Phrenic nerve palsy following coracoid infraclavicular brachial plexus block

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Various methods of infraclavicular brachial plexus block have been introduced in the past, of which Wilson’s coracoid infraclavicular brachial plexus block, a more lateral approach, consequently thought to be easier and safer. While only a few cases of transient ipsilateral phrenic nerve palsy after infraclavicular brachial plexus block have been reported, we describe a rare case of phrenic nerve palsy after Wilson’s coracoid infraclavicular brachial plexus block.

Key Words: Accessory phrenic nerve, Coracoid infraclavicular brachial block, Phrenic nerve palsy.

The choice of brachial plexus block (BPB) methods used for upper limb surgery depends on the type of surgery. Furthermore, to avoid the unwanted possibility of complications, the method of approach with less complication is usually chosen. Since interscalene BPB and supraclavicular BPB have a relatively high risk of pneumothorax and phrenic nerve palsy resulting in pulmonary dysfunction, various methods of infraclavicular BPB have been introduced in the past, of which Wilson’s coracoid infraclavicular BPB (CIB), a more lateral approach, consequently thought to be easier and safer [1].

While only a few cases of transient ipsilateral phrenic nerve palsy after infraclavicular BPB have been reported, we describe a rare case of phrenic nerve palsy after CIB.

CASE REPORT

A 46-year-old male (100 kg, 179 cm), classified as ASA physical status II, was scheduled for incision and drainage of a wound infected right hand from a prior third finger laceration. His past medical history was uneventful, with no known allergies, and no risk factors for pulmonary disease apart from obesity.

On arrival in the operating room, standard monitoring, including blood pressure (BP), electrocardiogram (ECG), and pulse oximetry (SpO2), was established, and 10 μg of sufentanil was administered intravenously (IV). The patient was placed in a supine position, his head turned to the left with his right arm resting at his side. Skin was prepped and draped after identifying the needle entry site approximately 2 cm medial and 2 cm caudal to the coracoid process described by Wilson [1]. An insulated nerve block needle (50-mm 22 gauge Stimuplex®, A. B. Braun, Germany) was directed posteriorly with nerve stimulation. The brachial plexus was localized at 4 cm depth with posterior cord stimulation (extension of the wrist) by gradually decreasing current intensity threshold from 1 mA to 0.4 mA (pulse width of 100 microseconds). After negative aspiration, 20 ml of 2% mepivacaine and 20 ml of 0.5% bupivacaine with epinephrine 1:200,000 was injected in 5 ml increments. After 10 minutes, sufficient surgical anesthesia was established and no remarkable event developed during surgery. The operation was successful and lasted for an hour.

Twenty minutes after arrival in the postanesthesia care unit (PACU), the patient suddenly complained of dyspnea and anxiety accompanied with high BP (200/90 mmHg) and tachycardia (110 bpm), nevertheless the SpO2 remained at 98%. 100% oxygen 6 L/min via facemask was applied and 2 mg of IV
midazolam was administered three times for sedation. The chest radiograph (CXR) obtained in the PACU immediately after this event revealed a right hemidiaphragmatic paralysis (Fig. 1). Approximately 30 minutes later the patient became asymptomatic from dyspnea and anxiety showing stable vital signs and was transferred to the ward. Five hours after the first injection of local anesthetics for CIB, the follow-up CXR showed improvement with a normalized diaphragm (Fig. 2) and the patient was relieved without further discomfort or complaint. The spiral chest computed tomogram which was taken after this incidence, did not show any evidence of pulmonary embolism.

**DISCUSSION**

Amongst many complications that can arise after BPB, phrenic nerve palsy, hoarseness and Horner’s syndrome are the more common events after interscalene BPB and supraclavicular BPB [2,3]. There have been only a few case reports of these complications occurring after infraclavicular BPB [4-6] and incidence are known to be rare [7] and even unannounced [8] when BPB is performed through Wilson’s CIB.

From comparing previous case reports we can deduce that there is a difference in the incidence of phrenic nerve palsy between interscalene BPB, supraclavicular BPB and infraclavicular BPB, and this may be the result of a septum, reported by Beck et al [9] (in abstract form), acting as a one-way valve dividing the neurovascular space of the brachial plexus into two compartments which can subsequently affect the distribution pattern of local anesthetics. There has also been a report that the distribution of local anesthetic solution is confined only to the infraclavicular space when given by way of CIB [10] and another study where no evidence of diaphragmatic dysfunction could be found after CIB [11]. Thus, we expect the reason of phrenic nerve palsy after CIB in this case to be the result of an anatomical variant.

The accessory phrenic nerve which runs caudally and joins the phrenic nerve distally may exist up to 48–75% as a normal variant and is divided into groups according to the site of origin; nerve to subclavius is most common (60.6%) followed by ansa cervicalis and nerve to sternohyoid [12-14]. And the existence of these accessory phrenic nerves is thought to be the cause of partial phrenic nerve palsy after supraclavicular BPB [12]. Noticeably, nearly 80% of the accessory phrenic nerves derived from the nerve to subclavius pass lateral to the external jugular vein [14], which may be the cause of diaphragmatic paralysis even when more lateral approaches of infraclavicular BPB are performed. Even though the drug distribution is supposed to be limited to the infraclavicular space after CIB, the possibility of unilateral diaphragmatic paralysis can be anticipated when the incidence rate and course of accessory phrenic nerve is put into consideration. The ultrasound-guided infraclavicular BPB, which is commonly known to be more manageable and safer [15], is not absolutely free from phrenic nerve palsy.
In this case, first, the delayed onset time of symptom after first CIB drug administration greatly decreases the possibility of systemic toxicity of local anesthetic agents to be the cause. Secondly, the spiral chest CT showed no abnormality, ruling out any chance of pneumothorax or pulmonary thromboembolism, and the ipsilateral hemidiaphragmatic paralysis was short lived as it was unapparent on the second follow-up CXR. The above facts along with the high incidence of a naturally present accessory phrenic nerve associated with the anatomical fact that this nerve runs lateral to the external jugular vein; we concluded this case to be the outcome of transient ipsilateral phrenic nerve palsy.

The delayed onset of dyspnea can be explained by quiet breathing, in which case the patient is relaxed in a supine position and without any forced movement or emotional change needs only to perform shallow breathing. When the patient starts movement and is positioned with an inclination of the upper body part, there is an increased need for deeper breathing, which made the ipsilateral hemidiaphragmatic paresis more obvious, leaving the patient gasping for air [2].

In conclusion, this case report demonstrates that phrenic nerve paralysis can develop following CIB, despite the generally accepted notion that CIB is safer due to a more lateral approach. A thorough knowledge of standard anatomy as well as anatomical variations of the accessory phrenic nerve and its course is essential for a safer brachial plexus block.

REFERENCES