The Journal of Laryngology & Otology
March 2004, Vol. **118,** pp. 193–198

Pulsatile tinnitus as a symptom of cervicocephalic arterial dissection

Outi Pelkonen, M.D., Tapani Tikkakoski, M.D.*, Jukka Luotonen, M.D.†, Kyösti Sotaniemi, M.D.‡

Abstract
The aim of this study was to investigate pulsatile tinnitus as a presenting symptom in cervicocephalic arterial dissection (CCAD). Of the 136 consecutive patients with confirmed CCAD, 16 presented with pulsatile tinnitus. On admission 10 patients presented with subjective tinnitus and five with objective tinnitus, tinnitus being the only presenting symptom in one case. In one further case with bilateral ICA dissection (ICAD) subjective tinnitus appeared three months after the initial symptoms of arterial dissection, despite a contralateral cervical bruit being evident on admission. Thirteen patients presented with headache or neck pain. Ischaemic symptoms were detected in six and Horner's syndrome in four patients. Vertigo and dysgeusia were reported in two patients each. Arterial dissection involved unilateral ICA in 11, bilateral ICA in two, unilateral vertebral artery (VA) in two and bilateral ICA and bilateral VA in one patient. In angiography the most common finding was irregular stenosis, and the majority of these abnormalities normalized during follow-up. To avoid delay in diagnosis a high index of suspicion and early angiography (digital subtraction or magnetic resonance angiography) are warranted.

Key words: Tinnitus; Vascular Diseases; Angiography

Introduction

Pulsatile tinnitus is an uncommon otological symptom, but accurate diagnosis is essential because a treatable aetiology can be identified in most patients.[1] It may be the presenting and only symptom of a serious and potentially life-threatening pathology, such as dural arteriovenous fistula, carotid cavernous fistula, atherosclerotic carotid stenosis, cerebral aneurysm or arterial dissection. Vascular variants and paraganglioma are common differential diagnostic problems.

Tinnitus is defined as pulsatile when the patient describes a sound synchronous with the heartbeat. When it is only heard by the patient, it is classified as subjective. When it is audible by auscultation, it is described as objective. In arterial dissection pulsatile tinnitus can present as the sole or an associated symptom.[2] In one study spontaneous ICAD caused pulsatile tinnitus in five of 84 patients,[3] whereas in another study none of the 100 patients with pulsatile tinnitus had ICAD.[1] Recently, unique cases have been reported[4–6] but no studies have focused specifically on the relationship between cervicocephalic arterial dissection and tinnitus, characterizing the prevalence, time course, the ratio of ICAD and vertebal artery dissections (VAD) or patient outcome. Our group has previously reported angiographic findings of extracranial and intracranial arterial dissections.[7,8] In this single-centre study we describe pulsatile tinnitus as the sole or associated symptom in cervicocephalic dissections.

Materials and methods

The medial records and films of the 136 consecutive patients diagnosed to have cervicocephalic artery dissection at the University Hospital of Oulu between August 1982 and March 2002 were reviewed by a radiologist (OP) and a neuroradiologist (TT). It was found that 114 patients had extracranial dissections: 62 unilateral and 10 bilateral ICADs, 30 unilateral and nine bilateral VAD, one ICAD + VAD, one ICAD + bilateral VAD, and one bilateral ICAD + bilateral VAD. Twenty-two patients had intracranial dissections: six unilateral ICADs, six unilateral and one bilateral VAD, three ICAD + medial cerebral artery dissection (MCAD), three MCADs, one posterior inferior cerebellar artery dissection (PICAD), one posterior cerebral artery dissection (PCAD), and one dissection of the basilar artery. Of these, 16 (11.8 per cent) patients (nine women and seven men, mean age 43.8 years, range 33–55 years) presented with pulsatile tinnitus as the sole or an associated symptom, and these patients constitute the material for this study.

From the Departments of Diagnostic Radiology, Otorhinolaryngology† and Neurology‡ Oulu University Hospital, Finland and the Department of Radiology* Keski-Pohjanmaa Central Hospital, Finland.
Accepted for publication: 23 December 2003.

Follow-up data were obtained from medical records, and in 13 patients also by telephone interview (OP) in November 2002.

All patients had undergone clinical examination and all but one cerebral computed tomography (CT). Two patients had undergone cerebral magnetic resonance imaging (MRI) at 1 Tesla. Intra-arterial digital substraction angiography via the transfemoral route was performed in all patients, and details of the examination are described elsewhere.[7,8] All patients had undergone aortic arch injections and 12 selective common carotid, vertebral or semiselective subclavian injections at least in two orthogonal directions. The symptoms, cranial CT, initial and follow-up angiographic findings and patient outcomes were evaluated.

Results

Details of the patients are presented in Table I. On admission, 10 patients presented with subjective tinnitus and five with objective tinnitus. In one of them (Case 2) pulsatile tinnitus was the only presenting symptom. In one further patient (Case 4) subjective tinnitus appeared three months after the first symptoms of bilateral ICAD, despite a contralateral cervical bruit being evident on admission. Thirteen patients presented with headache or neckache. Manifestations of cerebral ischaemia (dysphasia, aphasia, amaurosis fugax, hemiparesis) were detected in six patients. Horner's syndrome was found in four. Vertigo and dysgeusia were reported in two patients each. Possible predisposing factors included a history of arterial hypertension in eight, physical strain in four, fibromuscular dysplasia in two, whiplash injury in two, and a minor head trauma with possible concomitant whiplash injury in two.

The mean interval from the onset of symptoms to cranial CT was 26 days (range 0–300 days). Cranial CT was normal in all but two patients. A right cerebellar infarct was detected in one patient (Case 1) and a right parietal infarct in another (Case 4), both of them being symptomatic. Three further patients had clinically evident brain (Cases 5 and 12) or brainstem infarct (Case 3), despite a normal CT scan. Brain MRI was normal in both patients (Cases 11 and 14).

The mean interval from the onset of symptoms to the diagnosis confirmed by angiography was 16.6 days (range 3–71 days). The dissections found were as follows: unilateral ICAD 11, bilateral ICAD two, unilateral VAD two, and bilateral ICAD and bilateral VAD one. In angiography the most common finding was irregular stenosis, and the majority of these abnormalities normalized during follow-up (Figures 1(a)(b) and 2(a)(b)).

In 14 patients the initial treatment was i.v. heparin followed by warfarin (mean five months, range three–12 months), which was followed by acetylsalicylic acid. Ligation of the internal carotid artery was performed in one patient because of a pseudoaneurysm (Case 2). No medical treatment except acetylsalicylic acid was used in one patient who had subjective tinnitus and a pseudoaneurysm in the right ICA (Case 16).

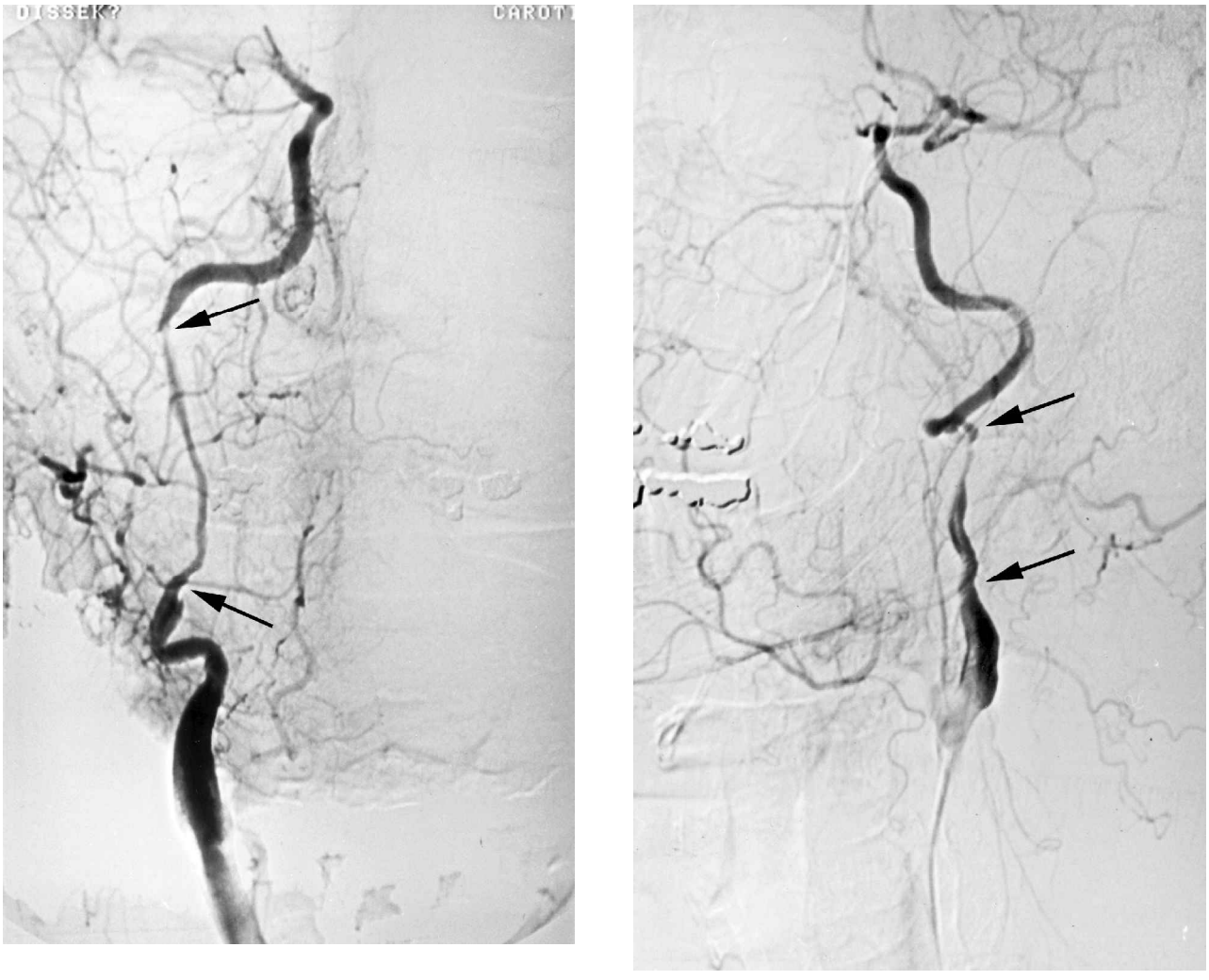
Duration of the pulsatile tinnitus varied greatly, from five min (Case 1) to one to seven days (Cases 3, 5, 8), to one to eight weeks (Cases 4, 10, 11, 12, 13, 14, 15), up to six months (Cases 6, 16). Three patients could not be reached by telephone and the duration of pulsatile tinnitus could not be defined based on medical records (Cases 2, 7, 9).

TABLE I
DETAILS OF THE 16 PATIENTS PRESENTING WITH PULSATILE TINNITUS

Patient*	Symptoms and signs	Affected vessel/initial/follow-up angiographic findings	Outcome
1/39/M	ST, headache, amaurosis fugax, vertigo	Right VA/occlusion at C2–7/no change	Asymptomatic
2/42/F	OT	Left ICA/pseudoaneurysm and intimal flap/post-operative occlusion	Poor
3/52/F	ST, vertigo, nausea, headache, Horner's sdr	Right VA/irregular stenosis at C1/2/normal	Nearly asymptomatic
4/41/M	Bruit, headache, neckache, hemiparesis, ST three months later	Bilateral ICA/irregular stenosis/normal	Poor
5/55/M	ST, aphasia, hemiparesis	Left ICA/irregular stenosis/normal	Nearly asymptomatic
6/53/M	ST, dysphasia, headache, Horner's sdr	Left ICA/irregular stenosis and pseudoaneurysm/pseudoaneurysm	Asymptomatic
7/47/F	ST, headache, Horner's sdr	Right ICA/irregular stenosis/normal	Nearly asymptomatic
8/42/M	OT, headache, Horner's sdr	Bilateral ICA/irregular stenosis/normal	Asymptomatic
9/36/F	OT, dysgeusia	Right ICA/pseudoaneurysm/pseudoaneurysm	Nearly asymptomatic
10/38/F	OT, headache, dysphasia	Right ICA/double lumen/double lumen	Asymptomatic
11/50/F	ST, headache, dysgeusia	Right ICA/pseudoaneurysm and intimal flap/no change	Asymptomatic
12/41/F	OT, headache	Left ICA/irregular stenosis and pseudoaneurysm/nearly normal	Asymptomatic
13/37/F	ST, headache	Left ICA/irregular stenosis/nearly normal	Nearly asymptomatic
14/33/F	ST, headache, dysphasia	Bilateral ICA and bilateral VA/irregular stenosis/normal	Asymptomatic
15/48/M	ST, headache	Left ICA/irregular stenosis/normal	Asymptomatic
16/47/M	ST, neckache	Right ICA/pseudoaneurysm/not done	Nearly asymptomatic

*Case number, age, gender; ST = subjective tinnitus; OT = objective tinnitus; VA = vertebral artery; ICA = internal carotid artery

https://doi.org/10.1258/002221504322927955 Published online by Cambridge University Press



(a) (b)

FIG. 1

A 41-year-old man presented with left hemiparesis, head and neck pain. On admission a left-sided bruit was evident on auscultation. On the third day of hospitalization bilateral internal carotid artery dissection (arrows) was detected in common carotid arteriography. (a) Right common carotid injection. (b) Left common carotid injection.

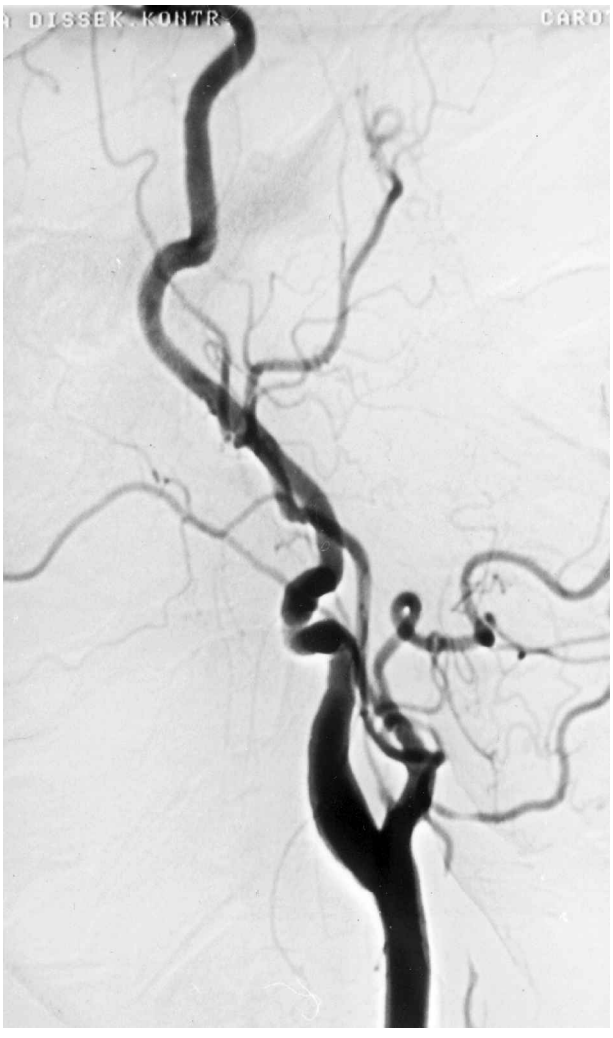
Follow-up angiography was performed in 15 patients three to 18 (mean 4.4 months) after the primary angiography. In eight patients with stenotic lesions the vessels showed complete or nearly complete normalization, and all but one of the pseudoaneurysms, intimal flaps and one vertebral artery occlusion remain unchanged (Table I).

According to the telephone interview (mean follow-up nine years, range one year and 10 months to 14 years and three months) six patients were asymptomatic, six were nearly asymptomatic and able to continue their previous work, and one patient had severe neurological deficits. According to the medical records of the remaining three cases not interviewed, two patients were asymptomatic and one had severe neurological deficits. No recurrences were detected during follow-up.

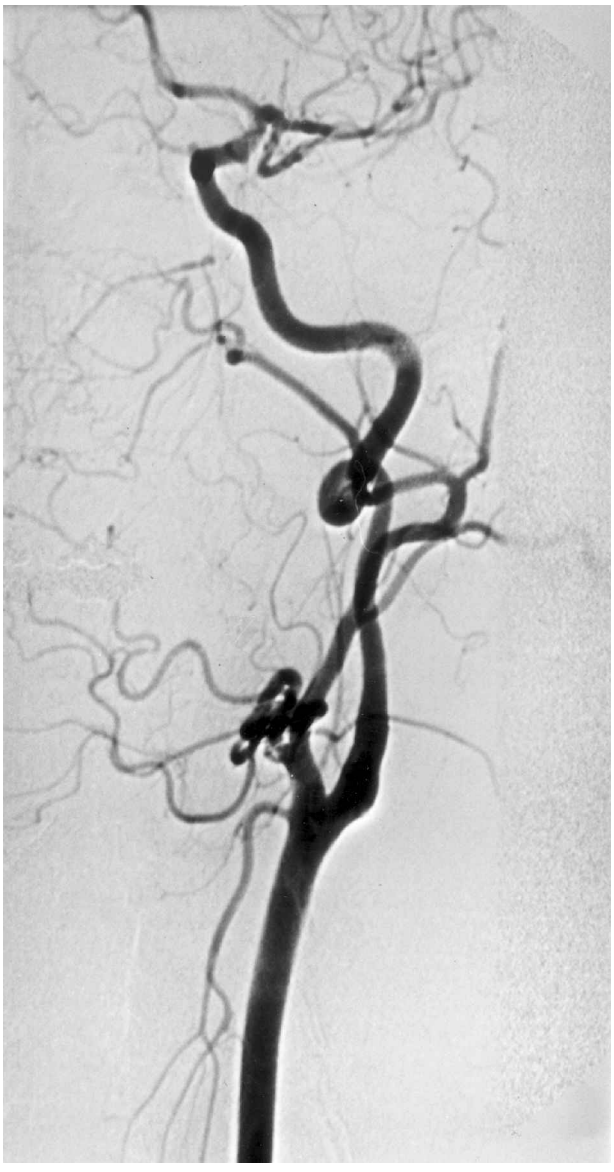
Discussion

Dissection occurs when the intima or media of the arterial wall disrupts, causing an intramural haematoma into the subintimal, medial or subadventitial layers. Dissection may be spontaneous or traumatic. Blunt or penetrating trauma to the neck is the most common cause of traumatic dissection.[9,10] Sudden severe stretch and/or compression, or prolonged extension, flexion or rotation of the neck may also cause extracranial dissections by stretching the ICA over the transverse processes of the upper cervical vertebrae or the VA over the lateral masses of the C1 and C2 vertebral bodies.[9–13] This was probably the case in two – possibly four – of our patients with whiplash injury, which is often reported as a predisposing factor in CCAD patients.[14–16] The diagnosis of CCAD is based on appropriate clinical presentation, characteristic radiological findings and demonstration of the absence of atherosclerotic disease.[17] Thus, the diagnosis is presumptive, because antemortem pathologic specimens are not available. In Minnesota, the average annual incidence of ICAD for all ages was 2.6/100 000,[18] but the true incidence is probably underestimated in the

https://doi.org/10.1258/002221504322927955 Published online by Cambridge University Press



(a)



(b)

FIG. 2

Right-sided subjective tinnitus appeared three months after left hemiparesis and persisted for two months. Follow-up carotid angiograms after three and a half months' anticoagulation showed complete recanalization of the dissections, but the patient's outcome was poor. (a) Right common carotid injection. (b) Left common carotid injection.

literature, because dissection, as in our previous study,[7] may be asymptomatic and patients with mild symptoms may not undergo extensive investigations such as angiography. In addition, arterial dissection is a dynamic disease and, with longer periods between the onset of symptoms and angiography, it becomes a rare finding.[19]

Spontaneous dissection is increasingly recognized as a cause of brain infarct in young adults and has a wide spectrum of symptoms. Head and neck pain, followed by ischaemic cerebral symptoms, Horner's syndrome and cranial neuropathies, are the most common symptoms.[2,19,20]

In this study special attention was paid to the symptom of pulsatile tinnitus. Pulsatile tinnitus is almost always the result of the sound of non-laminar blood flow that is transmitted to the inner ear. This can occur in systemic disease, causing general alteration of the haemodynamics, or in local disorders that are anatomically close to or within the petrous bone. Dural arteriovenous or carotid cavernous fistulae, extracranial and intracranial arteriovenous malformations, cerebral aneurysms, fibromuscular dysplasia, atherosclerosis or ICAD are the most common arterial causes of pulsatile tinnitus.[1,3] Pulsatile tinnitus associated with extracranial cervicocephalic arterial dissection has received little attention. In a review of 140 extracranial ICADs from 1975–1983 pulsatile tinnitus was the only symptom in four per cent, and an associated symptom in 35 per cent of patients.[2] Ast *et al.*[20] reported subjective tinnitus as a symptom in 7.5 per cent (five of out 68) of ICAD patients. In another study subjective or objective pulsatile tinnitus was

https://doi.org/10.1258/002221504322927955 Published online by Cambridge University Press

reported in 50 per cent (18/36) of patients with ICAD.[17] Steinke *et al.*[21] reported pulsatile tinnitus in 12 per cent of 48 patients. In two recent studies the frequency of pulsatile tinnitus was 16 per cent[22] and 27 per cent[23] out of 181 and 161 patients, respectively. Tinnitus was an associated symptom in all. Almost 12 per cent of our 136 patients with cervicocephalic dissection had pulsatile tinnitus, and in the majority it was an associated symptom and in only one was it the only presenting symptom. Contrary to pervious works[2,20,22] our material is a single-centre large representative series of consecutive angiographically confirmed dissections. Follow-up angiograms were done in all but one patient, and similar series with long follow-up data have not been published previously.

VAD has been thought to be a very rare cause of pulsatile tinnitus: in two studies VAD was never the cause of pulsatile tinnitus,[22,23] and in a third study only one patient out of 25 with VAD presented with tinnitus.[24] Of our patients 49/136 had VAD (three patients also simultaneous ICAD) and, of these, three (6.1 per cent) had pulsatile tinnitus. In one study ICAD without ischaemic events had a significantly higher prevalence of Horner's syndrome, cranial nerve palsy and normal ICA findings, whereas patients with ICAD and ischaemic events had a higher prevalence of high-grade (>80% per cent) stenoses and occlusions of the ICA. Pulsatile tinnitus was seen equally as a presenting symptom in both groups.[22] Cerebral infarct was diagnosed in five of our patients, and in two of these CT showed an infarct.

In the majority of our patients the pulsatile tinnitus subsided in less than two months and persisted for up to six months in only two patients. In previous case reports tinnitus resolved in one week[6] or in one month.[5] In another study tinnitus was transient in 10 patients, disappearing in 24 h to four months.[17]

We, like others,[4,5] used anticoagulant treatment in most cases (14/16), one received acetylsalicylic acid and one underwent ligation of the ICA because of a pseudoaneurysm. Contrary to Ast *et al.*,[20] none of our patients developed cerebral embolic events or worsening of the neurological condition after the start of treatment. In extracranial ICAD the majority of strokes occur in the first few days after the onset of the first symptoms, but it can occur as much as one month later[25] Despite multivessel disease the prognosis proved excellent or good in the majority of our patients. The risk of recurrent dissection is one per cent per year,[26] and we had no recurrences.

MRI can demonstrate the intramural crescentic haematoma itself, and when the dissection occurs in the subadventitial layer without relevant narrowing of the arterial lumen, or when the aneurysm is thrombosed, conventional angiography does not yield the diagnosis.[27] MRI and MR angiography (3D-TOF) has proved sensitive (84 and 95 per cent, respectively) and specific (99 and 99 per cent, respectively) in diagnosing ICAD, but the sensitivity proved poor (60 and 20 per cent, respectively) in detecting VAD.[28] MRI with MR angiography has gained popularity and is now the first-line imaging investigation for both cerviococephalic artery dissection and pulsatile tinnitus in many institutions.[1,4] Despite the advantages of MRI it is not readily available. In this subgroup of patients angiography has proved safe, and no worsening of symptoms has occurred during angiography at our institution. Our policy is to do four-vessel angiography to detect or rule out multivessel dissection, which occurred in 18.7 per cent (three out of 16) of our patients. In addition, contrary to MRI and MRA, only this option enables dural arteriovenous fistula to be excluded in a patient with pulsatile tinnitus and normal otoscopy.[29] Dissection is a dynamic disease, and although the prognoses of the patient and the affected vessel may diverge, follow-up angiography often finally confirms the diagnosis, and as it may also entail therapeutic decisions, we consider follow-up angiography appropriate.[7]

- **Previous case reports have described a relationship between cervicocephalic arterial dissection and pulsatile tinnitus**
- **This is a retrospective study that looked at the prevalence of pulsatile tinnitus in a population of 136 patients with confirmed arterial dissection**
- **Of this cohort, 10 patients presented with subjective and five with objective tinnitus**
- **Other symptoms included headache and neck pain, ischaemic symptoms, Horner's syndrome, vertigo and dysgeusia**
- **The treatment and outcome in these cases is**

We conclude that more than one out of 10 patients with cervicocephalic arterial dissection present with pulsatile tinnitus, and it may even be the only symptom of extracranial ICAD, VAD or multivessel dissection, although the majority of patients have concomitant head or neck pain, ischaemic brain symptoms, Horner's syndrome or cranial neuropathies. Tinnitus itself is a common symptom, but when it presents as pulsatile, either subjective or objective, it should remind the physician of the possibility of a vascular disorder. To avoid delay in diagnosis a high index of suspicion and early angiography (digital subtraction or magnetic resonance) is warranted.

References

1 Sismanis A, Smoker WRK. Pulsatile tinnitus: recent advances in diagnosis. *Laryngoscope* 1994;**104**:681–8
2 Hart RG, Easton JD. Dissections of cervical and cerebral arteries. *Neurol Clin* 1983;**1**:155–82
3 Waldvogel D, Mattle HP, Sturzenegger M, Schroth G. Pulsatile tinnitus – a review of 84 patients. *J Neurol* 1998;**245**:137–42
4 Harris JP, Horlbeck DM. Brew KH. Unique case of pulsatile tinnitus. *Ann Otol Rhinol Laryngol* 2000;**109**:1103–6

https://doi.org/10.1258/002221504322927955 Published online by Cambridge University Press

5 Vories A, Liening D. Spontaneous dissection of the internal carotid artery presenting with pulsatile tinnitus. *Am J Otolaryngol* 1998;**19**:213–15
6 Depauw P, Caekeneke J, Vanhoenacker P. Objective pulsatile tinnitus caused by intrapetrous dissecting aneurysm. *Clin Neurol Neurosurg* 2001;**103**:197–9
7 Pelkonen O, Tikkakoski T, Leinonen S, Pyhtinen J, Lepojärvi M, Sotaniemi K. Extracranial internal carotid and vertebral artery dissections: angiographic spectrum, course and prognosis. *Neuroradiology* 2003;**45**:71–7
8 Pelkonen O, Tikkakoski T, Leinonen S, Pyhtinen J, Sotaniemi K. Intracranial arterial dissection. *Neuroradiology* 1998;**40**:442–7
9 Provenzale JM, Barboriak DP, Taveras JM. Exercise-related dissection of craniocervical arteries: CT, MR and angiographic findings. *J Comput Assist Tomogr* 1995;**19**:268–76
10 Mokri B. Traumatic and spontaneous extracranial internal carotid artery dissections. *J Neurol* 1990;**237**:356–61
11 Stringer WL, Kelly DL Jr. Traumatic dissection of the extracranial internal carotid artery. *Neurosurgery* 1980;**6**:123–30
12 Sherman DG, Hart RG, Easton JD. Abrupt change in head position and cerebral infarction. *Stroke* 1981;**12**:2–6
13 Sheth TN, Winslow JL, Mikulis DJ. Rotational changes in the morphology of the vertebral artery at a common site of artery dissection. *Can Assoc Radiologists* 2001;**52**:236–41
14 Viktrup L, Knudsen GM, Hansen SH. Delayed onset of fatal basilar thrombotic embolus after whiplash injury. *Stroke* 1995;**26**:2194–6
15 Janjua KJ, Goswami V, Sagar G. Whiplash injury associated with acute bilateral internal carotid arterial dissection. *J Trauma* 1996;**40**:456–8
16 Chung YS, Han DH. Vertebrobasilar dissection: a possible role of whiplash injury in its pathogenesis. *Neurol Res* 2002;**24**:129–38
17 Mokri B, Sundt TM, Houser OW, Piepgras DG. Spontaneous dissection of the cervical internal carotid artery. *Ann Neurol* 1986;**19**:126–38
18 Schievink WI, Mokri B, Whisnant JP. Internal carotid artery dissection in a community Rochester, Minnesota, 1987–1992. *Stroke* 1993;**24**:1678–80
19 Haapaniemi H, Salonen O, Hillbom M, Juvela S. Carotid arterial dissection as a cause of severe brain infarction in young adults. *J Stroke Cerebrovasc Dis* 1996;**6**:89–92
20 Ast G, Woimant F, Georges B, Laurian C, Haguenau M. Spontaneous dissection of the internal carotid artery in 68 patients. *Eur J Med* 1993;**2**:466–72
21 Steinke W, Rautenberg W, Schwartz A, Hennerici M. Noninvasive monitoring of internal carotid dissection. *Stroke* 1994;**25**:998–1005
22 Baumgartner RW, Arnold M, Baumgartner I, Mosso M, Gönner F, Studer A *et al.* Carotid dissection with and without ischemic events. Local symptoms and cerebral artery findings. *Neurology* 2001;**57**:827–32
23 Silbert PL, Mokri B, Schievink WI. Headache and neck pain in spontaneous internal carotid and vertebral artery dissections. *Neurology* 1995;**45**:1517–22
24 Mokri B, Houser OW, Sandok BA, Piepgras DG. Spontaneous dissections of the vertebral arteries. *Neurology 1988*;**38**:880–5
25 Biousse V, D'Anglejan-Chatillon J, Touboul PJ, Amarenco P, Bousser MG. Time course of symptoms in extracranial carotid artery dissections. A series of 80 patients. *Stroke* 1995;**26**:235–9
26 Schievink WI, Mokri B, O'Fallon WM. Recurrent spontaneous cervical-artery dissection. *N Engl J Med* 1994;**330**:393–7
27 Sturzenegger M, Huber P. Cranial nerve palsies in spontaneous carotid artery dissection. *J Neurol Neurosurg Psychiatry* 1993;**56**:1191–9
28 Levy C, Laissy JP, Raveau V, Amareco P, Servois V, Bousser MG *et al.* Carotid and vertebral artery dissections: three-dimensional time of flight MR angiography and MR imaging versus conventional angiography. *Radiology* 1994;**190**:97–103
29 Shah SB, Lalwani AK, Dowd CF. Transverse/sigmoid sinus dural arteriovenous fistulas presenting as pulsatile tinnitus. *Laryngoscope* 1999;**109**:54–8

Address for correspondence:
Outi Pelkonen, M.D.,
Department of Diagnostic Radiology,
Oulu University Hospital,
Box 50,
FIN-90029 OYS,
Finland.

Fax: 358-8-3155420
E-mail: outi.pelkonen@ppshp.fi

O. Pelkonen M.D., takes responsibility for the integrity of the content of the paper.
Competing interests: None declared