Acute Brown-Séquard syndrome following brachial plexus avulsion injury. A report of two cases

臂叢神經撕脫損傷後的急性布朗－塞卡爾綜合徵。兩個臨床病例報告

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Introduction: Brown-Séquard syndrome is an unusual sequelae of pre-ganglionic brachial plexus injury. There have been sporadic case reports indicating that the cause of this condition is due to tethering of the cord, vascular ischaemia or direct avulsion injury of the nerve roots. This is a report of two patients with complete pre-ganglionic brachial plexus avulsion injuries who developed acute partial Brown-Séquard syndrome due to haematoma. ***Cases:*** Two patients developed acute partial Brown-Séquard syndrome associated with complete pre-ganglionic brachial plexus avulsion injuries. In the first case the neurology recovered fully after the evacuation of the large subdural hematoma. Whereas, in the second case the neurology only recovered after 4 weeks closed observation in view of the compression was due to small epidural haematoma. ***Conclusion:*** Acute Brown-Séquard syndrome occurring in association brachial plexus injury should be investigated with urgent magnetic resonance imaging to exclude any reversible haematoma compression. (Hong Kong j.emerg. med. 2011;18:347-351)

引言：布朗－塞卡爾綜合徵是節前臂叢神經損傷的不尋常後遺症。已經有零星的病例報告，指出造成這種情況的原因，可以是栓繫的脊髓，血管缺血或神經根直接撕脫傷。這兩例報告的是完全節前臂叢撕脫傷後，由於血腫導致急性布朗－塞卡爾綜合徵。**病例：**二名完全節前臂叢撕脫傷的患者，同樣有急性布朗－塞卡爾綜合徵。在第一個案例，清除大塊硬膜下血腫後，患者的神經完全康復。而在第二個案例，由於加壓的是小塊硬膜外血腫，經四週緊密觀察後患者的神經才恢復。**結論：**急性布朗－塞卡爾綜合徵與臂叢神經損傷同時發生下，要緊急使用磁共振成像技術來排除任何導致可逆性擠壓的血腫。

Keywords: Brown-Séquard paralysis, haematoma, hemispinal cord syndrome, paraplegia, radiculopathies

關鍵詞：布朗－塞卡爾麻痺、血腫、半橫斷脊髓綜合徵、截癱、神經根病變

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Introduction

Brachial plexus avulsion injuries commonly occur during motor vehicle or motorcycle crashes. During the collision, the arm and shoulder is severely stretched and results in avulsion of the nerve roots out of the cervical spinal cord. Brown-Séquard syndrome occurs as a result of disruption to the function of half the

spinal cord. It can be resulted from a variety of causes; direct penetrating injury or compression secondary to intra or extramedullary lesions. In complete Brown-Séquard syndrome, this condition manifests as an ipsilateral upper motor neuron lesion below the level of the insult with abnormal sensation to vibration and proprioception and contralateral abnormal sensation to pain and temperature.

Brown-Séquard syndrome is an unusual sequelae of pre-ganglionic brachial plexus injury. There have been sporadic case reports describing this condition occurring in association with brachial plexus avulsion injury. However, most of these reports were due to tethering of the cord, vascular insufficiency / ischaemia or direct avulsion injury of the nerve roots.[1-3] We report two patients who had developed acute partial Brown-Séquard syndrome following pre-ganglionic brachial plexus avulsion injuries, secondary to subdural and epidural haematoma respectively.

Case 1

An 18-year-old male was involved in motorcycle accident in 2009. His motorbike skidded and he fell over and landed on his right side with the neck tilted to his left. He presented with severe pain over the neck and shoulder with abrasion over his right shoulder region. He was presented with inability to move his right upper limb.

On examination the right upper limb myotomes demonstrated a Medical Research Council (MRC) power of 0 from C5 to T1. There was also complete loss of sensation over the C5 to T1 dermatomes with absence of reflexes over the biceps, brachioradialis and triceps. A right Bernard-Horner syndrome was present. There was absence of the right radial pulse with a capillary refill of more than 2 seconds.

Computed tomography angiography of the right upper limb showed there was injury to the third part of subclavian artery with intact collateral flows. The subclavian injury was managed conservatively in view of a good collateral perfusion. The diagnosis of a complete pre-ganglionic injury of the right brachial plexus with injury to the third part of the subclavian artery was made.

On the third day of admission, the patient developed acute weakness of the right lower limb. On further assessment, there was decreased power from L2 to S1 myotomes which demonstrated MRC power of 3 to 4. There was diminished sensation to pain and temperature on the contralateral side of the limb. However the reflexes, fine touch, vibration and proprioception sensations were preserved in both lower limbs. An urgent Magnetic Resonance Imaging scan of the cervical spine showed a huge collection of subdural haematoma over the right side extending from C5 to C7 levels resulting significant spinal cord compression. Extensive cervical cord oedema was also noted extending from C2 to C7 region (Figure 1). The patient was diagnosed to have a partial Brown-Séquard syndrome due to subdural haematoma secondary to right brachial plexus avulsion injury.

A hemilaminectomy of C6 and evacuation of subdural haematoma was performed (Figure 2). His neurology of both lower limbs recovered back to normal on the next day.

Case 2

A 17-year-old young lady was involved in a motor vehicle accident in 2010. She fell from the motorbike and landed over her right shoulder. She presented with complete weakness and loss of sensation of her right upper limb. Radiological investigations revealed that she had fracture of the spinous process of C7 and fracture of the transverse process of T1, fracture of the right first rib with pneumothorax and bilateral lung contusion. Bilateral chest tubes were inserted. Computed Tomography scan showed presence of traumatic brain injury with right frontal lobe injury noted with multiple facial bones fractures i.e. right mastoid, zygomatic arch and occipital bone. She was presented with a decreased Glasgow Coma Scale was intubated electively and later admitted to intensive care unit for the first 3 days.

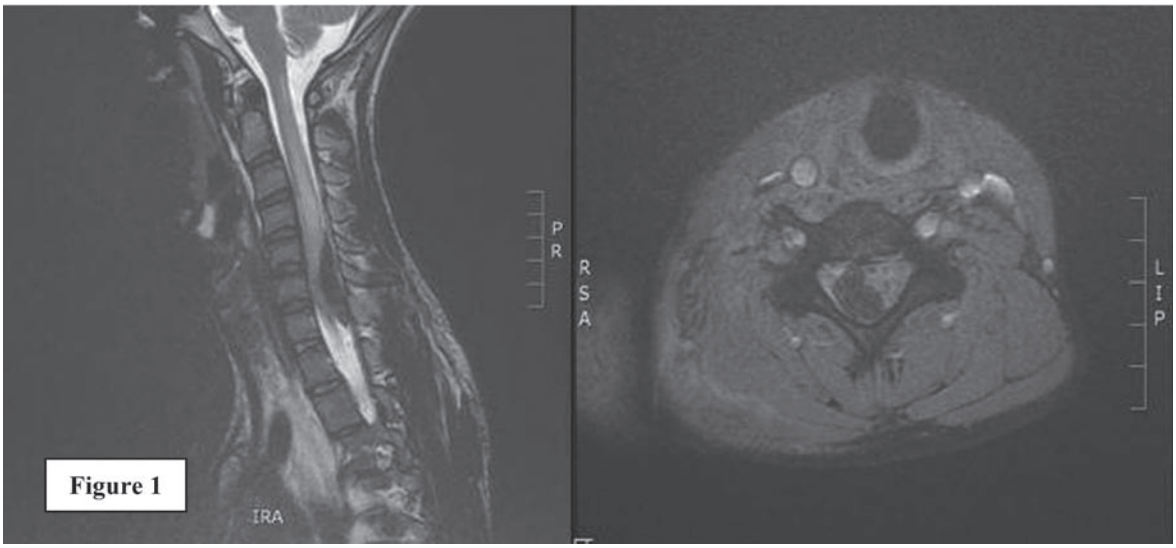

Figure 1. MRI scan showed a subdural haematoma collection extending from C5 to C7 which compressed the spinal cord. The spinal cord was shifted to the left side.

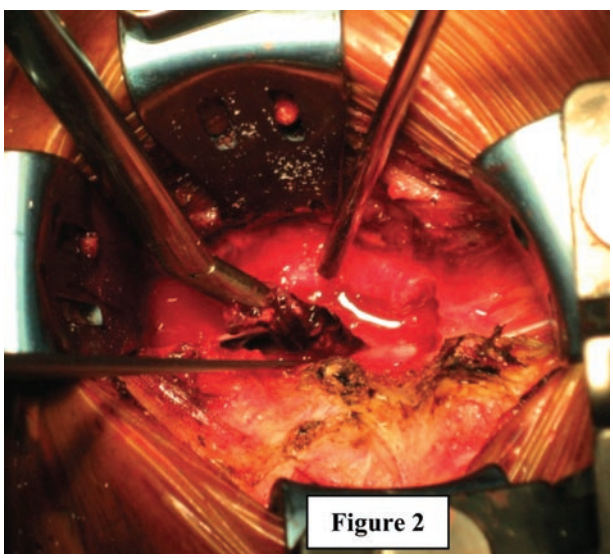

Figure 2. The evacuation of the subdural haematoma after a hemilaminectomy and a durotomy was performed over the right C6 level.

After extubation and discharge from the intensive care unit, she was noted to have a complete paralysis of the right upper limb with absence of sensation of her right C5 to T1 dermatomes. A Bernard-Horner sign was present on the right side however her vascular status was normal. The myotomes of the right lower limb demonstrated a MRC power of 4. There was also diminished sensation to pinprick and temperature over left from T7 downward. Sensation over the right side of the body was normal. Cervical MRI showed avulsion injury of right C7, C8 and T1 nerve roots with extensive prevertebral and extradural haematoma seen on the right side extending from C6 to T1 levels causing deviation of the spinal cord to the left.

As a result of no marked compression of her cervical spinal cord, she was treated conservatively with Aspen neck collar with closed neurological monitoring. Her neurological status did not deteriorate further and slowly improved within the next four weeks.

Discussion

The Brown-Séquard syndrome is one of the partial cord injuries. It usually occurs as a result of direct penetrating cord injuries or compression secondary to extramedullary or intramedullary lesions. This syndrome is usually diagnosed clinically by demonstrating ipsilateral weakness with loss of proprioception and vibration sense coupled with contralateral reduction in pain and temperature sense. The clinical manifestations can be varied and could be either complete or partial.

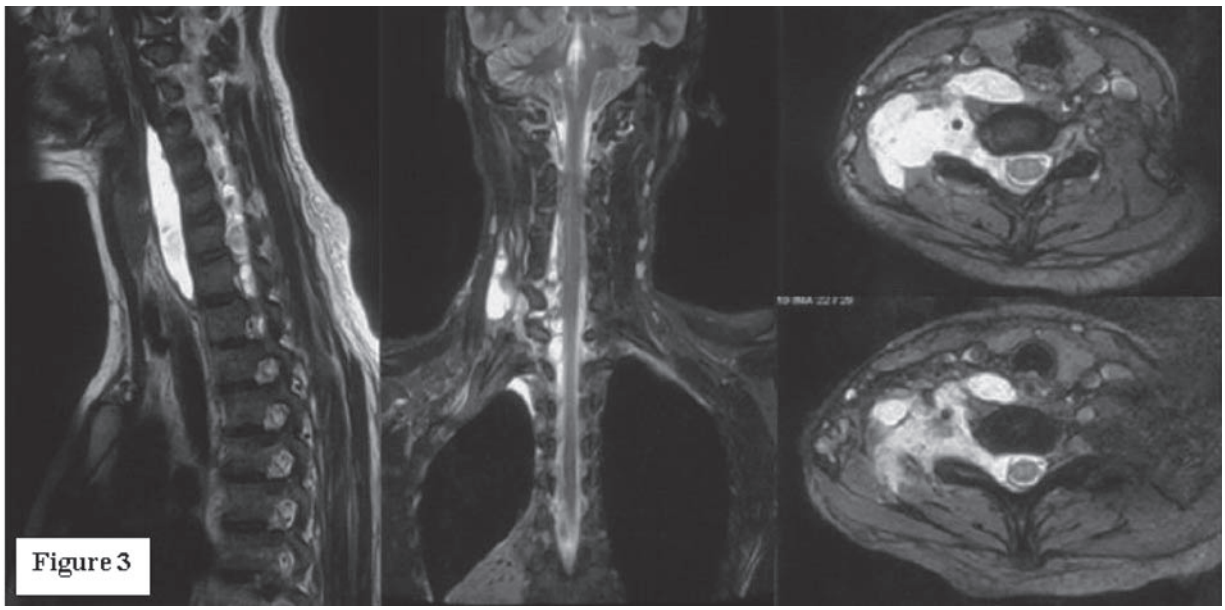

Figure 3. MRI scan showed a haematoma collection extending from C6 to T1 levels which compressed the spinal cord and resulting the spinal cord to be shifted to the left side. There was also presence of prevertebral haematoma noted.

This condition is unusual following a brachial plexus nerve root avulsion. One of the earliest reports of Brown-Séquard syndrome associated with avulsion type brachial plexus injury was by Penfield.[4] He described a delayed presentation of this syndrome which was due to severe traction of the cord into the intervertebral foramen following avulsion of the roots of the brachial plexus. Subsequently there were also other reports of Brown-Séquard syndrome associated with pre-ganglionic brachial plexus injuries. In a report of three cases by Nordin et al, they elucidated the cause of cord injury in these cases. Two of the cases occurred late after the acute brachial plexus injury. The first case presented six years after the acute brachial plexus injury with progressive symptoms and signs suggesting Brown-Séquard syndrome. Magnetic Resonance Imaging demonstrated tethering of the cord to the site of brachial plexus injury. The second case also had a delayed presentation and was attributed to a vascular ischaemic event due to the presence of haemosiderin on the magnetic resonance imaging. The third case which was described occurred acutely after the brachial plexus injury and was attributed to direct avulsion injury to the spinal cord which cause cord ischaemia due to concomitant avulsion of the radicular vessels.[1] This type of presentation (avulsion of the spinal cord) has also been described by Stephen et al.[2]

Our reported cases demonstrated several unique features. In the first case, the patient had avulsion of the right C6, C7 nerve roots and the MRI finding was consistent with a subdural haematoma formation that caused compression on the spinal cord from the right side. Prompt recovery of the power and sensation post-operatively confirmed that the development of the partial Brown-Séquard syndrome was due to the direct effect of haematoma compression rather than due to vascular event. In the second case, the patient developed partial Brown-Séquard syndrome due to the avulsion of the right C7, C8 and T1 nerve roots causing an extradural haematoma compressing on the spinal cord. This could be due to disruption and bleeding from the radicular vessels which later led to the formation of the epidural haematoma.[5] In this case, the extradural haematoma compression with the associated neurological deficit was mild, therefore conservative treatment was instituted and the patient neurology improved after four weeks.

The importance of the recognition of the two above cases lies in the reversibility of the neurological deficit if the condition was recognised early. A magnetic resonance imaging of the cervical spine needs to be performed promptly once the syndrome is clinically diagnosed. If any acute compression of the cord is demonstrated and the neurological status is worsening, urgent evacuation of the haematoma can be performed. If the haematoma is mild with no significant cord compression, a conservative treatment can be instituted.

Conclusion

Acute Brown-Séquard syndrome which is associated with avulsion type brachial plexus injury should be investigated with urgent magnetic resonance imaging of the cervical spine to exclude reversible compression of the spinal cord due to haematoma formation. Cord injury secondary to avulsion injury / ischaemic event of the cord should not be assumed.

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