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Original Article

Thoracotomy Paraplegia: A Case Report from the PACU

Tzu H. Huang

Advanced Practitioner, Perioperative Service, Post Anaesthesia Care Unit, The Canberra Hospital, ACT, Australia

ABSTRACT

The aim of this case report is to present the rare yet devastating complication of post-thoracotomy paraplegia and to raise awareness of the causative factors, as well as to discuss the nursing observation for prompt and effective clinical management in order to achieve successful healthcare outcomes. This case was secondary to the use of haemostatic agents during thoracotomy, and delayed post-operative identification of complication and management leading to long-term spinal cord impairment. Routine neurologic assessment focusing on movement and sensation of the extremities is recommended post-operatively to detect the potential complications as early as possible.

Key words: neurologic deficits; paraplegia; post-thoracotomy complication

INTRODUCTION

This case study represents paraplegia of a 52-year-old male, secondary to the use of haemostatic agents during thoracotomy and the delays in post-operative identification of the complication as well as management leading to permanent spinal cord impairment. The aim of this case report is to present the rare yet devastating complication of post-thoracotomy paraplegia and to raise awareness of the causative factors, as well as to discuss the nursing assessment for prompt and effective clinical management and achieve successful healthcare outcomes. Numerous post-thoracotomy paraplegia cases have been reported worldwide for previous decades in the surgical fields; nevertheless, very few nursing articles have addressed this post-operative complication. To continue improvement in the quality of immediate post-operative nursing care in the healthcare system, lessons need to be learned and the errors need to be prevented.

Correspondence to: T. H. Huang, Post-anaesthesia Care Unit, The Canberra Hospital, Yamba Drive, Garran ACT 2605, Australia. Tel: 0433 645 384; Fax: 02-6282 6984; E-mail: hweiche@hotmail.com

Paraplegia post-thoracotomy from an aortic operation is relatively well recognised, resulting from insufficiency of arterial flow of the spinal cord by either aortic cross-clamping or post-ligation of the posterior intercostal arteries [Attar *et al*, 1995; Dogan *et al*, 2005; Kocogullari *et al*, 2008]. The incidence of paraplegia had ranged from 6% to as high as 40% in those aortic cases [Livesay *et al*, 1985; Crawford *et al*, 1986]. The catastrophic incidence of post-thoracotomy paraplegia, however, is very uncommon and is estimated approximately at 0.08% [Attar *et al*, 1995]. Essentially, these post-thoracotomy paraplegia cases are generally caused by the compromised arterial blood circulation of the spinal cord due to the compression of the haemostatic material (oxidised cellulose).

A case report in the post-anaesthesia care unit (PACU)

A 52-year-old male was admitted to the PACU at 11:35 in the morning following a right-sided thoracotomy. General anaesthesia with one lung ventilation technique was adopted intraoperatively and no epidural anaesthesia was involved. The patient was haemodynamically stable in the PACU with adequate post-operative

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pain management by way of Fentanyl PCA (patient control analgesia) and was sitting up at 35 degrees to improve expansion of the lungs. The general PACU observations, chest drains and pain assessments were routinely conducted by the recovery nurse. At 12:40 in the afternoon, the patient asked the recovery nurse why it was that he was unable to feel as well as move both his legs.

The recovery nurse therefore performed a neurovascular assessment which indicated no muscle tone or sensation to both lower extremities and very weak muscle tone for both upper extremities. The relevant anaesthetist was informed, who came to assess the patient and then contacted the thoracic surgeons who reviewed the patient. An urgent magnetic resonance imaging (MRI) was required, indicating that a small 'spontaneous haematoma' was causing the significant spinal cord compression at the T5 level. The patient urgently required an emergency laminectomy to remove the mass compressing the spinal cord, returning to the operating theatre at 17:00 in the afternoon. The decompression surgery removed the haematoma that was in fact a coin-sized haemostatic material. The patient was extubated and transferred to a high dependency unit for close monitoring post-operatively. Despite surgery, the patient was still unable to move and sensate from the T5 dermatome downwards after 72 hours post-operatively and was diagnosed with paraplegia secondary to the use of oxidised cellulose.

DISCUSSION

Several factors contributed to this devastating post-operative complication, including large blood loss, hypotension, thrombosis, epidural haematoma and haemostatic agents, which will be discussed in this case report.

Since it was first introduced in 1942, oxidised cellulose (Surgicel Fibrillar® and Surgicel Nu-Knit®; Ethicon, Johnson & Johnson) has been commonly utilised during surgical procedures aiming to pack firmly against the bleeding until haemostasis [Achneck *et al*, 2010]. Owing to the oxidised cellulose's characteristic factors including easy application and good handling, they have been applied to neurosurgery. Moreover, oxidised cellulose is a low PH material with antimicrobial benefit. In terms of the antimicrobial effect, various pathogenic organisms would resist it because of this low PH material. The main mechanism of oxidised cellulose is to generate a small artificial clot for the process of haemostasis. However, oxidised cellulose with natural low PH could cause haemolysis leading to leakage of haemoglobin that, interacting with acid, will form acid haematin. The acidic reactions would enhance the risks of delay in wound healing resulting from inflammation of surrounding tissues [John *et al*, 2006; Achneck *et al*, 2010].

Most of the current published cases, bleeding during surgery, were managed by holding sponges, diathermy or suture ligation. The application of oxidised cellulose was therefore carefully packed, pushed and then left to secure the haemostasis [Attar *et al*, 1995; Brodbelt *et al*, 2002; Dogan *et al*, 2005; Henry *et al*, 2005; John *et al*, 2006; Achneck *et al*, 2010]. The dissolution of oxidised cellulose is generally variable between 2 and 6 weeks depending on the amount used intraoperatively [Achneck *et al*, 2010]. Importantly, oxidised cellulose with saturated blood can swell and form into a small clot causing the possibility of directed local compression. Although the paraplegia resulted from misplacement of the haemostatic material through the spinal foramen during the operation, the clinical signs and symptoms would be presented immediately in the post-anaesthetic phase. Yet some reported cases have showed delayed onset of symptoms, even presenting a few days post-operatively, due to migration of oxidised cellulose gradually into the epidural space through the intervertebral foramina causing spinal cord compression.

Owing to the relative pressure gradient between the two spaces, the intervertebral foramen and the spinal canal, by pulling up the ribs during closure of thoracotomy, the haemostatic material could subsequently migrate into the spinal canal. Moreover, the respiratory movement of the rib cage may also produce the external force pushing the haemostatic material into the spinal canal [Attar *et al*, 1995; Brodbelt *et al*, 2002; Henry *et al*, 2005; Kumar *et al*, 2009]. A literature review had analysed the common denominator that the more posterior the thoracotomy incision towards the vertebrae, the greater the risks of spinal cord compression [Attar *et al*, 1995].

Despite the manufacturer's recommendation of removing the material after the bleeding is controlled, it is commonly left in place due to its 'absorbed' character. The utilisation of oxidised cellulose for haemorrhage spots in the posterior angel of posterolateral thoracotomy should be approached in a cautionary manner to minimise the chance of spinal cord compression.

Nursing assessment and intervention in PACU

The neurological observation is a pivotal nursing assessment to uncover the potential and to detect early warning

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neurological deficits, which can lead to temporary and even permanent neurological impairment.

The critical and common manifestations for the progression in intra-spinal compression have clinically presented rapid or delayed neurologic deficits [Attar *et al*, 1995; Brodbelt *et al*, 2002; Henry *et al*, 2005; John *et al*, 2006; Kumar *et al*, 2009]. Owing to direct compression of the spinal cord by the foreign body, axonal disruption occurs following secondary vascular compromised and vasogenic oedema by releasing serotonin, prostaglandin E2 and interleukin-1. Consequently, both arterial and venous blood flow to the spinal cord would be significantly reduced by high pressure from the swelling within a short period of time. Moreover, the process of demyelination damage causes a blockage in nerve impulse transmission along the ascending and descending spinal cord tracts [McDonald and Sadowsky, 2002; Mavrogenis *et al*, 2009].

Early identification of the signs and symptoms of spinal cord compression plays an imperative role for acute care practice nurses. Initial clinical presentations will commonly show increasing partial or complete sensory loss, worsening of weakness of muscle tone, paralysis of the lower limbs and severe localised back pain or radicular pain from the dermatome. Importantly, the neurologic motor weakness, including heavy limbs and stiff extremities, is more aggressive than the pain of the compression area. Continuous spinal cord compression will subsequently result in motor loss. Furthermore, decreased sensation below the level of compression normally begins from the distal segment of the extremities and ascends to the centre of the body. Numbness as well as a 'tingling' in the extremities is a prevalent early sign as well as a lack of temperature. Pinprick sensation will eventually develop [Marrs, 2006; Pellatt, 2008; Kaplan, 2009]. The physical neurologic observation for post-thoracotomy patients is therefore focused on the muscle strength assessment for weakness of the upper and lower extremities, and on sensory evaluation (dermatomes) for the identification of loss of sensation on a specific spinal level by both sharp and dull responses.

Post-operative neurological assessment of patients undergoing thoracotomy without epidural or spinal anaesthesia is not often performed in the acute care setting. Considering the catastrophic spinal cord compression, clinical practice nurses may need to raise the alarm and perform brief motor as well as sensory assessment as baseline information in order to detect early possible signs and symptoms of spinal cord compression with this surgery.

With clinical presentations of neurologic deficit, MRI scans should be used to detect the mass and locate the level of spinal cord compression. MRI is the gold standard choice for those cases; timely management with other healthcare professionals is the key to save the compromised spinal cord from such a rare complication [Attar *et al*, 1995; Brodbelt *et al*, 2002; Dogan *et al*, 2005; Henry *et al*, 2005; John *et al*, 2006; Kumar *et al*, 2009].

CONCLUSION

Learning from past experiences, an increase in awareness, early detection and management is the cornerstone to preserve the function of the spinal cord. Either accidental misplacement or migration of the haemostatic agent can occur to compress the spinal cord. In this complicated and challenging healthcare environment to date, clinical practice nurses are acclimatised to utilising critical thinking on a daily basis, assessing post-operative patients effectively, responding and communicating with responsible medical teams to act promptly in order to deliver a high standard of professional nursing care.

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