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Incidence and predictors of early ankle contracture in adults with acquired brain injury

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Abstract

Objective: To determine the incidence and predictors of early ankle contracture in adults with acquired brain injury. *Methods:* A prospective cohort study of patients admitted to Neurosurgical Intensive Care Unit (NICU), University Malaya Medical Centre and referred for rehabilitation within a period of 12 months. Adult patients with newly diagnosed acquired brain injury with no prior deformity to lower limbs, Glasgow Coma Scale ≤ 12, no concomitant spinal or lower limb injuries, medical stability at inclusion into the study and agreed to participate for the total duration of assessment (3 months) were recruited. We conducted weekly review of ankle muscle tone and measurement of ankle maximum passive dorsiflexion motion. The end point is reached if ankle contracture developed or completed 3 months post injury assessment. *Results:* The cohort included 70 patients, of which only 46 patients completed the study. Twenty-eight patients suffered from severe brain injury whilst 18 from moderate brain injury. Out of the 46 patients, 13 (28%) developed ankle contracture at the end of the study period. Abnormal motor pattern was significantly associated with incidence of ankle contracture, which included spasticity ($p<0.001$), spastic dystonia ($p=0.001$) and clonus ($p=0.015$). Using univariate analysis, the predictors for ankle contracture were spasticity (OR 51.67, CI 7.53-354.52, $p<0.001$), spastic dystonia (OR 27.43 CI 2.84-265.35, $p=0.004$) and clonus (OR 4.18 CI 1.33-13.19, $p=0.015$).
Conclusion: Abnormal motor patterns are strongly associated with early incidence of ankle contracture amongst adult with new diagnosis of moderate to severe acquired brain injury despite a regular standard therapy program. This is an important clinical finding towards early prevention of ankle contracture.

INTRODUCTION

Ankle contracture is a common musculoskeletal complication of moderate to severe acquired brain injury (ABI) in adult, particularly related to traumatic or spontaneous diffuse subarachnoid or intracerebral haemorrhage.[1,2] It has been described to range from abnormal posturing of the foot and ankle with plantarflexor and/or invertor muscle overactivity during movement, to fixed joint contracture.[3] Thus, the term ankle contracture and equinovarus deformity has been used interchangeably in the literature as the presentations tend to co-exist.[3-5]

The mechanisms that cause joint contracture following brain injury have been discussed in the literature. During immobility, muscles are in a shortened position, which causes muscle unloading. This is followed by muscle atrophy, muscle shortening and accumulation of connective tissues to muscle fibres of the limbs, which leads to the first mechanism of muscle contracture.[5,6] The second mechanism that participates in contracture formation is muscle over activity, which results from loss of the central motor system's ability to synchronize and generate force from a muscle and to relax the muscle at rest.[5,6] In short, sustained abnormal posturing with associated muscle imbalance combined with muscle over activity can cause irreversible joint malalignment that leads to joint contracture.[5]

The pathological changes described above tend to occur rapidly following brain injury, as early as six hours of immobilization and progress throughout the following weeks.[5] Therefore, joint contractures were reported early following acute hospitalization in previous studies, as early as between one and 16 weeks after brain injury.[3] Clavet *et al.*[7] published a study looking at the

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incidence of joint contractures in Intensive Care Unit (ICU) setting, and reported that 39% of the patients developed joint contractures within their stay of at least 2 weeks in the ICU. From all the joint contractures noted, 51% were of ankle joints.

The type of lower limb motor pattern, severity of injury, length of hospital stay as well as duration of mechanical ventilation are among the factors predicting the development of ankle contracture.[1,3,8] One study found that dystonic muscle overactivity correlates closely with ankle contracture and severity of brain injury has a weak association with the development of ankle contracture.[3] Yarkony and Sahgal on the other hand, found that the incidence of multiple joint contractures amongst craniocerebral trauma patients was highest with duration of coma of more than three weeks.[1] Childers *et al*.[8] found that low Glasgow Coma Scale (GCS) on admission to the rehabilitation ward, muscle weakness, hyperreflexia and pelvic fractures as significant predictors for joint contractures in traumatic brain injured patients, whereas Clavet *et al*.[7] found that ICU stay of more than eight weeks was a significant risk factor for contracture.

Studies related to ankle contracture after brain injuries were mostly carried during the subacute stage and patients were recruited in a rehabilitation unit.[3,8,9,10] Presence of ankle contracture at this stage might delay patient's ability to achieve independent ambulation and shifted the therapy focus to various interventions to correct ankle contracture instead, such as the use of Botulinum toxin.[11] The aim of our study is to determine the incidence of early ankle contracture in adults with moderate to severe acquired brain injury despite early standardized physiotherapy and to determine the predictors of the deformity. Understanding the factors predicting early ankle contracture after brain injury is useful to plan appropriate preventive measures during the acute care.

METHODS

This is a prospective cohort study over a 12-month period from August 2010 until August 2011, at the Neurosurgical Intensive Care Unit (NICU), University Malaya Medical Centre (UMMC). Patients with ABI; comprised of traumatic brain injury (TBI) and haemorrhagic stroke admitted to NICU and referred for neurorehabilitation were selected for our study.

Neurosurgical Intensive Care Set-up

The NICU has 18 beds with 11 mechanical ventilators. Within the unit, there are two sections dividing the mechanically ventilated and non-mechanically ventilated patients but nonetheless requiring close medical monitoring. Once patients are deemed medically stable, they are transferred to a general surgical ward for further management. The admission criteria to the NICU are best illustrated in Figure 1.

Most of the patients are referred for rehabilitation while they are still in the NICU. The main aspect of rehabilitation care at this point involves physiotherapy, mainly to maintain muscle tone and muscle strength, joint protection care, pressure ulcer prevention and chest physiotherapy. The physiotherapist in charge would perform daily review and bedside interventions followed by once or twice weekly review by the rehabilitation physician. The regime varies slightly among the patients based on their clinical conditions but in general, the standard practice in NICU at our centre included: i) a one to one session between therapist and patient, ii) daily one-hour bedside therapy session that consists of positioning, passive range of motion, stretching and strengthening exercises of all the limbs, iii) ambulation therapy, iv) chest physiotherapy and v) education sessions to caregiver when necessary especially in more medically stable patients.

Orthosis prescription is not a standard treatment option for prevention of equinovarus in our centre, but rather a management option depending on case-to-case basis after the evaluation by a rehabilitation physician. The type of orthosis prescribed for the lower limb included Plaster of Paris (POP) cast and ankle-foot orthosis (AFO). Other spasticity management such as oral medication and Botulinum toxin-A injection are not part of a standard treatment in NICU but provided depending on case-to-case basis after the evaluation by a rehab physician.

Patient Recruitment and Methods

All patients who were referred with a first episode of moderate to severe brain injury were included in the study. Severity of brain injury was determined by the post-resuscitation GCS, and those with GCS of less than 12 were included. Haemorrhagic stroke patients were also selected according to the same GCS scoring method. Other inclusion criteria included patients who were 18 years and older, having the ability to give consent or eligibility of

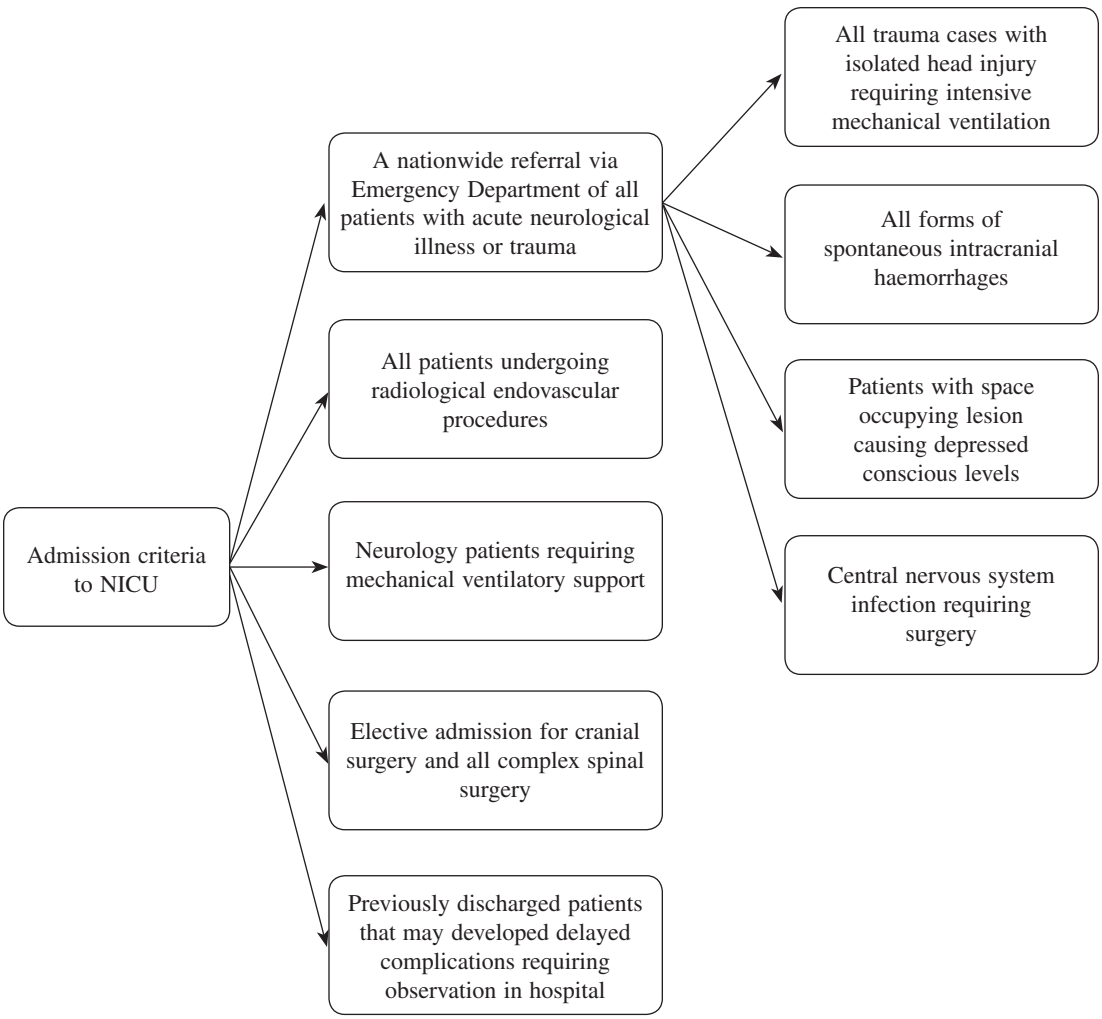

Figure 1. Flow diagram of the admission criteria to Neuro Intensive Care Unit (NICU)

a proxy to give consent, medically stable by the end of first week of brain injury onset, no previous musculoskeletal deformities in the lower limbs, no concomitant spinal or lower limb injuries (soft tissue injuries or fracture) and agreed to participate for the total duration of assessment of three months which include coming for outpatient clinical assessment if length of hospital stay is less than three months.

Patients who fulfilled the inclusion criteria were examined within the first week of admission into the NICU followed by weekly assessments until hospital discharge or until ankle contracture deemed present. All patients received the existing standard physiotherapy care throughout hospital stay. Education to caregiver was emphasized early after admission and the caregivers were also taught how to perform the proper prevention exercises upon patient discharged. Patients who were discharged before three months were followed up at the outpatient rehabilitation clinic monthly instead of weekly. This was done to improve follow-up compliance thus reducing the dropout rates normally observed in prospective cohort study. The end points for clinical assessment were three months from onset of brain injury or until the development of ankle contracture, whichever occurred earlier.

Clinical assessments

The assessments included measurement of ankle dorsiflexion passive range of motion (ROM) and evaluation of the ankle muscle tone. Two assessors specifically trained and assigned for this study were present throughout the assessments (both

Table 1: The definition for lower limb muscle tone pattern in this study

Muscle tone	Definition
Spasticity	A velocity-dependent increased resistance to passive muscle stretch
Spastic dystonia	Sustained muscle contractions causing twisting and repetitive movements or abnormal postures
Sustained clonus	Involuntary repetitive contractions and relaxations of the muscles in the presence of spasticity which is unfatiguable lasting more than 10 seconds

during hospital stay and during outpatient follow up). To measure the maximal ankle dorsiflexion passive ROM, the patient was first put in a supine position. Then, the first assessor would stand facing the lateral aspect of the tested foot and while grasping the calcaneus firmly, used his forearm to apply a maximal dorsiflexion force to the plantar surface of the foot. This force was maintained for a few seconds whilst the second assessor measured the ankle joint angle with a universal goniometer.[12] The measurement was done twice, with the knee at full extension and at 90 degrees flexion. Ankle contracture was considered to be present if two consecutive weekly measurements of maximum ankle dorsiflexion were less than 0 degree at knee extension. However, the value of ankle dorsiflexion angle at knee flexion was also documented to represent primarily soleus extensibility.

The duration from brain injury to onset of contracture was also documented. Patients who were discharged before the onset of ankle contracture were followed up monthly until ankle contracture was detected or for the total of three months from brain injury. At each follow up, a repeat assessment on the caregiver's education and exercise compliance was conducted. When necessary, re-enforcement on the proper techniques was carried out. Compliance was considered achieved through caregiver's testimony. Of those who had developed contracture prior to our clinic review, the onset of contracture was obtained retrospectively from the patient or the main caregiver in terms of weeks.

Lower limb pattern of muscle tone was divided into four categories: normal, hypotonia, spasticity, spastic dystonia or presence of sustained clonus. The definition of spasticity, spastic dystonia and sustained clonus is presented in Table 1.[3] The presence of the muscle tone would be recorded simultaneously if there is more than one category observed in the same patient. The categories were only recorded as 'presence or absence' except for spasticity. Modified Ashworth Scale (MAS) was used for the measurement of spasticity severity.

Patient's demographic data, types of brain injury, severity of brain injury, total length of hospital stay, duration of invasive mechanical ventilation and presence of sepsis were recorded. All patients were treated according to the standard rehabilitation care in UMMC during their hospital stay which included daily limb exercises (stretching and range of motion exercises) by the physiotherapist.

Statistical analysis

Patient demographic characteristics were presented using a descriptive analysis, with a section describing the characteristics of patients developing ankle contractures. The association of the variables with ankle contracture was obtained using Chi-Square method with Yates correction for smaller sample number cells. The odds ratios were obtained through univariate logistic regression analysis. P value of <0.05 was considered statistically significant. Time from NICU admission to diagnosis of ankle contracture was recorded in weeks. The total length of weeks was 12 weeks from the acute NICU admission.

RESULTS

Participants

Over the 12-month study period, 462 patients were admitted to the NICU. A total of 70 patients fulfilled the criteria and agreed to participate in the study (100% recruitment rate). Forty-six patients (65.7%) completed the study with 24 dropouts. Nineteen patients died before completion of the study and five patients were not contactable after discharge. Thirteen patients developed ankle contracture and 33 patients were designated as normal at the end of the study period.

Table 2 represents the total patients who completed the study. There were more males

Table 2: The descriptive outcomes on the incidence of ankle contracture among the brain injury patients

Variables	Ankle contracture	
Total	**No n=33**	**Yes n=13**
Mean contracture time		3 weeks
Age in years (mean age =38 years old)		
≤ 38	16 (66.7%)	8 (33.3%)
> 38	17 (77.3%)	5 (22.7%)
Gender		
Male	26 (68.4%)	12 (31.6%)
Female	7 (87.5%)	1 (12.5%)
Ethnic		
Malay	16 (80%)	4 (20%)
Chinese	7 (63.6%)	4 (36.4%)
Indian	9 (75%)	3 (25 %)
Other	1 (33.3%)	2 (66.7%)
Cause of injury		
Traumatic brain injury	21 (70%)	9 (30%)
Haemorrhagic stroke	12 (75%)	4 (25%)
Motor pattern spasticity		
Non spastic	31 (91.2%)	3 (8.8%)
Spastic	2 (16.7%)	10 (83.3%)
Spastic dystonia		
No	32 (82.1%)	7 (17.9%)
Yes	1 (14.3%)	6 (85.7%)
Clonus		
No	30 (81.1%)	7 (18.9%)
Yes	3 (33.3%)	6 (66.7%)
ABI		
Severe	18 (64.3%)	10 (35.7%)
Moderate	15 (83.3%)	3 (16.7%)
Length of stay in hospital (mean length=4.8 weeks)		
≤ 5 weeks	23 (76.7%)	7 (23.3%)
> 5 weeks	10 (62.5%)	6 (37.5%)
Mechanical ventilation		
No	9 (90%)	1 (10%)
Yes	24 (66.7%)	12 (33.3%)
Duration of mechanical ventilation (mean length =1.85 weeks)		
No mechanical ventilation	9 (90%)	1 (10%)
≤ 2 weeks	17 (73.9%)	6 (26.1%)
> 2 weeks	7 (53.8%)	6 (46.2%)
Sepsis		
No	23 (79.3%)	6 (20.7%)
Yes	10 (58.8%)	7 (41.2 %)

compared to females with the ratio of 4.75:1. There were two different age peak of study population. TBI occurred predominantly in the younger age group, whereas haemorrhagic stroke occurred among older age group. Of the remaining 46 subjects, 28 suffered from severe ABI with 18 subjects in the moderate category. The majority cases of severe brain injury were due to traumatic injury whereas hemorrhagic stroke yielded a bigger number in the moderate brain injury group. The mean length of hospital stay was 4.8 weeks and the mean length of time for mechanical ventilation was 1.85 weeks. The mean duration of time for contracture to occur was 3 weeks.

Ankle contracture

As seen in Table 3, 13 out of 46 patients (28%) developed ankle contracture. Presence of lower limb spasticity (MAS > 1), spastic dystonia and clonus exhibited higher number of contracture incidence with statistical significance (spasticity p=<0.001; spastic dystonia p=0.001; clonus p=0.015). There were a higher proportion of mechanically ventilated patients developed ankle contracture compared to non-ventilated patients. There was also a difference between the incidence of contracture and length of mechanical ventilation time. Only six patients who were ventilated for less than two weeks had ankle contracture compared to almost half of the patients who were ventilated for more than two weeks (26.1% versus 46.2%). There was a weak association between mechanically ventilated patients for the duration of more than two weeks with ankle contracture (p=0.052).

Almost a third of the TBI and haemorrhagic stroke patients had contracture (30% and 25% respectively). More patients with severe brain injury had ankle contracture compared to moderate brain injury group (35.7% versus 16.7%). Ankle contracture incident was also higher in the sepsis group. Nearly half the number of patients with sepsis had ankle contracture whereas less than one third number of patients without sepsis had ankle contracture. The incident of contracture was also lower for patients who stayed in hospital for less than five weeks compared to patients who stayed longer (23.3% versus 37.5%). The mean time length of contracture development was 3 weeks. Out of the 13 confirmed contracture cases, eight patients had bilateral ankle contracture (61.5%) with the remaining patients had unilateral ankle contracture (38.5%).

Abnormal motor pattern was found to be a significant risk factor for the development of ankle joint contracture (Table 4). This included spasticity, spastic dystonia and clonus (p=<0.05). Patients with spastic motor pattern were 51.67 times more likely to develop ankle contracture (OD 51.67, 95% CI 7.53-354.52; p=<0.001) than patients with non-spastic motor pattern. Although patients with hypotonia muscle tone were grouped together in the non-spastic motor group, analysis excluding hypotonia (which consisted of three patients) yielded a statistically significant risk for ankle contracture (OR 8.52, CI 13.00-24.18, p<0.001).

Spastic dystonia (OR 27.43, 95% CI 2.84-265.35, p=0.004) and presence of clonus (OR 4.18, 95% CI 1.33-13.19, p=0.015) were also both statistically significant risk factors. There is a weak risk between a longer duration of mechanical ventilation to developing ankle contracture (p=0.087). Being mechanically ventilated for more than two weeks gave 7.71 times more chance of developing ankle contracture although it was not statistically significant (OD 7.71, 95% CI of 0.75-79.77, p=0.087). Other variables tested including age, gender, ethnicity, cause of brain injury and length of hospital stay were not statistically significant risk of joint contracture formation (Table 4).

DISCUSSION

Almost a third of our patients (28%) developed ankle contracture within the first three months after ABI. There is a wide range of reported incidence of ankle contracture in previous studies. This could be due to the different inclusion criteria, definition of ankle contracture, study setting, and study duration as well as intensity of rehabilitation management. For example, one study reported a lower incidence of ankle contracture (16.2%) within the first 16 weeks after brain injury.[3] The study was conducted on new patients with ABI admitted to the rehabilitation unit, and contracture was considered only to be present on a minimum of three measurement occasions of ankle dorsiflexion less than 0^{o} on manual stretching. We adopted almost a similar method of ankle measurement but found a higher incidence of ankle contracture. The different result is most probably because our study was conducted in a different setting (an acute NICU setting) and the study population mainly comprised of TBI and haemorrhagic stroke patients.

Another study conducted in a general ICU setting reported similar finding of ankle contracture incidence as ours.[7] However the

Table 3: The association between multiple factors to incidence of ankle contracture

Variables	Ankle contracture	*P*	*P* *(Yates correction)*
Total	**n=13**		
Age (years)		0.425	0.638
≤ 38	8 (61.5%)		
> 38	5 (38.5%)		
Gender		0.409	0.511
Male	12 (92.3%)		
Female	1 (7.7%)		
Ethnic		*0.265	
Malay	4 (30.8%)		
Chinese	4 (30.8%)		
Indian	3 (23.1%)		
Other	2 (15.4%)		
Cause of injury		*0.723	
Traumatic brain injury	9 (69.2%)		
Non traumatic brain injury	4 (30.8%)		
Motor pattern spasticity		**<0.001**	**<0.001**
Non spastic	3 (23.1%)		
Spastic	10 (76.9%)		
Spastic dystonia		**0.001**	**0.001**
No	7 (53.8%)		
Yes	6 (46.2%)		
Clonus		**0.009**	**0.015**
No	7 (53.8%)		
Yes	6 (46.2%)		
ABI		0.161	0.287
Severe	10 (76.9%)		
Moderate	3 (23.1%)		
Length of stay in hospital		0.328	0.501
≤ 5 weeks	7 (53.8%)		
> 5 weeks	6 (46.2%)		
Mechanical ventilation		0.240	0.292
No	1 (7.7%)		
Yes	12 (92.3%)		
Duration of mechanical ventilation		*0.056	
No mechanical ventilation	1 (7.7%)		
≤ 2 weeks	6 (46.2%)		
> 2 weeks	6 (46.2%)		
Sepsis		0.136	0.25
No	6 (46.2%)		
Yes	7 (53.8%)		

* Variables using the Mann-Whitney U test to get the p-values.

Table 4: Univariate analysis of predictors for ankle contracture

	No. of patients	Univariate analysis Hazard ratio (95% CI)	*P*
Total	**46**		
Motor pattern spasticity			
No spasticity (normal + hypotonia)	34	1.00	
Spasticity	12	51.67 (7.53-354.52)	**<0.001**
Motor pattern spasticity			
No spasticity (hypotonia group excluded)	31	1.00	
Spasticity	12	8.52 (3.00-24.18)	**<0.001**
Clonus			
No	37	1.00	
Yes	9	4.18 (1.33-13.19)	**0.015**
Spastic dystonia			
No	39	1.00	
Yes	7	27.43 (2.84-265.35)	**0.004**
Duration of mechanical ventilation			
No mechanical ventilation	10	1.00	
≤ 2 weeks	23	3.18 (0.33-30.62)	0.317
> 2 weeks	13	7.71 (0.75-79.77)	0.087
Length of stay in hospital			
≤ 5 weeks	30	1.00	
> 5 weeks	16	1.97 (0.53-7.37)	0.313
Age (years)			
≤ 38	24	1.00	
> 38	22	0.59 (0.16-2.18)	0.427
Ethnic			
Malay	20	1.00	
Chinese	11	2.29 (0.44-11.86)	0.325
Indian	12	1.33 (0.24-7.34)	0.741
Cause of brain injury			
Traumatic	30	1.00	
Non traumatic	16	0.78 (0.19-3.08)	0.720
Severity of injury			
Severe	28	1.00	
Moderate	18	0.36 (0.08-1.55)	0.170
Gender			
Male	38	1.00	
Female	8	0.31(0.03-2.81)	0.297
Sepsis			
Yes	29	1.00	
No	17	2.68 (0.72-10.04)	0.143

definition of ankle contracture in that study was recorded as range of motion that was short of full range (0 to 19°) as opposed to our study which used stricter criteria to define ankle contracture. Another retrospective study reported an even higher number of ankle contracture (76%) but the contracture was observed within one year of craniocerebral trauma, which is longer than our study.[1]

We found a higher incidence of ankle contracture among patients with abnormal motor pattern. Spasticity group (p=<0.001), spastic dystonia (p=0.001) and presence of clonus (p=0.015) were significantly associated to incidence of ankle contracture. Similar findings were found in a study by Singer *et al*.[3], which reported that ankle deformity occurrence was highly related to the presence of abnormal muscle tone. However in the same study, no subjects with normal tone developed the deformity. In contrast to our findings that revealed presence of ankle contracture in the normal muscle tone group albeit at a lower incidence.

In our study, patients with TBI had a higher incidence of ankle contracture as compared to patients with haemorrhagic stroke. Because majority of the TBI patients were in the severe brain injury category, we speculate that the reason for the higher incidence could be due to the severity of brain injury rather than the aetiology of brain injury. Severe brain injury has been reported to be associated with the occurrence of ankle contracture. Singer *et al*.[3] reported two third of their patients with contracture had suffered from severe brain injury. They had further found a weak association between lower admission GCS score and the likelihood of developing ankle contracture. Yarkony and Sahgal[1] on the other hand reported that the number of contracture increased with the increased duration of coma.

When we analysed the possible predictors of ankle contracture, presence of abnormal motor pattern including spasticity, clonus or spastic dystonia emerged as strong predictors. The odds for developing ankle contracture were 51.67 among patients with spasticity, 27.43 among patients with spastic dystonia and 4.18 among patients with clonus. This is consistent with the mechanism in which damage to central motor pathways leads to muscle over activity. This can cause abnormal joint postures and coupled with immobilization, leads to muscle shortening and contracture formation.[5]

Another factor that showed a weak predictive factor for ankle contracture was duration of mechanical ventilation of more than two weeks. The odds ratio was 7.71 when compared to patients without mechanical ventilation. So far we have not found studies with similar result. Our findings revealed that 86% of patients with ankle contracture who were ventilated for more than 2 weeks, suffered severe brain injury affecting large cerebral areas or with multiple intraparenchymal haemorrhages. Booth *et al*.[13] have looked at the relationship between affected brain areas and ankle contracture; and reported that patients with brainstem lesions as opposed to cortical lesions had worse initial ankle deformity. However, we did not explore the similar relationship in this study. The other variables tested such as age, length of hospital stay, ethnicity, cause of injury, gender and presence of sepsis did not affect the risk of ankle joint contracture.

A different intensity and type of therapy given may yield a different result of the ankle contracture occurrence and progress. We applied the existing standard therapy program in our centre to all patients in this study. Similarly, Singer *et al*.[9] applied the standard therapy program at their centre and reported that contracture persisted or worsened in 17 of 40 cases, all of which exhibited dystonic muscle over activity producing sustained equinovarus posturing. This supports the notion that pathophysiology of muscle tone abnormality and muscle tone severity may play a pivotal role on contracture formation despite a regular physiotherapy program. A standard guideline on the intensity and type of early therapy program to prevent ankle contracture among brain injury survivors is not currently available. However, the safety and benefit of early rehabilitation have been established in several studies where improved functional outcome, mobility and reduced ICU-acquired weaknesses were reported.[14-16]

There were a few limitations in this study. Firstly, the number of patients in this study population was smaller compared to other studies, thus reducing the statistical strength of the study. There are a few factors contributing to this. We have stricter inclusion criteria and due to the nature of the brain injury severity, we had a high number of dropouts from mortality alone. Time constraint for the study duration was another factor for the small number of patients. Longer study duration would have improved the number of patients recruited.

Secondly, we were unable to have a control group of different therapy intensity in this study due to the limited human resource. Patients normally received a one-to-one therapy session

due to the acute condition. Modifying the duration and intensity could inevitably disrupt the normal day-to-day therapy services. However, since the main objective of this paper is not to assess the early therapy method effectiveness or the time of onset of contracture, but rather the incidence of early ankle contracture despite the existing standard therapy, we believe the findings will still be of importance to clinicians managing patients in similar condition. For future studies, it is worthwhile to compare ankle contracture occurrences amongst different therapy programs.

In conclusion, this study showed that incidence of ankle contracture after brain injury is high within the first three months despite early standard therapy program. The mean time of onset of ankle contracture was three weeks. Abnormal motor pattern such as spasticity, spastic dystonia and presence of clonus are strongly related to the likelihood of developing ankle contracture. These findings will assist clinicians in early detection and management of ankle contracture while the patients are still receiving treatment in the acute intensive care setting. Interventions to manage spasticity and other abnormal muscle tones may be incorporated early before any irreversible loss of range of motion, which can result in long-term functional impairments.

DISCLOSURE

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Conflict of interest: None

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