with adequate collateral circulation.[21] Symptomatic patients typically present with headache, neck pain, and neurological deficits related to the posterior circulation territory infarcted (e.g., long-tract signs, gait disturbance, vertigo, and Horner's syndrome).[21,24] The most common vertebral artery findings on radiographic imaging are luminal narrowing, dilatation, occlusion, intimal flap, and pseudoaneurysm.[25] Herein, the most frequently reported clinical and radiographic symptoms and signs of eTVAI considered by respondents were similar to those most frequently reported in the literature (Table 2).[9,14,20,21,26]

Screening for eTVAI

Screening protocols for eTVAI were implemented in the 1990s upon recognition of an early asymptomatic period and specific patterns of associated injuries.[27] Prior to this, stroke and mortality rates were 80 and 40%, respectively.[9,28,29] Despite an increase in the identification of BCVI, including asymptomatic eTVAI,[26] in centers applying such criteria, 20–30% of cases remained unrecognized until patients progressed and became symptomatic.[26] The modified Denver[27] and Memphis criteria[26] were subsequently proposed (Table 1) and led to a reduction in missed injuries, allowing for earlier initiation of treatment and prevention of stroke and stroke-related mortality.[13,14] Multiple studies have since highlighted the utility of comprehensive screening protocols, including the use of expanded screening criteria;[1,14,23,26,27] stroke in asymptomatic patients can be almost universally avoided with early detection and appropriate treatment.[27]

Despite the aforementioned evidence supporting the use of screening criteria, including recommendation in eTVAI practice guidelines,[8] the majority of respondents in this study indicated they do not use a criterion-based screening tool (Figure 3c). Reasons for not using a tool included "difficulty remembering," "low yield," "time-consuming," or "lack of evidence or applicability." The majority of respondents in this study were neurosurgeons. Use of criterion-based screening tools may vary with the specialty of the physician performing the trauma assessment. Trauma leaders responsible for decision-making may have background training in general surgery, anesthesiology, emergency medicine, or neurosurgery. Our study draws attention to the apparent need for more Canadian physicians managing eTVAI to recognize the value of screening criteria and employ them in routine practice. Targeted education around the utility of these criteria, awareness of guideline recommendations, and use of diagnostic algorithms could potentially facilitate increased awareness and use. Level I evidence pertaining to eTVAI is limited to screening modality;[8] in patients who meet the Modified Denver Screening Criteria for BCVI, CTA is recommended.[9,12,27] As such, it is not surprising that the majority of respondents herein would screen for eTVAI (Case 1, 78%; Case 2, 73%) with CTA (Case 1 and 2, 96%, respectively) (Table 3). Thin-slice, high-resolution CTA provides similar sensitivity and specificity compared to DSA for diagnosing eTVAI; it also is associated with fewer complications.[30] CTA is useful for detection of commonly associated vertebral fractures, subluxation, and facet dislocations.[5] Most respondents would not screen with magnetic resonance imaging (MRI)/MRA. This may be explained by the fact that CTA offers reduced duration of scanning and improved detection of arterial injury compared to MRI/MRA.[31] However, MRI/MRA may be warranted in cases of suspected spinal cord injury, vertebral ligamentous injury, or posterior circulation ischemia. Adjunct use of DSA is largely reserved for indeterminate cases and can assess collateral circulation or facilitate endovascular repair.

Treatment and Outcomes

Treatment of eTVAI is associated with a reduction in stroke and mortality.[4,21,23,28,32] The risk reduction of anticoagulation or antiplatelet therapy in both symptomatic and asymptomatic eTVAI remain unknown.[21] Reported treatment effects pertaining to stroke rates vary across studies. Guideline recommendations for the treatment of eTVAI include consideration of antiplatelet or anticoagulation (versus no therapy) based on characteristics of the vertebral artery injury, associated injuries, and risk of bleeding.[8] Particularly in the setting of concomitant traumatic brain injury or solid organ injuries, multidisciplinary discussion may guide determination of safe timing for the initiation of antithrombotic therapy. Cothren et al. reported equivalent stroke risk and vessel healing rates for both antiplatelet and anticoagulation therapies.[23] The CADISS study demonstrated anticoagulant therapy is not superior to antiplatelet therapy for preventing stroke or death, in symptomatic patients with cervical (carotid or vertebral) artery dissection.[4] However, the CADISS study included mostly spontaneous dissections and was likely underpowered to detect a difference in outcome.[33]

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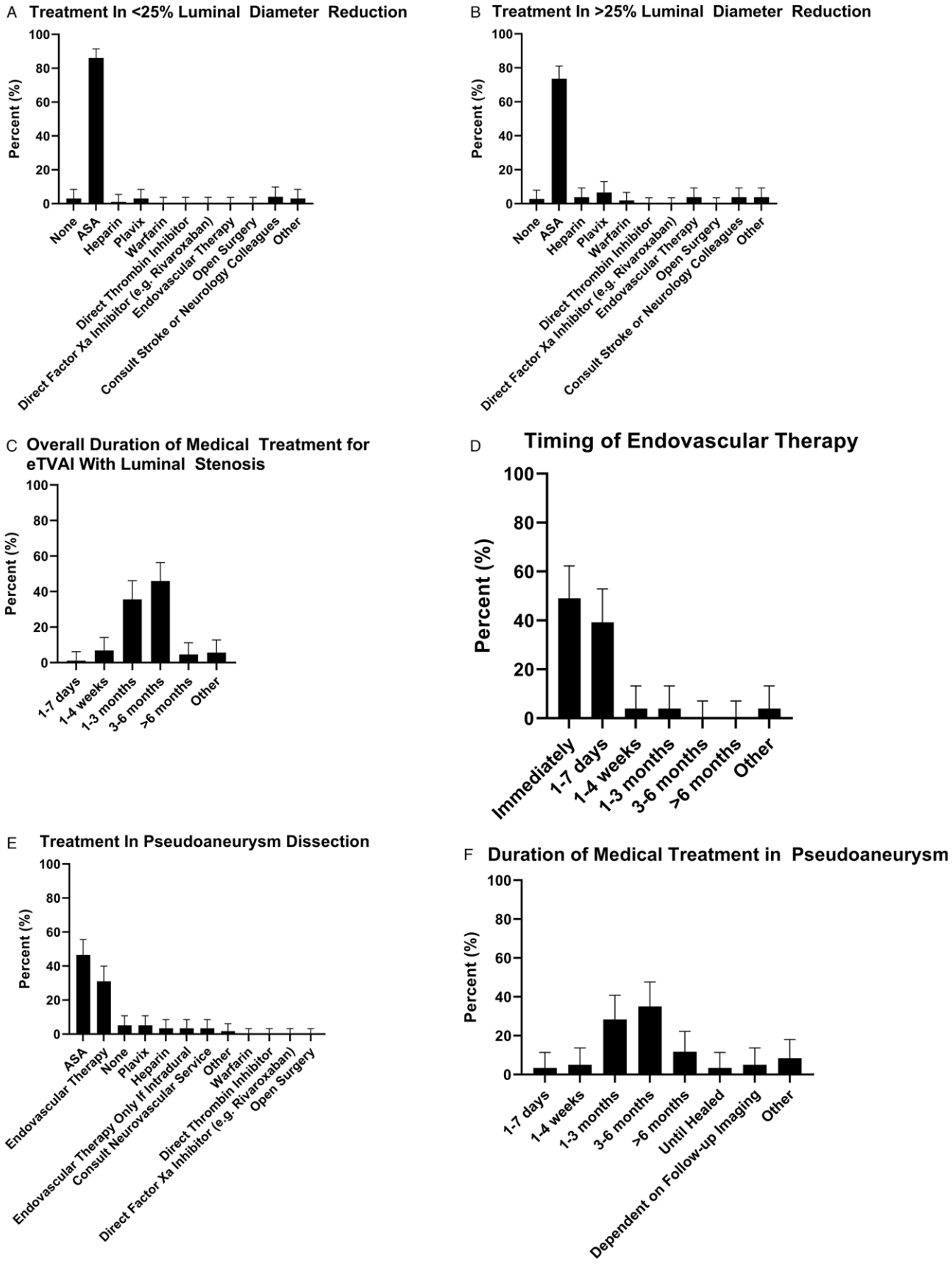

Figure 5: Case 1: (a) treatment of choice for eTVAI with <25% luminal diameter reduction and (b) >25% luminal diameter reduction; (c) overall duration of medical treatment. Case 2: (a) timing of endovascular therapy (if applicable); (b) treatment of choice for eTVAI with pseudoaneurysm dissection; and (c) duration of medical treatment in eTVAI with pseudoaneurysm dissection.

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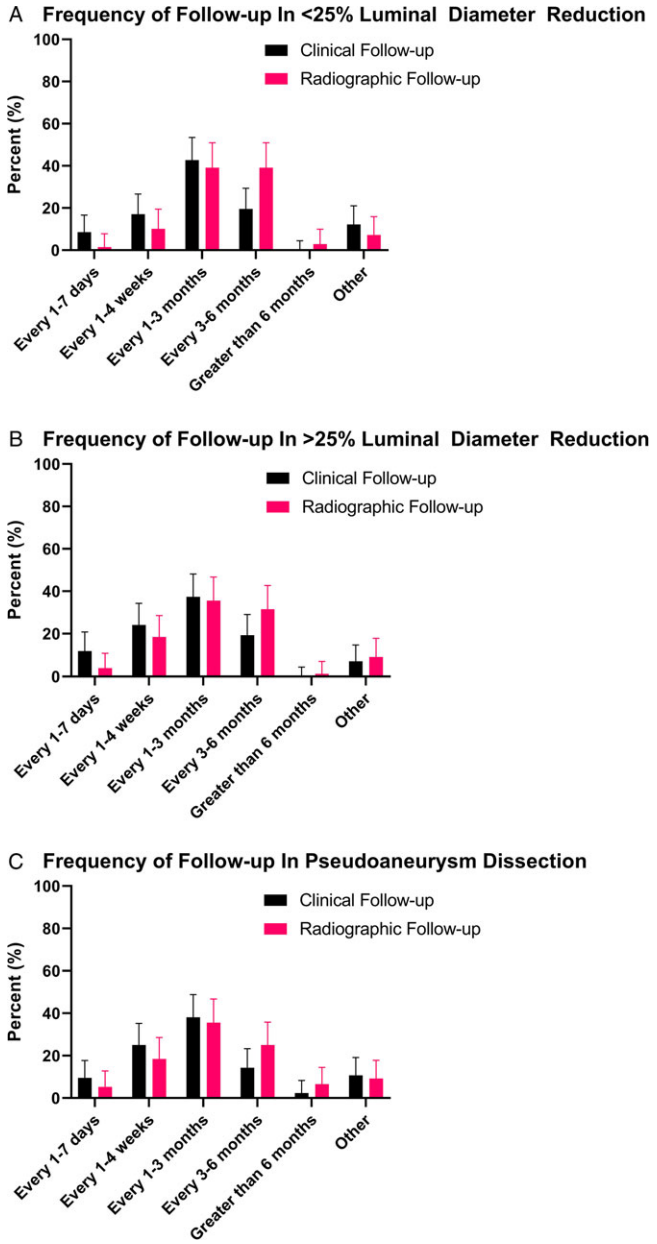

Figure 6: Frequency of follow-up for eTVAI with: (a) <25% luminal diameter reduction; (b) >25% luminal diameter reduction; and (c) pseudoaneurysm dissection.

Respondents in our study demonstrated a preference for antiplatelet therapy in both cases. A recent Canadian retrospective review of the British Columbia Trauma Registry found similar findings. They identified 186 patients with BCVI and 88.9% were treated with ASA monotherapy.[34] Few patients in their study received a loading dose of ASA, or dual antiplatelet therapy. Although evidence supports these practices for nontraumatic stroke,[35,36] their role remains unclear for stroke prevention in BCVI.

Endovascular Therapies

The utility of endovascular treatment of eTVAI remains relatively undefined compared to spontaneous vertebral artery dissections; recommendations regarding stenting, occlusion, and pseudoaneurysm coil embolization are based on low-quality evidence.[1]

Table 4: Potential management considerations for the screening, treatment, and follow-up of asymptomatic eTVAI

	Management considerations
Screening	To identify patients at risk of eTVAI, consider use of established screening criteria (e.g., modified Denver or Memphis screening criteria) and consider performing CT angiography.
Acute management	To reduce the risk of eTVAI associated stroke, consider treatment with antithrombotic therapy. Consider the risk of bleeding in the context of associated injuries. Treatment may be initiated as early as safely possible.
Follow-up	To monitor for progression or resolution, consider clinical and radiographic follow-up for 1–3 months.

CT = computed tomography; eTVAI = extracranial traumatic vertebral artery injury.

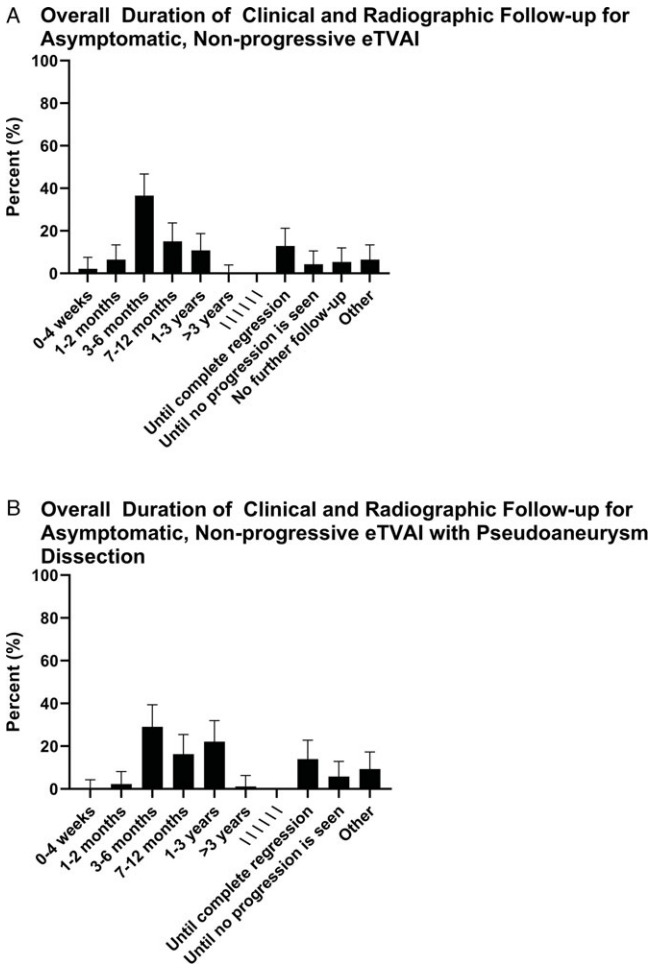

Figure 7: Overall duration of follow-up for eTVAI in (a) Case 1 and (b) Case 2.

A systematic review and meta-analysis found insufficient data for the assessment of efficacy of thrombolysis or stenting in the treatment of symptomatic TVAI.[2] However, complication rates in retrospective and non-randomized studies appear similar to those reported for thrombolysis in ischemic stroke or carotid artery stenting in cases of stenosis related to atherosclerosis.[2,7] Choice of technique may depend on eTVAI grade, injury site, and collateral circulation.[21] Few of our respondents would employ endovascular therapy in the absence of pseudoaneurysm dissection. The

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obligatory use of dual antiplatelet therapy following stenting represents a potential deterrent in trauma patients with risk of hemorrhage,[1] or concomitant traumatic brain injury (41% in one series).[4]

Follow-up for TVAI

Limited evidence is available to guide clinicians with regard to clinical and radiographic follow-up of eTVAI; routine follow-up may be recommended.[21,37] Given the potential for initial false-positive interpretations, to examine for vessel healing which has been shown to vary by injury grade,[10] low-quality evidence supports follow-up CTA at 7 days and 3 months following TVAI.[37] In keeping with published guidelines,[37] most respondents in our study would follow up clinically and radiographically in 1–3 months. Respondents emphasized a lack of scientific evidence in this domain. What Canadian clinicians do in actual practice may differ, as a recent study of Canadian patients with BCVI found only 35.7% received repeat imaging within 7 days.[34] The authors highlight a potential benefit to consistent follow-up imaging, including an influence on the duration of antithrombotic therapy, with reduction of late bleeding complications associated with prolonged therapy.[10] Reasons for suboptimal repeat imaging rates included a lack of appropriate documentation pertaining to their BCVI treatment algorithm in a third of discharge summaries, as well as potential difficulty in accessing neuroimaging and outpatient services in remote communities. They propose a routine pathway for arranging clinical follow-up and radiographic imaging, including clear communication with general practitioners responsible for follow-up care of patients with trauma.

Limitations

Limitations of this study relate to its survey design. However, we attained a high response rate and identified consensus across all survey domains. Furthermore, current management guidelines pertain to symptomatic eTVAI and are supported predominantly by Level 3 evidence. Responses obtained in this study pertain to eTVAI and are not generalizable to patients with spontaneous dissections. We did not attempt to define or compare stroke rates.

Future Work

Further research may seek to determine the optimal dosage, treatment period, choice of medical therapy, and treatment effect for asymptomatic patients with eTVAI. Prospective studies should further define subgroups of asymptomatic eTVAI at risk of progression and potentially require additional therapy. Additionally, the role of endovascular therapy should be clarified. National consensus guidelines for the management of eTVAI may be useful. Prospective validation of screening tools for eTVAI in the context of current trauma practices may facilitate increased early recognition of eTVAI to improve clinical outcomes.

Conclusion

We identified consistency in national practice patterns across 20 academic institutions for the screening, treatment, and follow-up of asymptomatic eTVAI. We presented two clinical scenarios featuring asymptomatic eTVAI, stratified based on trauma mechanism, fracture complexity, and degree of vessel injury. In both cases, the majority of respondents opted to screen for eTVAI with CTA, initiate antithrombotic therapy for 3–6 months, and follow-up clinically and radiographically within 1–3 months, respectively. The findings herein are limited by the survey design but may be useful to neurosurgeons, spinal surgeons, stroke neurologists, and neuro-interventionalists, facilitating a shared decision-making approach with patients and their families.

Supplementary Material. To view supplementary material for this article, please visit https://doi.org/10.1017/cjn.2022.292.

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Study Quality Guidelines. This study adheres to CROSS guidelines for reporting survey studies.

References

1. Stein DM, Boswell S, Sliker CW, Lui FY, Scalea TM. Blunt cerebrovascular injuries: does treatment always matter? J Trauma. 2009;66:132–43, discussion 143-4.
2. Weber CD, Lefering R, Kobbe P, et al. Blunt cerebrovascular artery injury and stroke in severely injured patients: an international multicenter analysis. World J Surg. 2018;42:2043–53.
3. Schicho A, Luerken L, Meier R, et al. Incidence of traumatic carotid and vertebral artery dissections: results of cervical vessel computed tomography angiogram as a mandatory scan component in severely injured patients. Ther Clin Risk Manag. 2018;14:173–8.
4. Biffl WL, Moore EE, Elliott JP, et al. The devastating potential of blunt vertebral arterial injuries. Ann Surg. 2000;231:672–81.
5. Rodriguez M, Tyberghien A, Matgé G. Asymptomatic vertebral artery injury after acute cervical spine trauma. Acta Neurochir (Wien). 2001;143:939–45.
6. Gurdjian ES, Hardy WG, Lindner DW, Thomas LM. Closed cervical cranial trauma associated with involvement of carotid and vertebral arteries. J Neurosurg. 1963;20:418–27.
7. Friedman D, Flanders A, Thomas C, Millar W. Vertebral artery injury after acute cervical spine trauma: rate of occurrence as detected by MR angiography and assessment of clinical consequences. AJR Am J Roentgenol. 1995;164:443–7; discussion 448-9.
8. Harrigan MR, Hadley MN, Dhall SS, et al. Management of vertebral artery injuries following non-penetrating cervical trauma. Neurosurgery. 2013;72 Suppl, 2:234–43.
9. Biffl WL, Moore EE, Offner PJ, et al. Optimizing screening for blunt cerebrovascular injuries. Am J Surg. 1999;178:517–22.
10. Biffl WL, Ray CE, Moore EE, et al. Treatment-related outcomes from blunt cerebrovascular injuries: importance of routine follow-up arteriography. Ann Surg. 2002;235:699–706, discussion 706-7.
11. AlBayar A, Sullivan PZ, Blue R, et al. Risk of vertebral artery injury and stroke following blunt and penetrating cervical spine trauma: a retrospective review of 729 patients. World Neurosurg. 2019;130:e672–9.
12. Cothren CC, Moore EE, Ray CE, Johnson JL, Moore JB, Burch JM. Cervical spine fracture patterns mandating screening to rule out blunt cerebrovascular injury. Surgery. 2007;141:76–82.
13. Ciapetti M, Circelli A, Zagli G, et al. Diagnosis of carotid arterial injury in major trauma using a modification of Memphis criteria. Scand J Trauma Resusc Emerg Med. 2010;18:61.
14. Geddes AE, Burlew CC, Wagenaar AE, et al. Expanded screening criteria for blunt cerebrovascular injury: a bigger impact than anticipated. Am J Surg. 2016;212:1167–74.

15. trial investigators CADISS, Markus HS, Hayter E, et al. Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial. Lancet Neurol. 2015;14:361–7.
16. Burlew CC, Biffl WL, Moore EE, et al. Endovascular stenting is rarely necessary for the management of blunt cerebrovascular injuries. J Am Coll Surg. 2014;218:1012–7.
17. Berne JD, Norwood SH. Blunt vertebral artery injuries in the era of computed tomographic angiographic screening: incidence and outcomes from 8,292 patients. J Trauma. 2009;67:1333–8.
18. Schneidereit NP, Simons R, Nicolaou S, et al. Utility of screening for blunt vascular neck injuries with computed tomographic angiography. J Trauma - Inj Infect Crit Care. 2006;60:209–15.
19. Sharma A, Minh Duc NT, Luu Lam Thang T, et al. A consensus-based checklist for reporting of survey studies (CROSS). J Gen Intern Med. 2021;36:3179–87.
20. Fassett DR, Dailey AT, Vaccaro AR. Vertebral artery injuries associated with cervical spine injuries: a review of the literature. J Spinal Disord Tech. 2008;21:252–8.
21. Desouza RM, Crocker MJ, Haliasos N, Rennie A, Saxena A. Blunt traumatic vertebral artery injury: a clinical review. Eur Spine J. 2011;20:1405–16.
22. Tulyapronchote R, Selhorst JB, Malkoff MD, Gomez CR. Delayed sequelae of vertebral artery dissection and occult cervical fractures. Neurology. 1994;44:1397–9.
23. Cothren CC, Biffl WL, Moore EE, Kashuk JL, Johnson JL. Treatment for blunt cerebrovascular injuries: equivalence of anticoagulation and antiplatelet agents. Arch Surg. 2009;144:685–90.
24. Kim Y-K, Schulman S. Cervical artery dissection: pathology, epidemiology and management. Thromb Res. 2009;123:810–21.
25. Gottesman RF, Sharma P, Robinson KA, et al. Imaging characteristics of symptomatic vertebral artery dissection: a systematic review. Neurologist. 2012;18:255–60.
26. Emmett KP, Fabian TC, DiCocco JM, Zarzaur BL, Croce MA. Improving the screening criteria for blunt cerebrovascular injury: the appropriate role for computed tomography angiography. J Trauma. 2011;70:1058–63; discussion 1063-5.
27. Burlew CC, Biffl WL, Moore EE, Barnett CC, Johnson JL, Bensard DD. Blunt cerebrovascular injuries: redefining screening criteria in the era of noninvasive diagnosis. J Trauma Acute Care Surg. 2012;72:330–5; discussion 336-7, quiz 539.
28. Fabian TC, Patton JH, Croce MA, Minard G, Kudsk KA, Pritchard FE. Blunt carotid injury. importance of early diagnosis and anticoagulant therapy. Ann Surg. 1996;223:513–22; discussion 522-5.
29. Bromberg WJ, Collier BC, Diebel LN, et al. Blunt cerebrovascular injury practice management guidelines: the Eastern Association for the Surgery of Trauma. J Trauma. 2010;68:471–7.
30. Chen C-J, Tseng Y-C, Lee T-H, Hsu H-L, See L-C. Multisection CT angiography compared with catheter angiography in diagnosing vertebral artery dissection. AJNR Am J Neuroradiol. 2004;25:769–74.
31. Vertinsky AT, Schwartz NE, Fischbein NJ, Rosenberg J, Albers GW, Zaharchuk G. Comparison of multidetector CT angiography and MR imaging of cervical artery dissection. AJNR Am J Neuroradiol. 2008;29:1753–60.
32. Miller PR, Fabian TC, Bee TK, et al. Blunt cerebrovascular injuries: diagnosis and treatment. J Trauma. 2001;51:279–85, discussion 285-6.
33. Kasner SE. CADISS: a feasibility trial that answered its question. Lancet Neurol. 2015;14:342–3.
34. D'Souza K, Birnie BW, Ko YM, Evans DC, Field TS, Joos É. Management of blunt cerebrovascular injuries at a Canadian level 1 trauma centre: are we meeting the grade? Can J Surg. 2022;65:E303–9.
35. Su T-H, Chan Y-L, Lee J-D, et al. To load or not to load? Aspirin loading in acute ischemic stroke: a study of clinical outcomes. J Stroke Cerebrovasc Dis. 2016;25:2439–47.
36. Wang Y, Wang Y, Zhao X, et al. Clopidogrel with aspirin in acute minor stroke or transient ischemic attack. N Engl J Med. 2013;369:11–9.
37. Brommeland T, Helseth E, Aarhus M, et al. Best practice guidelines for blunt cerebrovascular injury (BCVI). Scand J Trauma Resusc Emerg Med. 2018;26:90.

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