Case Report

Neck pain and headache as a result of internal carotid artery dissection: implications for manual therapists

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1. Introduction

Physical therapy (PT) knowledge and literature relating to vascular issues in the cervical spine has traditionally paid particular attention to vertebrobasilar insufficiency (VBI) related to manipulation. Although internal carotid artery dissection (ICAD) and its sequelae are well reported in medical and chiropractic literature, only a few reports appear in PT texts. Internal carotid artery pathology has been documented as presenting with acute onset headache, facial and neck pain (Sturzenegger, 1995). Indeed, Smith et al. (2003) have suggested quite logically, that patients may seek spinal manipulative therapy (SMT) in the presence of a pre-existing cervical artery dissection which presents as neck and/or head pain. We report a case of ICAD in a 51-year-old male Physiotherapist. The ICAD appears to be the result of sneezing whilst in full cervical rotation, which to the authors’ knowledge has not been previously documented. The case study lends weight to the contention that arterial injury can present as mechanical onset neuromusculoskeletal (NMS) pain.

The authors contend that subtle clues obtained during the subjective history taking and clinical reasoning process may alert the aware clinician of a potential vascular hypothesis, thus informing the decision with regard to management and potential SMT.

Such case studies illustrate that a knowledge of haemodynamic issues relating to the cervical arteries are an essential prerequisite for all manual therapists using movement techniques including exercise and mobilisation. This case illustrates that certain individuals with no apparent risk factors may be prone to arterial injury as a result of cervical movement and trauma.

2. Case report

This case report is compiled with consent from the account and records of the patient.

A 51-year-old Chartered Physiotherapist and Manual Therapy tutor attended a pre-Christmas dinner party. Whilst sitting at the table he felt a sudden urge to sneeze. Conscious of the need not to spread his germs over the food, he turned his head fully to the left and sneezed several times. He jarred his neck at the time and gradually became aware of left sided mid-upper cervical pain and “aching” in the region of the left temporomandibular joint and mandible (see Fig. 1). He was able to complete his meal in some discomfort and felt that he had “strained his neck”.

On waking the next day he was aware of ongoing symptoms, presenting as pain in the left sub-occipital region and he had difficulty turning his head to the left. He was also aware of a general left sided headache affecting the left frontal region and eye. These symptoms remained constant at a pain level estimated at 6/10 and continued throughout the day requiring the use of analgesia/NSAIDS. Unable to contact a Physiotherapist for treatment over the Christmas holiday period, he
self-managed his condition with continued analgesia. There was no change in his condition over a 48-h period with the distribution and level of pain remaining constant.

Whilst driving to work 48 h later he became aware of ptosis (drooping) of the left eyelid and constriction of the pupil on the left. He realised that he had developed Horner’s Syndrome—defined as injury or interruption to the sympathetic nerves of the face (Thomas and Venes, 1999).

The patient consulted his GP, who discussed the case with a Professor of Neurology. It was suggested by the latter that there might be a possible dissection of the carotid artery based on the clinical picture. He was advised to monitor his symptoms and call back urgently if there was any alteration particularly with regard to any signs of transient ischaemic attack (TIA). Increasingly concerned he presented to the local accident and emergency department. An ultrasound scan and a subsequent CT scan at this stage failed to confirm the clinical diagnosis. However, he was admitted for one night and prescribed Warfarin and Clexane.

A diagnosis of ICAD was eventually confirmed by magnetic resonance arteriography (MRA) 2 weeks post-injury (Fig. 2) and a management plan of further anticoagulation (Warfarin combined with Aspirin 300 mg) implemented. He was advised to rest from work (6 weeks) and particularly exertion because of the potential danger of embolisation.

One month after the onset of the symptoms the patient was still in considerable discomfort and had developed paraesthesia in the occipital region and a sensation of a “brittle feeling” as if “something was about to break”. He still required NSAIDS to manage the symptoms. In addition he had become aware of pulsatile tinnitus, which was ascribed, by the vascular specialist, to the formation of collateral circulation in the external carotid.

The patient was monitored with Duplex ultrasound and regular blood tests over a 12-month period. He returned to work 6 weeks after the onset of his symptoms. The pain and Horner’s syndrome partially resolved over the next 12 months though the patient still reports mild ptosis and intermittent transient pain in the cervical/carotid and frontal region. The pain appears related to pressure on the supraclavicular region or effort of the upper limb and shoulder girdle (i.e. during his work as a Physiotherapist).

Fig. 1. Pain charts showing symptom distribution immediately after onset.

Fig. 2. Magnetic Resonance Arteriogram (MRA) demonstrating the left internal carotid artery (ICA) tapering to an occlusion (arrow A). This classic ‘rat tail’ sign is consistent with the diagnosis of ICAD. The blood flow within this vessel should normally be comparable with the opposite sided ICA (arrow B).
The patient reported no underlying risk factors for vascular disease. There was no family history of vascular disease and the patient was not taking any medication. The patient could not relate to any past medical history of a single traumatic event. However, he did make the observation that as a postgraduate physiotherapy tutor he had spent a number of years involved in teaching practical cervical treatment techniques involving varying degrees of cervical range of motion to end range. Furthermore, he had for a number of years suffered an undiagnosed problem of right-sided upper limb and hand fatigue and discomfort (Fig. 3) associated with playing a percussion instrument. Later examination of the right side revealed blanching of the hand associated with prolonged effort whilst playing the spoons.

A further interesting feature of the case was the development (“some weeks” after the initial injury) of symptoms of paraesthesia and discomfort on the right side of the head. These symptoms were investigated and though unexplained by the vascular consultant were thought not to be related to the arterial dissection. They have since been treated successfully with manual therapy and have resolved.

3. Discussion

The carotid artery despite its common link to stroke and TIA is seldom considered in PT education as a potential source of symptoms related to cervical and head pain syndromes. It has however, been implicated directly in case studies leading to fatal dissection following manipulation (Peters et al., 1995). Its significance in relation to differential diagnosis and treatment is not commonly considered in PT undergraduate or postgraduate training. Whilst it is outside the scope of this paper to detail the precise haemodynamic factors involved, it is well documented that the carotid artery is subject to external and internal stresses linked to compression, kinking and looping (Grego et al., 2003). Rivett (1997) has already detailed a theoretical basis for intimal trauma to the carotid artery due to compressive factors in the upper cervical region. Duplex flow ultrasonography has demonstrated reduced flow in the contralateral internal carotid artery during end range cervical rotation (Refshauge, 1994). The hypothesised mechanism was compression or constriction of the artery between the layers of muscular tissue, which are stretched on full end range rotation. Although the precise pathological mechanism is unknown this case study is important because it provides a salient example of how an arterial lesion can present as a typical musculoskeletal presentation—mechanical onset of neck pain, headache and restriction of range of motion.

This patient, a physiotherapist himself, would by his own admission have sought treatment during the first 48 h (i.e. before the onset of neurological symptoms) had it not been for the holiday period. It is not outside the realms off possibility for a patient presenting with those initial symptoms to be treated with postural advice, exercise, spinal mobilisation techniques and/or manipulation. Indeed it is a commonly held view backed up by current research that acute onset “mechanical” pain responds well to early manipulation (Cassidy et al., 1992; Baltaci et al., 2001).

Unfortunately clinicians do not have the equivalent of a dermatomal or myotomal map for arteries. However, it is known that arterial pathology can mediate pain at various anatomical sites such as the aorta for the lumbar spine (Yabuki et al., 1999), the external iliac artery for the lower limb (Schep et al., 2002) and the subclavian artery for the shoulder and arm (Yao, 1998). Nicholls et al. (1993) were able to conclusively demonstrate the ability of arteries to produce neck, head and even trapezious pain via the inflation of an angioplasty balloon inside the vertebral and basilar arteries in healthy subjects. Similarly Munari et al. (1994), demonstrated cervical, facial and headache in patients undergoing percutaneous angioplasty of the carotid artery.

In this case the patient described unilateral neck/ facial pain and severe headache with a mechanical onset. Sturzenegger (1994) detailed acute onset of neck pain and headache in a series of 14 patients with both vertebral and carotid artery dissection. The paper described headaches “unlike any other” with a
However, it should not be misconstrued as a green light that, and is included for interest and discussion. Therapy following this particular case of ICAD is purely well have led to a different outcome.

Whilst arteries are known to be very tough or resilient on their outer (tunica adventitia) layer; repeated stress to the inner layer (endothelium) has been shown to lead to pathological changes in the arterial wall at various anatomical sites (Ross, 1993; Schep et al., 2002). The patient in this case described one potential source of arterial trauma—his repeated practical teaching sessions involving various cervical manoeuvres and sustained end range positions. The link is purely hypothetical but is worthy of consideration when arterial pathobiomechanics are considered. One further significant finding in the history of this case was the co-existence of another pain/fatigue syndrome affecting the upper limb. This undiagnosed problem had been long standing and pre-existed the arterial dissection by “some years”. However, the nature of the symptoms purely from the history (exercise/activity induced fatigue/discomfort) was strongly suggestive of a vascular hypothesis. As vascular pathology (atherosclerosis for example) is known to co-exist at different, and specific, anatomical sites then this factor adds information to the clinical picture and may inform decision-making.

Of particular interest is the fact that contralateral symptoms (thought to be joint related and not of vascular origin) were successfully treated using upper cervical manual therapy techniques. This perhaps adds some credence to the concept that careful and reasoned manual therapy is not totally contraindicated in cases of resolving arterial dissection—a theory put forward by a number of chiropractic texts (e.g. Michaud, 2002). It is important that Manual Therapists incorporate knowledge of haemodynamic principles related to movement and range using the best of the available scientific evidence into their clinical reasoning and decision-making matrix. However, the authors’ would like to draw the reader’s attention to the fact that this is a special case of a Manual Therapist treating a colleague. As such both the care provider and more importantly the recipient were both acutely aware of the potential risks and benefits of the treatment techniques. Furthermore treatment was provided with fully informed consent. The fact remains however, that the risk of TIA, stroke or death during or following treatment was, whilst unquantifiable, very real. In the case of the more common therapist to patient relationship, the clinical decision-making process and risk–benefit matrix may well have led to a different outcome.

The factual information related to the use of manual therapy following this particular case of ICAD is purely that, and is included for interest and discussion. However, it should not be misconstrued as a green light for manual therapy in such cases. The fact that a catastrophic event did not occur in this case is of course no guarantee that it may not occur in the future.

It is entirely feasible that patients with an acute arterial injury may present themselves for treatment for a perceived “neck sprain” which may or may not actually co-exist. There are two specific considerations for any therapist confronted with that situation:

1. Is the pain mediated from a vascular injury (with or without concomitant NMS injury) such as a dissection or aneurysm?
2. Could treatment techniques in the presence of a NMS lesion alter local haemodynamics and lead to vascular injury?

The conundrum then is to establish quickly via sound history taking, clinical reasoning and risk benefit analysis whether and indeed what type of management is appropriate. This of course requires a basic knowledge of haemodynamic principles and mechanisms of injury or compromise of the arterio-venous system. Interestingly Chiropractic and Osteopathic literature is often a revealing source of educational material and knowledge of the vascular system. Chiropractors have been compelled to improve their knowledge in order to provide a creditable defence against detractors of their profession and manipulative techniques in particular.

Physical Therapists, who tend to manipulate less commonly but, who frequently use through and end range mobilisation techniques, may have been lulled into a false sense of security by literature, which suggests that non-manipulative techniques are less likely to produce cervical spine injury including arterial dissection (Di Fabio, 1999). This assertion is challenged directly by a number of authors (Terrett, 2000; Michaeli, 1993) who contend that it is not the “thrust” that is dangerous but rather the extremes of movement. This theory is backed up to some extent by the numerous case reports of arterial dissection following visits to the hairdresser, yoga, ceiling painting, stargazing and archery (Zetterling et al., 2000). Interestingly as part of their defence of their own techniques some chiropractic texts directly implicate McKenzie cervical end range techniques as being extremely stressful on the vertebral artery (Michaud, 2002). These debates should be seen as healthy rather than destructive and should encourage further research to establish the real effects of movement and treatment techniques in this most vital of areas.

4. Conclusion

It is the authors’ belief that all practitioners who assess and treat the cervical spine with any form of movement technique whether manipulative or not,
require a robust knowledge of the haemodynamic principles affecting that area. This case is an illustration that cervical rotation combined with the "trauma" of sneezing can result in arterial injury which for 48 h at least, presented as a typical mechanical onset of pain and for which there were no obvious contra indications for SMT.

What is clear is that cases like this will present to Manual Therapists and seek treatment. The job of the clinician is then to assess whether the clinical picture fits and then, based on a risk benefit analysis and sound clinical reasoning, decide whether treatment should commence and what format that treatment should take. Acute onset of headache, described as “unlike any other” with or without apparent trauma and unresponsive to manual therapy may be a warning sign of underlying arterial injury.

References


