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CASE REPORTS

Peripheral neuropathy secondary to traction injury resulting from the use of physical restraints

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Abstract

We describe the case of a 46 year-old woman who developed weakness of the upper limbs while admitted for relapsed chronic schizophrenia. She was combative, necessitating the use of physical restraints in the form of wrist bands that were used to secure her to the bed frame. In addition, she was treated with haloperidol and benzhexol. Her physical findings were consistent with a lower motor neuron injury confined to the upper limbs and a nerve conduction study confirmed a mixed demyelinating axonal injury resulting in a predominantly motor neuropathy. She made an almost complete recovery by 6 months. We suggest that her condition was due to neuropraxia secondary to traction injury affecting the brachial plexus as an indirect consequence of the use of physical restraints.

INTRODUCTION

Restraint is defined as "a method or device used to restrict a person's freedom of movement, physical activity, or normal access to their body."[1] A 'physical restraint' suggests limiting movement by manually restricting movements for short periods or by using devices for longer period while 'chemical restraint' denotes the use of pharmacological agents to subdue patients.

Focal peripheral neuropathies are commonly seen in clinical practice and may be classified as neuropraxia, axontmesis or neurotmesis. Axonotmesis refers to injury of the peripheral nerve in which the axon is divided but the epineurium remains intact, allowing regeneration of tissue.[2] Clinically this presents as peripheral neuropathy with motor and sensory deficits, while the nerve conduction study shows delayed latency in motor and sensory modalities with reduced motor amplitudes. Axontmesis represents a mid-point in the spectrum of peripheral neuropathy, with neuropraxia and neurotmesis at either end. Neuropraxia refers to segmental or focal demyelination with preservation of the axon while neurotmesis describes complete division of the nerve. Complete recovery is expected in the former while permanent loss of function is associated with the latter. We present a patient with schizophrenia who developed brachial plexus traction injury secondary to physical restrain.

CASE REPORT

A 46-year-old lady was admitted to a district hospital with week-long history of auditory hallucinations, paranoid delusions, agitation and increasing violence. Her stay in the District Hospital was marked by aggression and she had to be restrained physically. Using fabric cuffs, her upper limbs were bound at the wrists and she was tied down to the bed frame. She was also prescribed haloperidol and benzhexol but nothing else. She was discharged at her request and was brought to our hospital a day later. She was admitted to the Psychiatry ward where she was again restrained as described due to persistent psychiatric symptoms and aggression. Upon release of her restraints a day later she was noted to have weakness of the upper limbs.

Based on a telephone conversation with the Medical Officer at the District Hospital, we confirmed that she was restrained continuously for 5 days and was noted to have weakness of the upper limbs on discharge. Her arms hung to her sides and she was unable to use her them to carry out daily activities, but she could walk without assistance.

On examination, both upper limbs were hypotonic with power of 3/5 affecting both extensors and flexors, with absent deep tendon reflexes consistent with a lower motor neuron injury. Sensation to pin-prick was relatively intact

based on observed withdrawal of the limb rather than subjective testing, as the patient was not co-operative. Muscle tone, power and tendon reflexes of the lower limbs were grossly normal and the plantar response was flexor. While examining the patient, we noted that she would repeatedly sit up in and strain forward causing her upper limbs to extend backwards in internal rotation.

Her blood investigations were normal and these included blood counts, renal and liver function, serum B12 and folate levels, thyroid function, connective tissue screen and VDRL. An MRI of the cervical spine was normal.

A review of her medication revealed no identifiable cause of neuropathy. She did not use recreational drugs or traditional medication nor consume alcohol.

A nerve conduction study (NCS) of the upper limbs revealed a mixed demyelinating and axonal injury with predominantly motor neuropathy affecting the median and ulna nerves bilaterally. The lower limbs returned a normal study. (Table 1). A needle electromyography was attempted but abandoned due to non-cooperation.

The patient was discharged following a two week admission with no change in the condition of her upper limbs. She but did not attend her scheduled out-patient appointment but we were able to confirm over the phone that she had recovered full use of her arms over 9 months.

Table 1: Nerve conduction studies of the patient

Nerve/Site	Latency ms	Amp Pk-Pk Mv	Dur ms	Area mVms	Amp %	Lat diff	Velocity m/s
L Median (APB)							
1. Wrist	4.60	0.2	4.45	0.2	100	4.6	
R Median (APB)							
1. Wrist	3.30	7.6	5.40	19.1	100	3.30	59.7
2. Elbow	7.15	7.4	5.60	18.0	99.4	3.85	73.7
3. Axilla	8.10	6.9	5.40	15.7	93.8	0.95	56.2
4. EP	12.55	1.3	5.75	2.5	13.9	4.45	59.5
L Ulnar (ADM)							
1. Wrist	5.80	0.2	3.75	0.3	100	5.80	55.4
2. Below elbow	9.95	0.2	2.85	0.2	79.3	4.15	
3. Above elbow							
4. Axilla							
5. EP							
R Ulnar (ADM)							
1. Wrist	2.40	8.4	6.10	22.0	100	2.40	
2. Below elbow	5.95	9.9	6.10	23.3	117	3.55	63.4
3. Above elbow	7.50	8.2	5.95	18.2	94.5	1.55	64.5
4. Axilla	8.25	8.4	6.10	19.3	98.8	0.75	80.0
5. EP	11.45	5.1	6.20	13.1	65.8	3.20	78.1

The findings are consistent with a mixed injury involving the axon and myelin sheath more prominent on the left than the right as suggested by the missing values that were unobtainable. The lower limb and sensory studies were normal .
APB: abductor pollicis brevis
EP: Erb's point
ADM: abductor digit minimi

DISCUSSION

Our patient was likely to develop her weakness during the initial hospital admission and her physical findings were consistent with lower motor neuron pathology secondary to nerve injury. Based on her history, negative blood investigations and imaging studies, mechanical injury was the most likely cause. The nerve conduction study confirmed a mixed motor and sensory deficiency and her self-reported lack of persistent neurological deficit currently suggests a neuropraxic injury.

Acute intermittent prophyria (AIP) was considered in our patient due to the pathognomonic combination of episodic psychiatric symptoms and neuropathy. While it is an important differential to exclude, AIP is unlikely considering the duration of her psychiatric symptoms and the absence of neuropathy or any other non-psychiatric symptom in the patient's history, although AIP might present in a variety of ways.[3,4]

We also considered the possibility of chronic inflammatory polyneuropathy (CIP) secondary to a systemic inflammatory response (SIRS) [5] seen in patients with prolonged hospital stay. However, the patient was not sufficiently ill and the condition preferentially affected her upper limbs making CIP unlikely.

We suggest she suffered a traction type injury to the brachial plexus bilaterally as a result of habitually straining forward while restrained at the wrist as we described. The manner in which the physical restraints were secured to the bed-frame with the elbows flexed allowed her to do this. Her upper limbs would have been forced into a position of extension and internal rotation at the shoulder exerting traction on the plexus. The injury might have been prevented if her elbows were fully extended before being secured or if such a means of restraint had not been necessary in the first place.

Traction injury is a mechanical cause of neuropathy and is more commonly associated with trauma sustained in MVA or industrial accidents. Whilst there were many examples of brachial plexopathy due to other causes and similarly, numerous reports of injury and even death in psychiatry patients who had been physically restrained, there were no reported cases suggesting a causal link between brachial plexopathy and physical restraints.

The use of restraints in medical practice is seen as a necessary evil and violent, disruptive patients lower the threshold of medical personnel in favour of restraint. Popular culture might equate restraint with a straight-jacket but the reality is that most patients are instead secured to bed frames at their ankles, wrists or both. Media scrutiny has lead to investigation of the use of restraints and a Congressional investigation confirmed the risks associated with their use.[6] The increased public attention has given rise to guidelines governing use of physical or chemical restraints for various groups such as psychiatric patients with the main aim of reducing their usage. The alternative use of chemical restraints and use behavioural therapy have been encouraging.[7]

Malaysian data documenting the use of medical restraint and risk of injury is lacking based on our research of the literature. This is probably a result of under-reporting. There are neither national consensus statements nor clinical practice guidelines governing the use of restraints in hospitals although individual hospitals especially the major psychiatric institutions would probably have their own.

Our objective in reporting this case is to increase awareness of possible injuries that might result from the use of physical restraints and to encourage exploration of alternative ways of managing disruptive patients.

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