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CASE REPORT

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An unusual anatomical variant of the left phrenic nerve encircling the transverse cervical artery

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During educational dissection of cadavers, we encountered anatomical variability of the left phrenic nerve (PN). In this cadaver, nerve fibres from C3 and C4 descended and crossed behind the transverse cervical artery (TCA), a branch of the thyrocervical trunk, at the level of the anterior scalene muscle. On the other hand, nerve fibres from C5 descended obliquely above the TCA and then joined the fibres from C3–C4 on the medial side of the anterior scalene muscle to form the PN. To our knowledge, the encircling of the TCA by the left PN in the neck has not yet been reported and may pose a potential risk for nerve compression during movement of the neck. We discuss several types of anatomical variants of the PN and the associated risk during thorax and neck dissection procedures. (Folia Morphol 2021; 80, 4: 1027–1031)

Key words: phrenic nerve, transverse cervical artery, variation

INTRODUCTION

The phrenic nerve (PN) is one of the most important nerves in the body, due to its role in respiration. The PN mainly arises from the fourth cervical ventral ramus (C4) and receives contributions from the third (C3) and fifth (C5) cervical ventral rami in the posterior triangle of the neck. It descends obliquely along the surface of the anterior scalene muscle from the posterior margin to the anterior, then enters the thorax by passing in front of the subclavian artery (SCA). Phrenic nerve injury can lead to diaphragmatic paralysis, temporary dyspnoea, and eventually worsen respiratory function due to the motor supply to the major respiratory muscles of the diaphragm; however, there is scarce literature on the anatomy of the nerve during neck dissection [23].

Unilateral diaphragmatic paralysis is frequently caused by an iatrogenic injury due to the anatomical variants of the PN that are encountered during thorax and neck procedures, including surgical, anaesthetic, or chiropractic complications [10, 19]. There are several anatomical variants of the PN that have been described; however, most are related to the duplication of the PN, as well as medial and lateral deviations of the PN [3]. Duplication of the PN is associated with early developmental events of the nascent diaphragm [20, 22], while aberrant origin and course of the thyrocervical trunk (TCT) arising in the SCA are thought to be the causes of deviations of the PN [16]. In practice, branches of the SCA, as well as branches of the TCT, can compress the PN and become potential sites of nerve impairment [9, 16].

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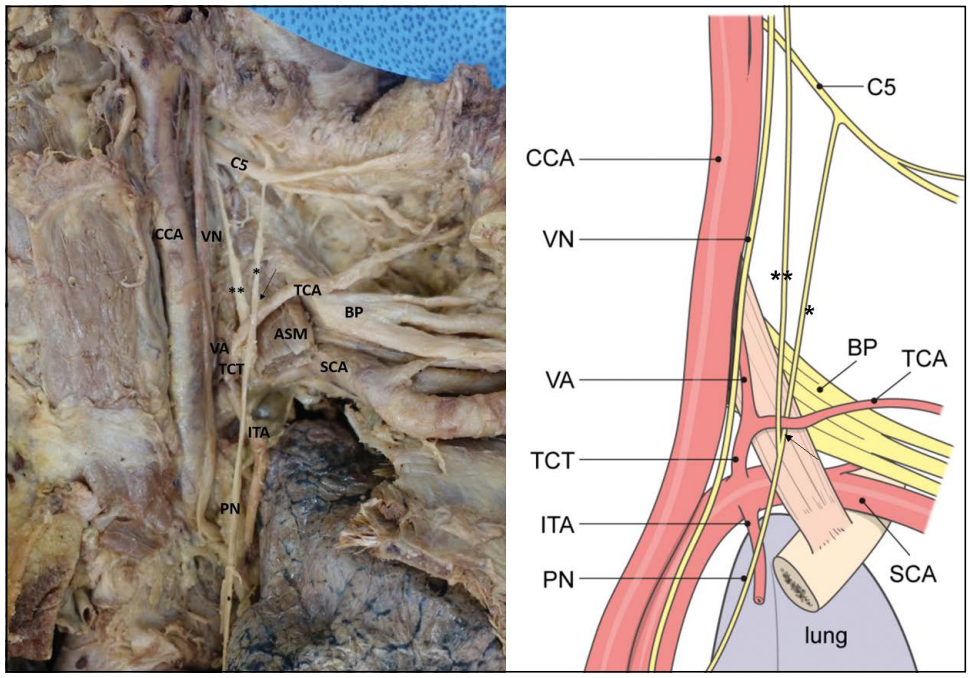

Figure 1. The common carotid artery (CCA) and subclavian artery (SCA) arise directly from the aortic arch. The vagus nerve (VN) enters the thorax between the CCA and SCA. The first portion of the SCA divided into the vertebral artery (VA), the internal thoracic artery (ITA), and the common trunk of thyrocervical trunk (TCT). The common trunk is located close to the medial edge of the anterior scalene muscle (ASM). The nerve fibres from C3 and C4 (**) descend passing the lateral side of VN and then cross the transverse cervical artery (TCA) posteriorly on the anterior scalene muscle. In contrast, the nerve fibres from C5 (*) descend obliquely above the TCA toward the thorax. The fibres from C3–C4 and C5 join together and then form a loop around the TCA. After encircling the TCA, the phrenic nerve (PN) accompanies the ITA descending toward the thorax; **nerve fibres from C3–C4; *nerve fibres from C5, C5 — ventral ramus of C5; BP — brachial plexus.

Injury to the PN caused by anatomic variations of the accessory PN have also been reported [15].

Recently, it has been suggested that the PN might be pulled more laterally by the transverse cervical artery (TCA), a branch of the TCT, based on changes in posture and movement of the neck [9]. In addition, disruption in the normal quality of the prevertebral fascia between the TCA and the PN on the anterior scalene muscle may result in adherence between the two structures and eventually lead to vascular compression of the nerve following surgical and interventional vascular procedures [11].

Thus, it is important to understand the anatomic relationship between anatomic variants of the PN and the TCT itself and/or the TCA in the neck region, in order to reduce postoperative complications. In this report, we present the potential risks of left PN injury, which is caused by the TCA, one of the branches of the TCT, which we believe has not yet been reported.

CASE REPORT

During a routine educational dissection, a rare encircling of the left PN around the TCA was found in an 83-year-old Korean male cadaver. The SCA and common carotid artery arose directly from the aortic arch. The vagus nerve entered the thorax between the common carotid artery and SCA. The first portion of the SCA divided into the vertebral artery, the internal thoracic artery, the TCT, the common trunk of TCA, the suprascapular artery, and the inferior thyroid artery. The common trunk, close to the medial edge of the left anterior scalene muscle, continued to extend approximately 3 cm from the SCA and then divided into the TCA and the inferior thyroid artery (the inferior thyroid artery had already been removed by the students) (Fig. 1). The left anterior scalene muscle crossed under the TCA before being inserted into the first rib.

The left PN arose from ventral ramus of C3 to C5. The nerve fibres from C3 and C4 first descended passing the lateral side of the vagus nerve, and then crossed behind the TCA on the anterior scalene muscle. In contrast, the nerve fibres from C5 descended obliquely above the TCA toward the thorax. The fibres from the C3–C4 and C5 joined with each other, and then formed a loop around the TCA on the medial side of the anterior scalene muscle (Fig. 1). Therefore, the left PN may be greatly affected by the

movement of the neck and/or change in posture at the level of the anterior scalene muscle. In addition, TCA compressions can be one of the mechanisms of left PN injury, due to altered conditions that lead to changes in the course and calibre of the TCA occur, such as inflammation. After encircling the TCA, the left PN accompanied the internal thoracic artery and descended toward the thorax.

DISCUSSION

Diagnosis of PN injury requires great discretion due to non-specific signs and symptoms, including unexplained shortness of breath with exertion and/or supine position, increased fatigue, loss of energy, gastrointestinal reflux, bloating (left sided paralysis), and insomnia [6]. In unilateral diaphragmatic paralysis, the patient is often asymptomatic at rest and has dyspnoea only during exertion. In comparison, patients with bilateral paralysis always present shortness of breath [13]. However, mortality and morbidity associated with PN injury, and subsequent diaphragmatic paralysis, depend on the underlying causes and status of pulmonary function [12]

The motor fibres of the PN command the contraction of the diaphragmatic muscles and are controlled mainly by the C3–C5 segments of the neural tube. The axons of the right and left PNs descend along the vertebrae and pericardium, enter the diaphragm, and subdivide into several branches that innervate the respiratory muscles [20]. In the posterior neck, the cervical PN crosses obliquely to the anterior scalene muscle. It also traverses below the TCA and suprascapular artery, a branch of the TCT arising from the SCA [9].

Considerable variation of the standard anatomy of the PN in the neck has not been extensively described in the literature. Interestingly, frequent PN variation occurs in patients with supraclavicular decompression surgery for neurogenic thoracic outlet syndrome; however, there are few reports based on cadaver research [3]. In this report, anatomic variations of the PN were identified in 28 (28%) patients with duplicated PNs, accessory PN, and medially or laterally displaced PNs. Duplicated PN is defined as the same-sized medial and lateral contributions of the PN, regardless of its relationship to the anterior scalene muscle. Phrenic nerve duplication may originate in early stages of ontogenesis, as a result of alternative developmental pathways, in which axons of the PN, specific to a given domain of the diaphragm, running separately at a specific stage of development, instead of forming a single stem [20, 22]. In addition, accessory PN arising from superior trunk of the brachial plexus, ansa cervicalis, or nerve to subclavius was reported [15]. The accessory PN usually lies lateral to the PN and traverses the neck with a highly variable course [4]. This variable course of the accessory PN increases the complexity of the surgical procedure performed in the neck. Displacement of the PNs have also been observed, specifically with a completely medial or lateral course relative to the belly of the anterior scalene muscle. Topographical studies of the relationship between the PN and variation of the origin of the TCT demonstrate that lateral displacement of the PN results from the unusual origin of the TCT, which arises from the third portion of the SCA [9, 16]. Lischka et al. [14] reported that the TCT arose from the third portion of the SCA in 2 among 166 possibilities (83 cases, 2 sides each). In general, the TCT is formed by the fusion of the suprascapular artery, TCA, and the inferior thyroid artery, which originate from the first portion of the SCA. The aberrant origin of the TCT can lead to potential impairment of the PN. Hamada et al. [5] also reported that lateral displacement of the PN could increase the risk of PN injury during internal jugular or subclavian venous catheter placement and brachial plexus block. In addition, marked lateral deviation and subsequent strain of the PN in the neck could increase the risk of nerve injury or nerve dysfunction [16]. Pretterklieber et al. [18] described that left PN passed the diaphragm dorsal to the apex of the pericardium, though its passage is described as the oesophageal hiatus in many textbooks. In present case, left PN passed not the oesophageal hiatus but the diaphragm. Anatomical study about the PN was rare, therefore, its further study should be performed with larger cases.

Post-procedural and post-surgical nerve injuries may result from direct nerve transection, stretching, or may be secondary to the fibrosis and scarring that occurs after inflammation. Loss of normal tissue properties and adherence between normally separate anatomical structures can lead to compression and nerve dysfunction [9]. In this case, the nerve fibres from the C3 and C4 ventral rami transverse the anterior scalene muscle superiorly in the posterior neck, just under the prevertebral fascia, before joining the nerve fibres from C5. We postulated that trauma or manipulation in the neck, including neck surgery, may result in inflammation of the prevertebral fascia,

scalene muscle induration, and, in association with an intramuscular haematoma, can sometimes cause adhesion and compression of the ventral rami of the left PN where it is interposed between the muscles and the inflammatory semi-rigid prevertebral fascia.

This case also shows that nerve fibres from C3–C4 and C5 separately cross the TCA posteriorly and superiorly on the anterior scalene muscle forming a loop around the TCA. Kaufman et al. [9] reported 3 patients with diaphragm paralysis caused by TCA compression of the PN. They described that this artery normally originated from the TCT in the first portion of the SCA. They called this condition the 'Red Cross Syndrome' and reported that one in three patients suffered severe dyspnoea when turning their head to the affected side. In our case, as in the report by Kaufman et al. [9], the root of TCA arose from the TCT. We postulated that the potential risk of PN compression by the TCA is based on the movement of the neck or change in posture. In addition, the left PN and/or PN loop might be pulled more laterally by the altered course and calibre of the ipsilateral TCA. Thus, a vascular nerve compression may result in either an ischaemic or demyelinating neurapraxia. Vascular compression of a central and peripheral nerve has been well examined in various locations throughout the body. Examples of such events include the vertebral artery compression of a cervical root, cerebral arterial compression of the intracranial trigeminal nerve, vascular compression of the vestibulocochlear nerve, vascular compression of the occipital nerve causing migraine headaches, and radial nerve palsy secondary to a thickened recurrent radial artery [2, 7, 8]. In particular, the radial nerve palsy condition may occur in a similar manner to our case in that, prior trauma or inflammation may alter the calibre or course of the TCA and/or the spatial relationship between it and the involved PN.

Iatrogenic damage to the PN is most commonly due to cardiothoracic or neck surgery and is described after interscalene brachial plexus block and central vein catheterisation [17]. Other studies have shown that carotid-subclavian bypass grafting (CSBG) procedures were associated with PN injury, with levels of morbidity after iatrogenic injury varying across patient types [21]. Cohen et al. [1] showed that patients with chronic obstructive pulmonary disease (COPD) and PN injury after CSBG had significantly worse survival rates than those of patients with only COPD or PN injury undergoing the same procedures.

CONCLUSIONS

In the present article, we described a variant of the left PN, which can be compressed by the TCA depending on the movement of the neck or even changes in posture. Additionally, variants of the PN can increase the risk of iatrogenic injury during neck procedures, including surgery. Moreover, PN injury can have a substantial negative impact on a patient's quality of life compared to that in other groups. Based on the increasing use of invasive procedures, accurate knowledge of the anatomic variants of the PN is of considerable importance for surgeries and interventional vascular procedures. Therefore, we believe that this case report will be helpful for clinicians who perform these procedures.

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