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Correspondence :

Editorial Office :

Prof (Dr) Rathindranath Haldar
Department of Physical Medicine & Rehabilitation,
Institute of Post Graduate Medical Education & Research & SSKM Hospital,
244, AJC Bose Road, Kolkata - 700020.
E-mail:indianjournalofpmr@gmail.com Phone no : 00 91 9830152173

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2. The Rehabilitation Specialist's Handbook (Rehabilitation Specialist's Handbook (Rothstein), Jules M. Rothstein, Serge Roy, Steve Wolf, David Scalzitti. 3rd edition: 2005,F.A. Davis Company; 3 edition.
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1. *Jorge H. Villafañe, Guillermo B. Silva, Mark D. Bishop, Josue Fernandez-Carnero*. Radial Nerve Mobilization Decreases Pain Sensitivity and Improves Motor Performance in Patients With Thumb Carpometacarpal Osteoarthritis: A Randomized Controlled Trial.Archives of Physical Medicine and Rehabilitation, March 2012; **93(3):** 396–403.
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Original Article

Correlation of Gross Motor Function with Topographical Diagnosis in Children with Cerebral Palsy

Tufail Muzaffar[1], Nonica Laisram[2], S Y Kothari[3]

Abstract

There is a need for appropriate classification to describe gross motor function status in a child with cerebral palsy (CP). It was hypothesised that: greater the number of limbs involved, higher would be the Gross Motor Function Classification System (GMFCS) level; and, there would be spectrum of GMFCS level for each of the topographical types of the cerebral palsy.

A cross-sectional study of 182 children of both sexes in the age group of 7 months to 30 years having spastic CP who attended CP clinic from 2008 to 2009 in tertiary care hospital were assessed for topographical diagnosis and GMFCS levels. Topographical distribution showed diplegia (42%), quadriplegia (30%), hemiplegia (23%), triplegia (4%) and monoplegia (1%). GMFCS levels were almost evenly distributed, level II (26%) was most common followed by level V (23%). Statistical analysis was done using Cramer's ratio and Pearson's Chi-square test.

Cramer's ratio of 0.277 showed fairly weak correlation between GMFCS levels and topographical CP types. Pearson's Chi-square (12) =41.7, p=0.000 indicates that there is significant difference between expected and observed values of number of limbs involved in GMFCS levels, further substantiating the weak correlation.

These results mean that GMFCS in different topographical groups have different distributions. It was also observed that GMFCS had weak correlation with the number of limbs involved, thus reflecting that the GMFCS is a better indicator of gross motor function impairment than the traditional topographical categorisation of CP that specifies the number of limbs involved.

Key words : **Cerebral palsy, topographical, correlation, spasticity.**

Introduction:

Assessing the cerebral palsy (CP) child remains an enigma for researchers and clinicians. This study is an attempt to analyse the gross motor function status in accordance with the Gross Motor Function Classification System (GMFCS), and its correlation with the topographical diagnosis in CP.

CP describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of CP are often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour; by epilepsy, and by secondary musculoskeletal problems.[1]

CP is a clinical description and by itself is not informative about the outlook of these infants. The lesions in the developing brain or CP aetiology alone also do not provide information about function and prognosis. In our day to day practice of CP rehabilitation, it is important to follow a classification system that tells the parents and patients about the functional status and future prognosis, at the same time helping the managing team to plan intervention for the CP child, as well as for measuring the

Author's affiliations:
[1] MBBS PGT DNB (PMR), Department of PMR VMMC and Safdarjang Hospital
[2] MBBS, DCH, DPMR, DNB(PMR), Professor and Senior Specialist PMR, Department of PMR VMMC and Safdarjang Hospital
[3] MBBS, MS(Orth.), DNB(PMR), Special DGHS

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Correspondence :
Dr Nonica Laisram MBBS, DCH, DPMR, DNB(PMR)
Professor and Senior Specialist PMR
Department of PMR VMMC and Safdarjang Hospital,
New Delhi 29.
E-mail: drnonica@gmail.com

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outcome of rehabilitation. Unfortunately the many different classifications of the various types of CP that are in use lead to great confusion, and uniformity of observation is impeded at a time when increasing attention to disabled children gives some promise of real advances in management.[2]

Given the complex and variable nature of the movement disorders in children with CP, experts believed that consensus should be reached as an essential step in the development of a valid classification system and subsequent use of the system in clinical practice and research. The results of the nominal group process and Delphi survey consensus methods provided evidence of the validity of the GMFCS.[3] The international groups of experts were unanimous in their agreement that there is a need for a classification system for children with CP that is based on the construct of disability and functional limitation. The GMFCS is a five-level classification that differentiates children with CP based on the child's current gross motor abilities; limitations in gross motor function, and need for assistive technology and wheeled mobility.[3]

Until recently, the severity of CP was described in subjective terms such as mild, moderate, and severe. The Gross Motor Classification System (GMFCS) provides the physiatrists, orthopaedic surgeons, neurologists, paediatricians and therapists with a common language to describe functional status of children with CP. It is reliable and stable over time. It is easy to learn and can be worked out for specific child in about five minutes. The use of common classification to describe gross motor function in a child with CP improves communication and understanding between all professionals taking care of the CP child.

The purpose of this study was to observe correlation of GMFCS with topographical diagnosis in children with spastic cerebral palsy. It was hypothesised that (1) greater the number of limbs involved, higher would be the MFCS level, (2) there would be a spectrum of GMFCS level for each of the topographical CP types.

Patients and Method:

A cross-sectional study was done in 182 CP children of both sexes in the age group of 7 months to 30 years who attended CP clinic in Department of PMR, VMMC and Safdarjang Hospital, New Delhi from March 2008 to May 2009. Children were assessed for number of limbs involved (topographical diagnosis) and corresponding GMFCS levels. Only those patients with spasticity who were diagnosed as CP, (as per definition of Executive committee Chaired by Rosenbaum *et al*)[1] were included in the study. Data obtained from the clinical examination was recorded and interpreted. For statistical analysis, the data were coded and compiled accordingly and processed by using computer based program SPSS version16. Pearson Chi-square test with 95% confidence interval and Cramer's ratio were done to see the level of significance of correlation.

Results:

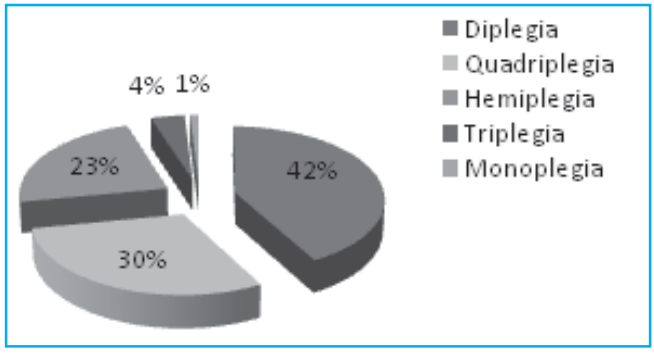
Male: female ratio 3:2; male predominance.

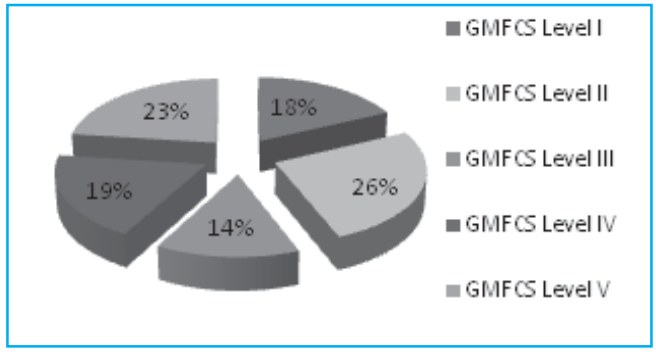
Table 1: ***Age Distribution (n=182)***

Age	No. of cases	Percentage (%)
Upto 2 years	50	27
>2-4 years	47	26
>4-6 years	45	25
>6-12 years	37	20
More than 12 years	3	2

*Age distribution was almost uniform from 2 to 12 years of age

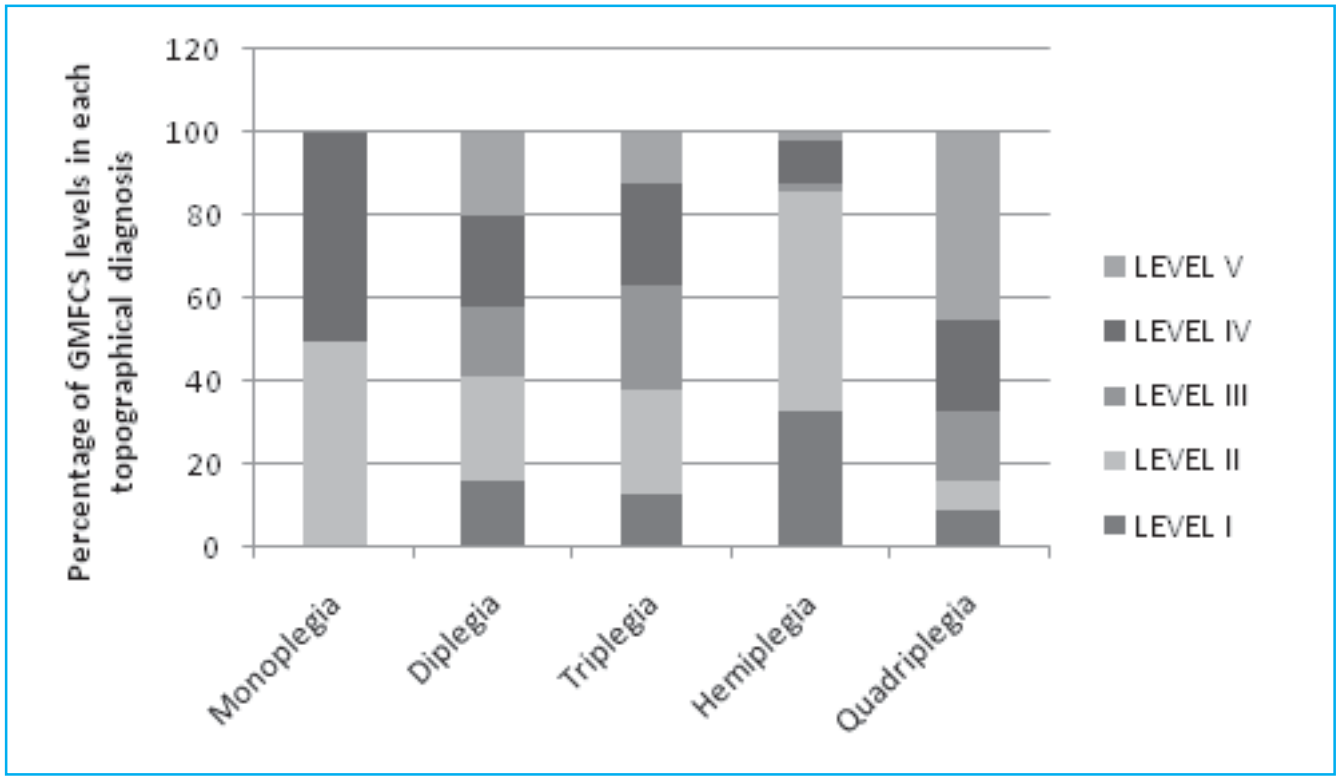
Fig 1: ***Percentage Representation of Each Topographical Type and Each GMFCS Level (n=182)***





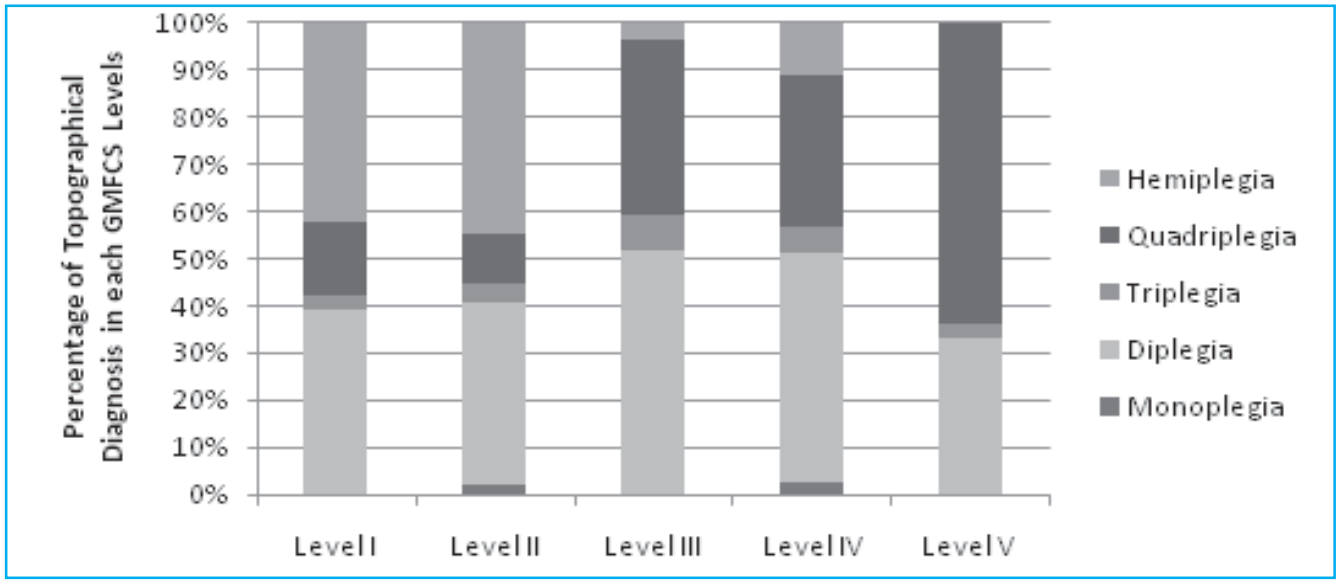
*In our study (Fig 1) topographical distribution was diplegia (42%), quadriplegia (30%), hemiplegia (23%), triplegia (4%) and monoplegia (1%). GMFCS levels had almost uniform distribution; level II (26%) was most common followed by level V (23%).

Fig 2: ***GMFCS Levels within Different Types of Topographical Diagnosis (n =182)***



***** In diplegics GMFCS levels I to V was almost evenly distributed. Quadriplegia had (63.6%) on the severe side of spectrum; levels IV and V, however it had significant percentage representations in other levels as well. Hemiplegia had more favorable GMFCS levels (level I -34% and level II 54 %.). Triplegia had equal (25%) representation in levels II to IV and (12.5%) on either end of spectrum in level I and level V. Of the two cases of monoplegia where there was upper limb involvement in both cases, one had GMFCS level II and other had level IV.

Fig 3: ***Topographical Diagnosis within Different GMFCS Levels (n=182)***



*In level I diplegia (39%) and hemiplegia (42%) were dominating which continued in level II as well (diplegia 39% and hemiplegia 45 %.). Level III and IV had maximum number of diplegia and quadriplegia combined (more than 80% in each level), with a continued trend of rise of level V in Quadriplegia, while hemiplegia was just (4%) and (11%) respectively. In GMFCS level V quadriplegic group had maximum (54%) representation followed by diplegia (33%) with only minimal representation in the other topographic types. Our results in Figs 2 and 3 show that a CP child can have varied levels of function with different number of limbs involved.

Table 2: *Comparison with Other International Studies*

GMFCS level	I %	II %	III %	IV %	V%	Total
SJH India	17.60% (n=32)	25.80% (n=47)	13.70% (n=25)	19.20% (n=35)	22.50% (n=41)	100% (n=182)
Victoria [8]	35.30% (n=114)	16.40% (n=53)	14.20% (n=46)	16.10% (n=52)	18.00% (n=58)	100% (n=323)
Ontario [8]	27.90% (n=183)	12.20% (n=80)	18.60% (n=122)	20.90% (n=137)	20.50% (n=135)	100% (n=657)
Sweden [8]	40.70% (n=68)	18.60% (n=31)	13.80% (n=23)	11.40% (n=19)	15.60% (n=26)	100% (n=167)

*In our study least percentage(13.7%) of cases were in GMFCS level III and highest (25.8%) in level II. Representations in levels I, IV and V are comparable to those of internationally observed results.

In our study it was hypothesised that more the number of limbs involved, higher the GMFCS levels. However on statistical analysis with Cramer's ratio (for correlation of two variables), result was 0.277 implying that topographical classification and GMFCS are weakly correlated. On Pearson's Chi-square test of significance at degree of freedom 12, the value was 41.7 with significance of p=0.000. This tells us that there is significant difference between expected and observed values of number of limbs involved in GMFCS levels, further substantiating the weak correlation of GMFCS with topographical diagnosis.

Discussion:

Male : female (3:2) with higher incidence of male children in our study is comparable to other studies.[4-9] In our observation diplegia (42%) was the most common topographical group followed by quadriplegia (30%) and hemiplegia (23%) respectively; triplegia (4%) and monoplegia (1%) were least common. These findings are in accordance to Vohr *et al*[10] where the most common type of CP was spastic diplegia (39%) followed by quadriplegia (27.3%), hemiplegia (13.8%), hypotonic CP (9.9%), triplegia (6.0%), and monoplegia (3.9%). However there was significant difference in topography compared to Howard *et al*[8] in Victorian study where hemiplegia (35%), diplegia (28%) and quadriplegia (37%) were found. In a study of adults with CP by Margre *et al*[9] in Brazil, quadriplegia was the most common group followed by diplegia and hemiplegia.

Our study showed that GMFCS level II was most common (26%) followed by level V (23%), level III was least common (14%). Similar observations were found in Victorian study where level I was most common (35%) followed by level V (18%), and level III was least common (14%).[8]

Vohr *et al*[10] studied a longitudinal cohort of 282 children with CP and found children with more limbs involved had more abnormal GMFCS levels reflecting more severe functional limitations. Spearman rank-order correlations were run to assess the relationships among the topography classification for spastic CP, and the GMFCS levels. There was strong correlation between topography and the GMFCS (r = 0.894; p =.0001). However, for each CP topographical category, there was a spectrum of gross motor functional levels. Though our results had a spectrum of GMFCS for each topographical type, in contrast to Vohr *et al*[10] the two classifications were not as strongly correlated.

Howard *et al*[8] in Victorian study found within the spastic group, differences in motor function among the three topography groups were extremely clear-cut. Compared to children with hemiplegic distribution, children with diplegia were more severe on the GMFCS scale. Children with spastic quadriplegia had the lowest levels of function, being significantly higher on the GMFCS scale than those with hemiplegia and diplegia. Our observation of hemiplegic children are in accordance with those of Howard *et al*[8] who also found hemiplegic children had better functional profile than bilateral syndromes. However our study did not show statistically strong correlation between number of limbs involved and GMFCS levels.

Gorter *et al*[11] also found that majority (87.8%) of children with hemiplegia were classified as level I as observed in our study where hemiplegic children had maximum representation in levels I and II (34% and 54%) respectively. They also observed children with a

bilateral syndrome were represented in all GMFCS levels, with most in levels III, IV, and V which is similar to our study.

In our study we found GMFCS and topographic diagnosis had a fairly weak correlation with Cramer's ratio and Pearson's Chi-square test. Similar observation was made by Gorter *et al*[11] who found classifications by GMFCS and 'limb distribution' or by GMFCS and 'type of motor impairments' were significantly associated, though the correlation for limb distribution by GMFCS was low. The association between limb distribution and GMFCS levels was modest at best. Gorter *et al*[12] found that GMFCS classification in infants is less precise than classification over time in older children. Children can be classified by the GMFCS early on, but there is a need for reclassification at age 2 or older as more clinical information becomes available.

Conclusion:

GMFCS and topographical distribution of limb involvement are weakly correlated. There is a variable spectrum of GMFCS levels in each topographical type of CP. A mild quadriplegia with GMFCS II can differ vastly from a severe quadriplegia with GMFCS V. The GMFCS is hence a better indicator for grading the child's gross motor functional performance than the topographical diagnosis. . GMFCS is a simple, intuitive and reliable tool to classify gross motor function in children with CP. GMFCS tells the parents and the patient about the functional status and the future prognosis, at the same time helping the managing team to set goals for measuring the outcome of rehabilitation.

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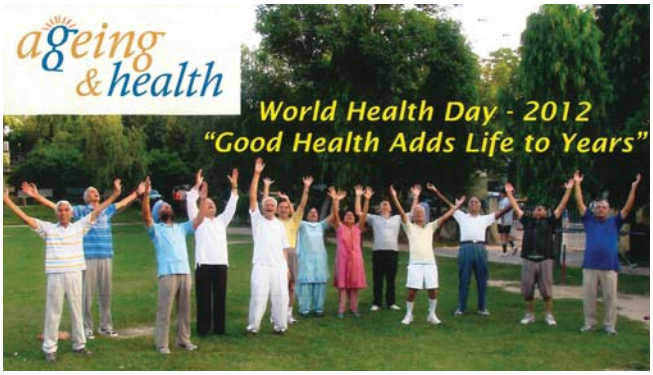
Editorial



Good Health Adds Life to Years

World Heath Day (WHD) is celebrated every year on 7 April to mark the anniversary of World Health Organization (WHO) founded in 1948. World Health Day is a global campaign, inviting everyone – from global leaders to the public in all the countries- to focus on a single health challenge with global impact. The topic of World Health Day in 2012 is "Ageing and Health" with the theme "Good Health Adds Life to Years". The focus is on how good health throughout life can help older men and women to lead full and productive lives and be an invaluable resource for their families & communities.

Ageing is a lifelong, inevitable universal phenomenon, common to all communities, nations & sexes. The number of people today aged 60 & over has doubled since 1980. People aged 80 years will almost quadruple to 395 million between now and 2050.Within the next five years, the number of adults aged 65 and over will outnumber children under the age of 5.By 2050, these older adults will outnumber all children under the age of 14. Now the majority of older people live in low- or middle-income countries which will be increased by 80% by 2050.

The 2001 census counts the proportion of Indian population above the age of 60 years as 77 million, which constitutes about 7.4% of the entire population. It is estimated that by2051 the population of 60 plus is expected to reach over 300 million, translating to 17% of the total population. This increases the burden of chronic illness and ailments specific to these twilight years tending to bring down the quality of life. Population ageing will hamper the achievement of socioeconomic and human development goals, if it is not taken today.

Good health in older age can be achieved by promoting health across the life –course, creating age-friendly environments that foster the health and participation of older people, providing access to basic primary health care, long-term care & palliative care, acknowledging the value of older people and helping them to participate fully in family and community life.

The challenge for India as for all countries over the world is not just to add further years to life but to add "Life to Years". To enable the elderly to live full enriching and productive lives, they need to remain connected to the world around them, staying amongst and in harmony with their beloved one's and others of all ages in their communities. Let us join hands to promote a society in which the elderly are respected and treasured, stay connected to their families and their communities and live full and productive lives.

The biggest challenge of this hour for the physiatrist, more precisely for the young budding energetic rehabilitation physician of India is to play a pivotal role for the programme. They must be well equipped with proper training in clinical skills and acumen in managing these generations for better living and to re-able them as a resource of country rather than a liability.

Hence the need of this 21st century rehabilitation world is to engage more & more Physiatrist in the field of geriatric rehabilitation, to create a rehabilitation care system for the elderly. If we can expertise at optimum level in geriatric medicine then only we can really 'Add Life to Years not Years to Life'.

Prof. R N. Haldar

Case Report

A Patient with Fixed Flexion Deformity of Hip and Knee

R Pramanik[1], P Das[2], A Basak[3], D Ghorai[4], P P Pan[5], D K Khatua[6]

Abstract

A 17 years old female patient presented to PMR OPD with fixed flexion deformity of left hip and knee and cachexia. Five years back a severe pain was suddenly developed in her left knee and thigh which was investigated for juvenile inflammatory arthropathy and rheumatic arthritis. At that time all the serological markers (ANA, RF, ASO titre) and x-ray of knee were normal. Subsequently left hip pain and restricted ROM were developed which made it clear that the knee pain was actually referred from hip. A plain x-ray of hip was done to rule out Perthe's disease which was reported as avascular necrosis of femur.

When the patient was examined at PMR OPD, a CT scan of hip, routine hemogram, CXR, Manteux test was advised considering a provisional diagnosis of infective pathology like TB hip with a differential of neoplaia in or around hip keeping in mind about cachexia and weight loss. Surprisingly CT scan showed a big mass originating from glutei muscles evading back of the thigh and even left sphincter ani muscle. Fortunately patient was continent at that time. Interestingly the pathological report suggested a relatively rare diagnosis which practically made the patient bedridden with commonly featured fixed flexion deformity.

Key words* : *Fixed flexion deformity, rehabilitation.

Introduction:

Fixed flexion deformity (FFD) is one of the common clinical entities presented to PMR outpatient department for rehabilitation. FFD of hip is often associated with fixed knee flexion and hyperextension of the lumbar spine.[1] Most of the time flexion deformity occurs due to contracture of joint capsule or of muscle.

Author's affiliations:
[1] MD, MRCP (UK). Assistant Professor, Department of PMR, IPGMER, Kolkata
[2] MD. Assistant Professor, Department of PMR, IPGMER, Kolkata
[3] MBBS, Post graduate trainee, Department of PMR, IPGMER, Kolkata
[4] MBBS, Post graduate trainee, Department of PMR, IPGMER, Kolkata
[5] MD. Associate Professor, Department of PMR, NBMC, West Bengal
[6] MD. Associate Professor, Department of PMR, BSMC, West Bengal

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Correspondence :
Dr R Pramanik,
Flat 3A, 'Digantika', 453 DumDum Park, Tank 5,
Kolkata, West Bengal. Pin-700055.
Email: rpramanik2000@yahoo.com, Ph.: 0091 9432594882

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FFD is the commonest deformity of the hip and very common in some form of arthritis like pyogenic, tuberculous, osteo-arthritis, rheumatoid arthritis etc.[2] In India tuberculosis infection is really prevalent particularly in lower socioeconomic condition.

Case Report:

A seventeen years young lady was referred to outpatient department of Physical Medicine and Rehabilitation from department of Orthopaedics for rehabilitation of FFD of hip. This cachectic lady was suffering pain in her left lower limb for five years and difficulty in walking for last two years. She was absolutely fine 5 years back. Suddenly severe pain started in her left knee and lower part of left thigh and general practitioner treated her with analgesics and cold therapy. At that time ASO titre was done to rule out rheumatic fever although none of the other clinical features of modified Jone's criteria were present. Then she was seen by a specialist physician who investigated her to rule out oligo-articular variety of juvenile spondylo-arthropathy. All the serological markers like rheumatoid factor, antinuclear factor, HLA B27 were negative. At that time she was also suffering from little bit of left hip pain. Subsequently she has been advised to go for physiotherapy. Different types of modalities including deep heat therapy and IFT were applied without any positive

outcome. Physiotherapist assessed that pain as a radiating pain from low back. After reviewing the x-ray (Fig 1) of lumbosacral spine the therapist suggested her to take an opinion from neurologist because there was spina bifida of fifth lumbar vertebra.

But her parents brought her to an orthopaedician's clinic. The orthopaedician diagnosed her as a case of Parthe's disease considering hip pain of a 17-year-old girl and thought knee pain as referred pain from her hip. That's why he advised her to go for a hip x-ray and prescribed only analgesics. Interestingly the x-ray (Fig 2) was reported as avascular necrosis of femoral head. In the meantime the pain was increasing in spite of so many consultation and little bit of deformity was also developed. The patient was then assessed by a neurologist who documented this pain as musculoskeletal problem. He sent the patient to another orthopaedic team who diagnosed her as a case of FFD and referred her to department of Physical Medicine and Rehabilitation.

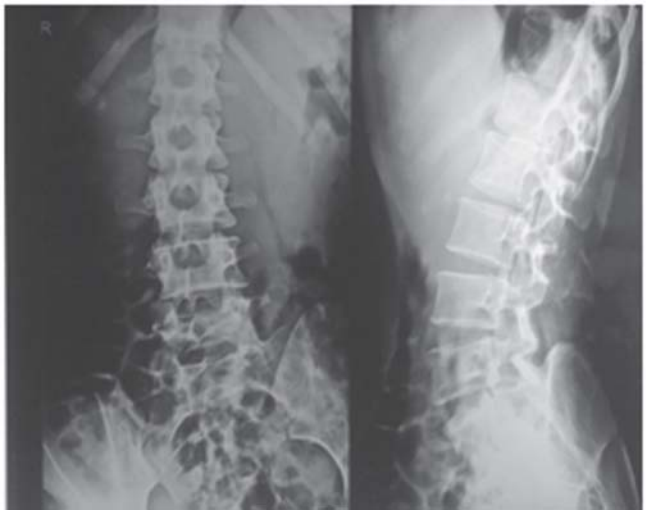

Fig 1

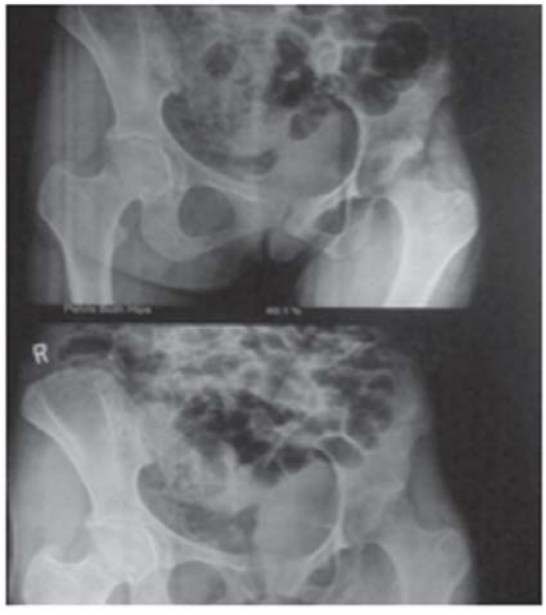

Fig 2

Eventually the patient visited PMR OPD with FFD of hip and knee. At that time pain score was minimal without any constitutional symptoms, palpable lymphadenopathy, positive respiratory signs. She was cachectic with history of weight loss, anorexia but no vomiting. She had no diahrroea, palpitation, tachycardia, goitre. On goniometric examination her knee range of motion (ROM) was 45-75 degree and hip ROM was 30-60 degree. There was no clearly palpable mass anywhere in her body.

Keeping in mind about hip pain, FFD, cachexia, weight loss a differential diagnoses of tuberculosis infection and neoplasia in or around the hip were considered. For confirmatory diagnoses routine blood test, chest x-ray, Mantoux test and a repeat x-ray of hip and knee were advised. As per her investigation reports she had mild anaemia and raised ESR without any lymphocytosis or lung abnormality and Mantoux positivity. Interestingly little periosteal reaction of femur was noted in her x-ray (Fig 3).

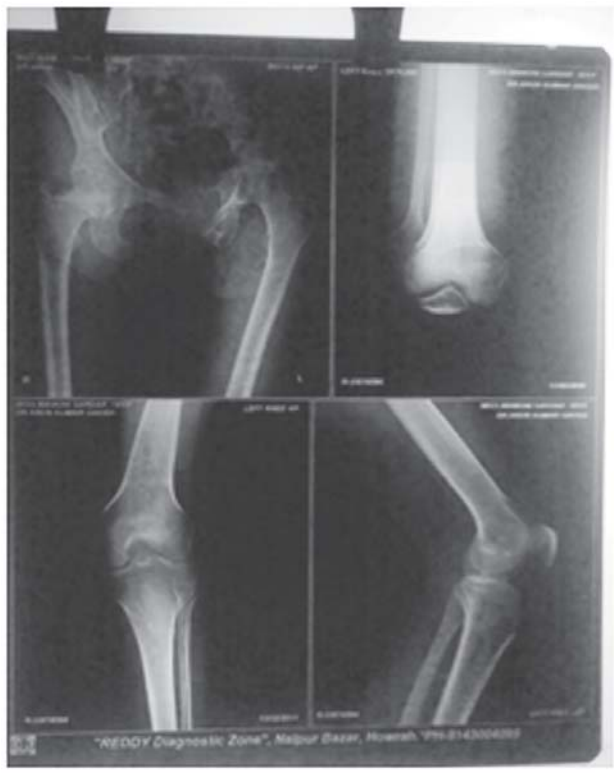

Fig 3

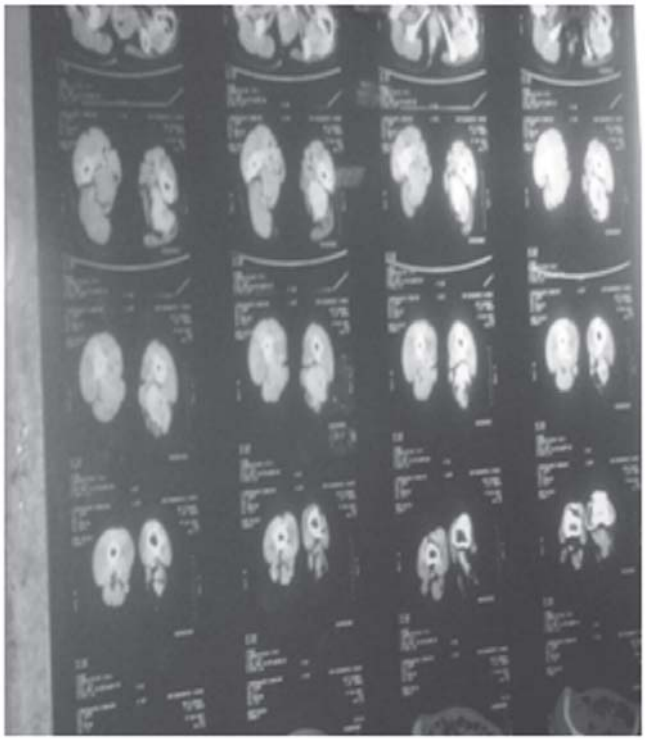

Fig 4

Fig 5

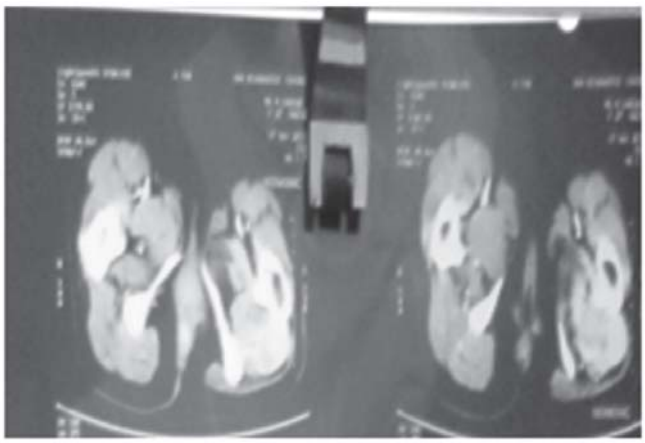

Fig 6

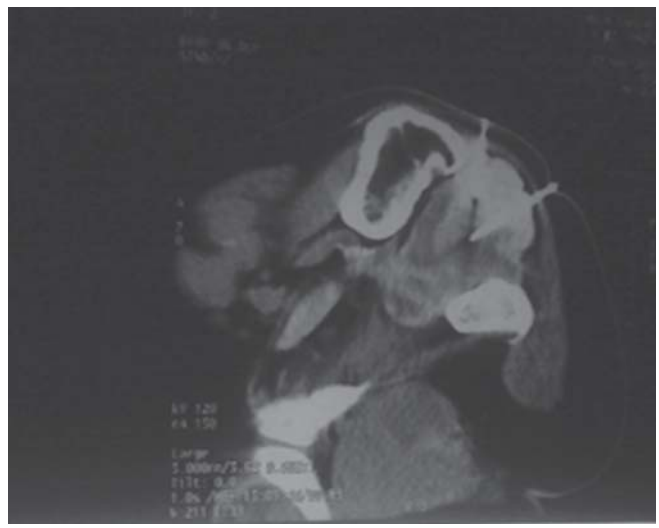

Fig 7

That's why a CT scan and CT guided FNAC were advised to rule any neoplasia. In CT scan (Figs 4-6) a big mass was seen involving gluteal and thigh muscle of her left side with bit of involvement of periosteum. Even the mass was extended up to the left sphincter ani muscle. Fortunately this young girl was continent throughout the disease course. According to CT scan report there was strong possibility of soft tissue sarcoma of the left lower limb.

CT guided FNAC (Fig 7) from the mass picked up the diagnises. After thorough review of the pathological slides (Figs 8–9) it was seen that multiple clumps of benign spindle cells having blunt nuclei without significant mitotic activity confirming histopathologically as a case of benign fibrous histiocytoma. After that the patient was sent to surgical team for wide local excision of the mass. At last patient became pain free and independent with post-surgical rehabilitation programme.

Discussion:

Benign fibrous histiocytoma is a rare entity that was first described by Dahlin in 1978. This lesion occurs most

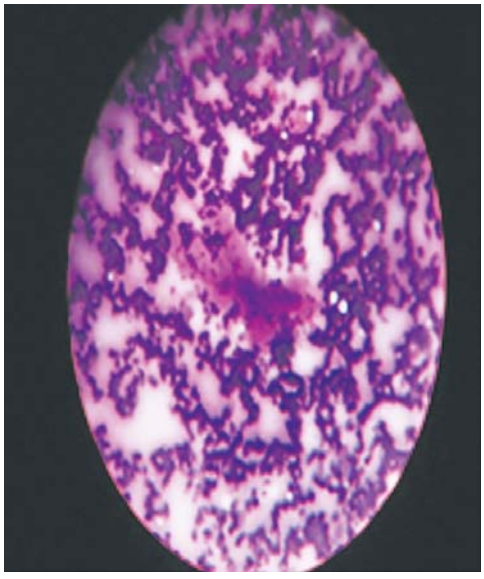

Fig 8

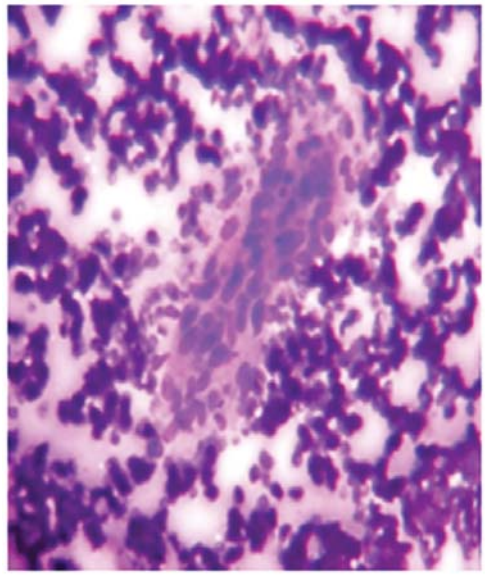

Fig 9

frequently in soft tissue and less often in bones. Benign fibrous histiocytoma may occur in the diaphysis or epiphysis of long bone or in the pelvis.[3] Common bones involved are femur, tibia, humerus etc, according to Oxford text book of Orthopaedics and Trauma.[4] In this young girl the mass originating from the glutei and hamstring evaded femur. According to the literature it may occur in any age group though most common age of presentation is 30 to 40 years.[5] Our patient presented at much younger age group. Although it may present in much earlier stage of life even in first year.[6] Benign fibrous histiocytoma is radiologically seen as well-defined lytic expanding lesion with little periosteal reaction. In contrast to non-ossifying fibroma this lesion is considered as true neoplasm. Because of its tendency for local recurrence extended curettage or wide excision is recommended.[3] Preoperative assessment of neurovascular entrapment and involvement of muscles or compartments and expected residual functional capacity of the limb after surgery are very helpful for overall prognoses of the patient.

Conclusion:

Although benign fibrous histiocytoma is a very rare clinical condition but may be a cause of FFD of hip and knee. Cross checking of clinical diagnoses and investigations for confirmatory diagnosis is an integral part of rehabilitation process.

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Original Article

Long Term Assessment of Neurogenic Bladder Following Myelopathies by Repeat Urodynamic Study and Correlation with Neurological and Functional Recovery

Anupam Gupta[1], Arun B Taly[2]

Abstract

Study Design: Prospective follow-up study

Objective: To assess neurogenic bladder following traumatic and non-traumatic myelopathies during inpatient rehabilitation by performing urodynamic study (UDS). Procedure repeated at least after 12 months follow-up to observe any change in the bladder behaviour and correlation between neurogenic bladder and neurological and functional recovery of the patients during the same period.

Setting: Rehabilitation unit in university tertiary hospital.

Methods: Thirty-one patients (24 males) with myelopathies (23 non-traumatic and 8 traumatic), mean age of 31.2±11.9 yrs (8-65 yrs) admitted for inpatient rehabilitation. All had neurogenic bladder with initial UDS suggestive of 17 patients with overactive detrusor and 14 with underactive/normal detrusor. Management advised accordingly. After minimum 12 months follow-up (12-23 months, 15.5±3.5), UDS was repeated.

Results: Significant change ($p<0.001$) in detrusor behaviour observed comparing initial and follow-up UDS findings. During both occasions there was no significant correlation ($p>0.05$) between bladder behaviour and neurological recovery (using American Spinal Injury Scale-ASIA) and functional recovery (using Barthel Index scale-BI). Significant functional recovery ($p<0.001$) observed between admission and discharge and discharge and follow-up in patients according to BI scores. Neurological recovery was significant between admission and discharge ($p<0.001$) but insignificant ($p>0.05$) between discharge and follow-up according to ASIA scale.

Conclusions: Detrusor behaviour following myelopathies is dynamic and not dependent on neurological and functional recovery. Repeat UDS is essential at regular intervals for effective management of neurogenic bladder and to avoid urinary complications.

Key words : **Myelopathies, urodynamic study, neurological and functional recovery.**

Author's affiliations:

[1] MBBS, MD (Physical Medicine & Rehabilitation), Associate Professor, Neurological Rehabilitation Division, Department of Psychiatric & Neurological Rehabilitation, National Institute of Mental Health & Neuro Sciences (NIMHANS), Bangalore, INDIA

[2] MBBS, MD, DM (Neurology), Professor of Neurology & I/C Head, Neurological Rehabilitation Division, Department of Psychiatric & Neurological Rehabilitation, National Institute of Mental Health & Neuro Sciences (NIMHANS), Bangalore, INDIA

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Correspondence :

Dr. Anupam Gupta, MD

Email ID: drgupta159@yahoo.co.in

Phone No.: +91 80 26995282, Fax No.: +91 80 26564830

Mobile No.: +91 8762689540

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Background:

The National Spinal Cord Injury Statistical Centre (NSCISC) estimated the prevalence of spinal cord injury about 755 per million population, with a range of 679–870 per million population and the annual incidence of spinal cord injury to be approximately 40 cases per million population or approximately 11,000 new cases each year.[1-3] In one of study conducted in this centre, 60% of the spinal cord injury (SCI) admitted patients had non-traumatic spinal cord lesions.[4]

Spinal cord injuries are well known to cause neurogenic bladder dysfunction with patients having urinary complaints. Urinary continence and the volitional control of voiding influence a SCI patient's potential for independence both at home and at workplace and the ability to function effectively. After SCI dynamic status

of the bladder/urethra depends on the level, extent and completeness of the lesion.[5]

Causes of mortality after SCI have changed in the last 5-6 decades from being primarily due to urinary tract diseases to increasing numbers of deaths from cardiovascular diseases and respiratory complications.[6-9] Urodynamic testing of bladder and management based on the findings has been one of the methods suggested to have contributed to reduce mortality and morbidity following myelopathies.[10]

There is consensus among the medical professional dealing with SCI cases that all such patients should undergo urodynamic evaluation with the initial study done after the patient has recovered from the initial spinal-shock phase. Management of the urinary tract in SCI individuals should be based on urodynamic principles and findings rather than on the neurologic history.[11,12]

Urodynamic study (UDS) has been considered the ''gold standard'' for evaluating bladder and sphincter function and for documenting the effectiveness of new drugs or other treatment modalities. UDS is recommended to be conducted at regular intervals after SCI to evaluate lower urinary tract function and to prevent upper and lower tract complications in SCI patients. Ideally it should be done once a year following SCI in the initial 5 years post lesion and after that it can done once in 2 years provided the patient continues to have neurogenic but stable bladder.[13] Limited studies have been done in the past to assess long term behaviour of neurogenic bladder following both traumatic and non-traumatic myelopathies.[10,12,14] Present study was conducted to assess neurogenic bladder following traumatic and non-traumatic myelopathies during inpatient rehabilitation by performing UDS. Procedure was repeated at least after 12 months follow-up to observe any change in the bladder behaviour by repeat UDS during this period and to see if there is any correlation between neurogenic bladder according to UDS and neurological and functional recovery of the patients during the same period.

Materials and Methods:

This prospective, follow-up study was conducted in the rehabilitation unit of the university tertiary research hospital. Approval from the institute's internal ethics committee was taken. The study was conducted over a period of 2 years (between April 2009 and March 2011) and included both traumatic and non-traumatic SCI patients admitted for inpatient rehabilitation. Patients consented to participate in the study, had monophasic spinal insult with injury in the cervical, dorsal and lumbar region and neurogenic bladder were included. Patients undergoing UDS for the first time and reported minimum 1 year after initial admission were only included. Patients with recurrent myelopathies, no urinary complication and doing voluntary micturition were excluded from the study.

MRI scan was done to ascertain level and type of lesion in the spinal cord in all the patients. Detailed neurological examination including sacral examination done to ascertain type and severity of injury. American Spine Injury Association (ASIA) classification was used to determine neurological level and functional abilities and were assessed using Barthel Index (BI) score.

All patients underwent UDS using multichannel urodynamic equipment-Primus (Lifetech Biomedica), which included recording of events during both filling and voiding phases. Normal saline used as medium of filling. Sphincter-electromyography was done in all patients. The bladder management of the patients was based on UDS findings.

Patients were called for regular follow-up. Thirty-one such patients reporting till minimum of 12 months and undergoing repeat UDS after minimum of one year were included in this study. Their neurological and functional status in the follow-up was recorded. Antimuscarinic medications were discontinued 1 week before performing repeat procedure and bladder management was based on UDS findings. Difference in bladder (detrusor) behaviour according to initial and follow-up UDS was analysed. Repeat UDS findings were correlated with neurological and functional recovery at 1 year follow-up to observe any definitive pattern.

Data Analysis:

Analysis was done using SPSS 15.0 version. Descriptive statistics included frequency, mean and standard deviation for quantitative variables such as age, duration of illness, duration of stay and BI scores.

Paired Student's t-test was used for the assessment of functional recovery using mean BI scores at admission, discharge and follow-up. The Wilcoxon non-parametric test was used for the assessment of neurological recovery by comparing admission, discharge and follow-up ASIA scale scores. Same test was used to assess change in detrusor behaviour between initial and follow-up UDS. Spearman correlation co-efficient test was used to observe correlation between detrusor behaviour and neurological and functional recovery.

Results:

Study included 31 patients (24 males, 7 females). During the study period 92 patients with traumatic or non-traumatic myelopathies were admitted for inpatient rehabilitation. The age of the patients varied from 8 to 65 years (31.2±11.9 years). Duration of illness at the time of initial admission ranged from 1 to 9 months (2.8±2.4 months). Mean duration of stay in the rehabilitation unit was 65.9 days (range 14-281 days, SD 60.7). Aetiology of myelopathy is reported in Table 1.

Ten patients (32.3%) had cervical myelopathy, 14 patients had dorsal myelopathy with lesion between D1-D6 in 4 patients (12.9%) and lesion between D6-D12 in 10 patients (32.3%). Seven patients (22.6%) had lumbar myelopathy.

Majority of the patients (27/31-87.1%) had urinary complaints in the form of increased frequency, urgency and urge urinary incontinence during initial admission/ after removal of indwelling catheter with 4 patients also had associated stress incontinence. Only 4 patients had complaints of hesitancy and straining to void with poor stream.

Bladder management after initial UDS was based on the detrusor behaviour according to findings. Sixteen patients were advised one or the other antimuscarinic (tolterodine, solifenacin, oxybutynin or propantheline), 4 patients advised adrenergic agonists along with behavioural and supportive measures to maintain balanced bladder and avoid any urinary complications.

Significant neurological and functional recovery was observed according to ASIA impairment scale and BI scale respectively during discharge as compared to admission scores ($p<0.001$). Similar trend was observed after minimum 1 year follow-up in the functional recovery with patients showing significant recovery as compared to discharge scores according to BI scale ($p<0.001$) (Table 2). Although there was trend for further neurological recovery during follow-up as compared to discharge but it didn't reach significant level ($p=0.06$) using ASIA scale.

At mean 15 months follow-up (12-23 months) UDS was repeated. Twenty-four patients were started on anti-muscarinic medications along with behavioural and supportive management according to repeat UDS findings. Significant change in detrusor behaviour

Table 1: ***Aetiology of Myelopathies***

Sl. No.	Aetiology	No. of cases	%
1	Traumatic spinal cord injury	8	25.8
2	Tuberculosis	4	13.0
3	Acute transverse myelitis	7	22.6
4	Arteriovenous malformation	4	13.0
5	Primary tumours	2	6.5
6	Prolapsed intervertebral disc	3	9.7
7	Ossified posterior longitudinal ligament	3	9.7
		31	**100**

Table 2: ***Neurological and Functional Recovery in Patients during Study Period***

Neurological recovery – ASIA scale					
	Admission	**Discharge**	**p value (Admission vs discharge)**	**Follow-up**	**p value (Discharge vs follow-up)**
A	19	6	<0.001	6	0.06
B	2	0		0	
C	6	8		4	
D	0	13		15	
E	0	0		2	
Cauda equina	4	4		4	
Functional Recovery – Barthel Index score					
	(29.7±20.5) 0 - 85	(68.7±19.3) 20 - 100	<0.001	(80.5±18.5) 40 - 100	<0.001

Table 3: *Urodynamic Study (UDS) Findings:*

S. No.	Detrusor Type according to UDS	Initial UDS	Follow-up UDS	p value
1	Overactive Detrusor without sphincter Dyssynergy	14	21	<0.001
2	Overactive Detrusor out sphincter Dyssynergy	3	3	
3	Underactive Detrusor	13	5	
4	Normal Detrusor	1	2	

($p<0.001$) was observed when comparing follow-up UDS finding with initial UDS findings (Table 3). The management of bladder also changed accordingly. Although there was significant change in detrusor behaviour with time as also was the case with functional and neurological recovery but during both initial and follow-up UDS, no significant correlation was found ($p>0.05$) between detrusor behaviour and neurological and functional recovery in patients.

Discussion:

UDS help the clinician in advising the patient on the available choices of bladder management.[15] Majority of the patients in the present study had recovered from the spinal shock by the time they were admitted in rehabilitation unit. Performing UDS is very important as according to our experience, most of such patients are either encouraged by treating team to go for Crede's method or straining to pass urine once they are decatheterised in general practice. On the other hand there are patients who are still advised to do clamping of indwelling catheter and release it after a certain time period. Both practices are dangerous and jeopardise the safety of bladder in the short term and may cause complications in the upper urinary tract in the long term run as well.

Although majority of the patients in the present study had irritative urinary complaints, initial UDS was suggestive of overactive detrusor (with or without sphincter dyssynergy) in only 55% (17/31) of patients who were started on one of the antimuscarinic medications. Remaining patients were advised timed voiding and fluid restriction only. These findings again highlight the importance of performing UDS in all patients so that the detrusor behaviour can be documented and bladder managed accordingly.

Several studies have been conducted in the past to observe correlation between neurogenic bladder and neurological recovery in the patients. Shenot *et al*[16] (1998) in their study with myelopathy patients observed that patients with sacral sparing during initial 72 hours examination had much bright chance of doing voluntary micturition after 1 year whereas patients with complete injury were found to have different types of detursor at 1 year UDS follow-up and required assisted micturition. Another longitudinal study by Generao *et al*[17] (2004) on paediatric SCI patients for up to mean of 5.5 years (range 1 to 15.5) found that bladder characteristics change with time in majority of patients with growth and serial urodynamics confirm dynamic nature of detrusor behaviour. Our study's findings are similar to both these studies as significant change in detrusor behaviour was observed in follow-up with majority of patients with underactive detrusor initially were later observed to have overactive detrusor and their bladder management protocol also changed accordingly.

Significant neurological and functional recovery was observed in follow-up with nearly 80% of the patients (24/31) were ambulatory with or without orthoses and assistive devices. Whereas only 5 patients (16.1%) were doing voluntary micturition with 4 of them were on anti-muscarinic medication to control overactive detrusor. Patki *et al*[18] (2006) in their longitudinal study with myelopathy patients observed that despite relatively near total neurological recovery, patients with incomplete SCI have neuropathic bladder unless proved otherwise.[18] Similarly Nosseir *et al*[19] (2007) in their longitudinal study with myelopathy patients found that the treatment strategy of neurogenic bladder dysfunction in patients with SCI has to be modified in almost all patients for protection of the upper urinary tract and maintenance of continence, based on regular urodynamic follow-up. The findings of our study are similar to these studies although we did follow-up of patients for only up to 23 months with UDS repeated only once. In the repeat UDS, there was significant change in detrusor behaviour (as evident from Table 3) but no significant correlation was found between detrusor behaviour and functional and neurological recovery ($p>0.05$) in the SCI patients conforming to the findings of earlier studies.

Wyndaele[20] (1997) in a study with SCI patients tried to correlate level of spinal cord lesion with bladder behaviour according to UDS and observed significant correlation between neurological examination and UDS findings, with higher lesions (above D10) correspond more with an overactive detrusor and somatic motor activity, lower lesions more with areflexic detrusor. With a lesion between thoracic 10 and lumbar 2 as many reflexic as

areflexic detrusor were found. The objective of our study was different but the mentioned study again highlights the point that there is no consistent correlation between bladder behaviour and level and severity of spinal cord lesions.

Conclusions:

Detrusor behaviour following myelopathies is dynamic and not dependent on neurological and functional recovery. Repeat UDS is essential at regular intervals for effective management of neurogenic bladder and to avoid both upper and lower urinary tract complications.

Conflict of Interest:

The authors declare no conflict of interest.

Carry home message:

Repeated urodynamics is essential for management of neurogenic bladder following myelopathies.

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Case Report

An Interesting Form of Osteochondrodystrophy –A Case Report of a Family

Jagannatha Sahoo[1], P Hemanta Kumar[2], G Jagadeesh[3]

Abstract

A 12-year-old boy presented with progressive increasing deformity of both knee joints since last 10 years. The radiograph of femor, tibia and phalanges showed different dysplastic changes of epiphysis. It showed a different skeletal dysplastic nature to multiple epiphysial dysplasias. Silfverskiöld described similar types of skeletal dysplasia.

Key words : **Multiple epiphyseal dysplasia (MED), osteochondrodysplasia, epiphysis.**

Introduction:

Multiple epiphyseal dysplasia (MED), common type of osteochondrodysplasia, is an uncommon inherited condition resulting in the formation of abnormal epiphyses.

The term chondro-osteodystrophy was first given by Brailsford in 1929 to a disorder of the skeleton which manifested itself during the first three years of life. In the same year Morquio described "a form of familial osseous dystrophy" in four members of a family of five[1]. The disorder was manifested as multiple areas of abnormal growth and ossification of the epiphysis. It affected predominantly hips, knees, ankles, and wrists. Although present at birth, symptoms of MED do not develop until years later[2]. The radiologic finding of multiple abnormal epiphysis ossification centres is diagnostic. The affected epiphyses are small, irregular, mottled and/or fragmented, irregularly mineralised, late ossifying, and usually found in the long bones of the lower limbs. The femur are irregular symmetric, and commonly have early acetabular changes and mild metaphyseal flaring.

Case Report:

A 12 years boy came to our outpatient department with chief complaint of genuvalgum. There was history of similar deformity of his two male siblings. Among his three elder siblings, eldest one died at the age of 4 years due to unknown disease. The second elder sibling was normal and not suffering with similar type of deformity. But the third elder and younger siblings affected with the similar deformity (Fig 1). Parents noticed the progressive deformity in the entire above mentioned child after 2 years when they start walking.

On examination we found large head, base of nose

Author's affiliations:
Balaji Institute of Surgery Research and Rehabilitation For the Disabled, Tirumala Tirupati Devastanam, Tirupati, AP-517507
[1] MBBS, DNB(physical medicine & rehabilitation), DNB(Ortho)
[2] MBBS, MS(Ortho), Asst. Professor (Ortho)
[3] MBBS, MS(Ortho), Director

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Correspondence:
Dr Jagannatha Sahoo,
MBBS, DNB(PMR), DNB(ORTHO)
Plot No-1144, Shrabani Residency, Gadasahi
Nayapalli,Bhubaneswar-751012, Odisha
PHONE: 09437081814(M), 0674 2561814
E-MAIL: jagannatha_sahoo@yahoo.co.in
Received on 11/09/2011, Accepted on 17/11/2011

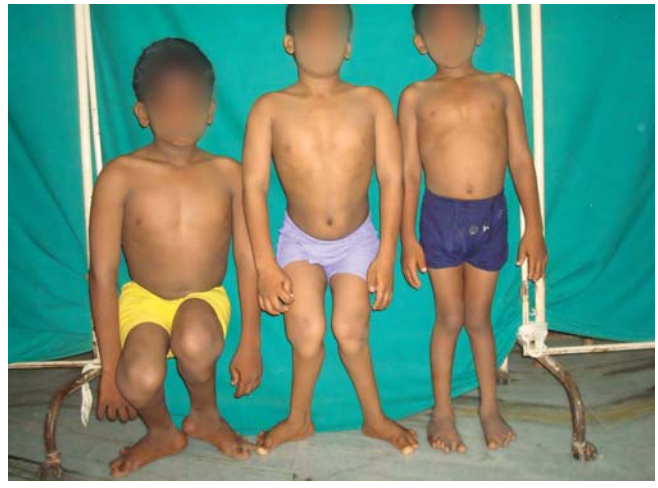

Figure 1

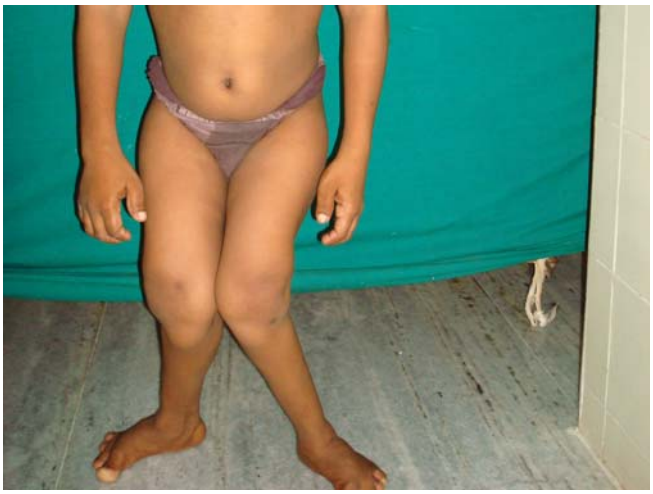

Fig 2

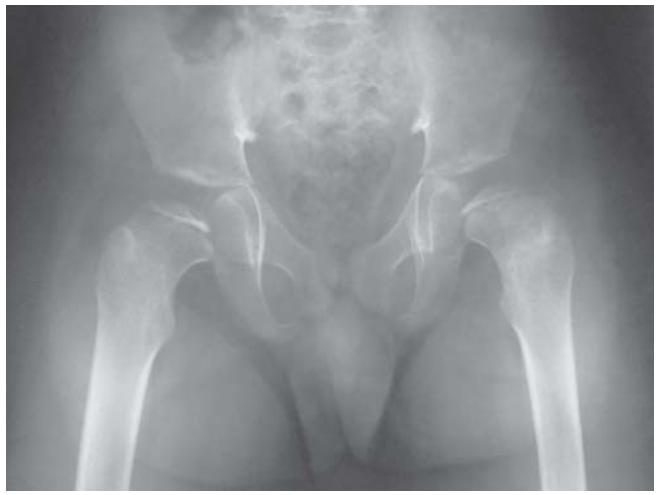

Fig 3

flattened, broad and short phalanges, hip flexion deformity, laterally displaced patella, genuvalgum with externally rotated tibia, broad and shortened toe, 4th toe overriding 3rd and 5th toes and arm was comparatively shorter than forearm (Fig 2).

On biochemical analysis : Blood group B +ve,TC-8150/cmm, DC-W-54, L-40, E-6, Hb%-10.4, ESR- 26/hour, BT-0′46″, CT-5′12″, calcium-10.2mg/dl, phosphorus-4.4mg/dl, alkaline phosphatase-192IU/l, T4-105ng/ml, TSH-0.7μIU/ml, urine-RE/ME -normal.

On radiological analysis there were dysplastic changes seen in epiphysis of both ends of femur, both ends of tibia, both wrist joints. All phalanges were short (Figs 3-5).There was fish mouth appearance of spine seen in lateral view (Fig 6). In orthoscanogram there was multiplanar deformity seen in both femur and tibia (Fig 7).

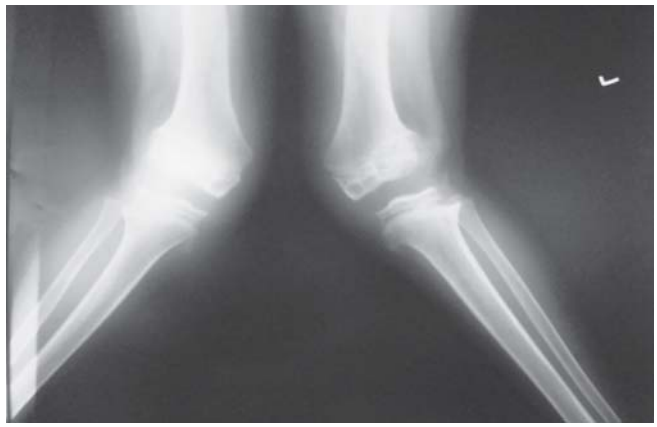

Fig 4

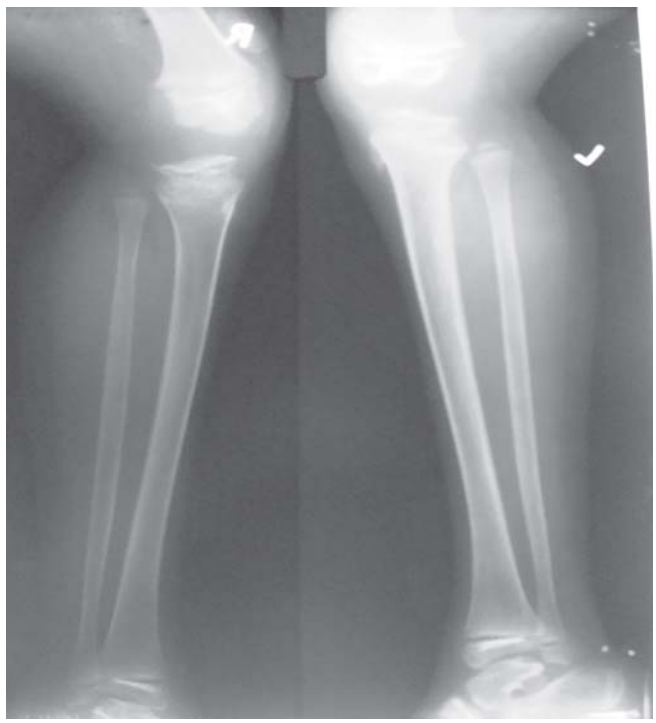

Fig 5

Discussion:

In 1925 and 1926 Silfverskiöld[3] described a total of four patients among five had different skeletal dysplasia. As the disease pictures vary greatly, it was impossible to give any useable definition. Silfverskiöld's patient was an eleven-year-old boy in whom the symptoms included disproportionate dwarfism with short legs, large head, flattened nose, broad chest and large trochanters. Hans Fredrik Helweg-Larsen (1917-1969) and Mørch later reported the syndrome in a family.

Apart from the description of Silfverskiöld and Hans Fredrik Helweg-Larsen, we noticed fourth toe overriding 3rd and 5th toes (Fig 8), Arm was shorter compared to forearm, laterally displaced patela and 4th metatarsal and phalanx were shorter than others in our cases.

Murphy *et al* (1973)[4] observed that roentgenograms showed well recognised typical features of the disease:

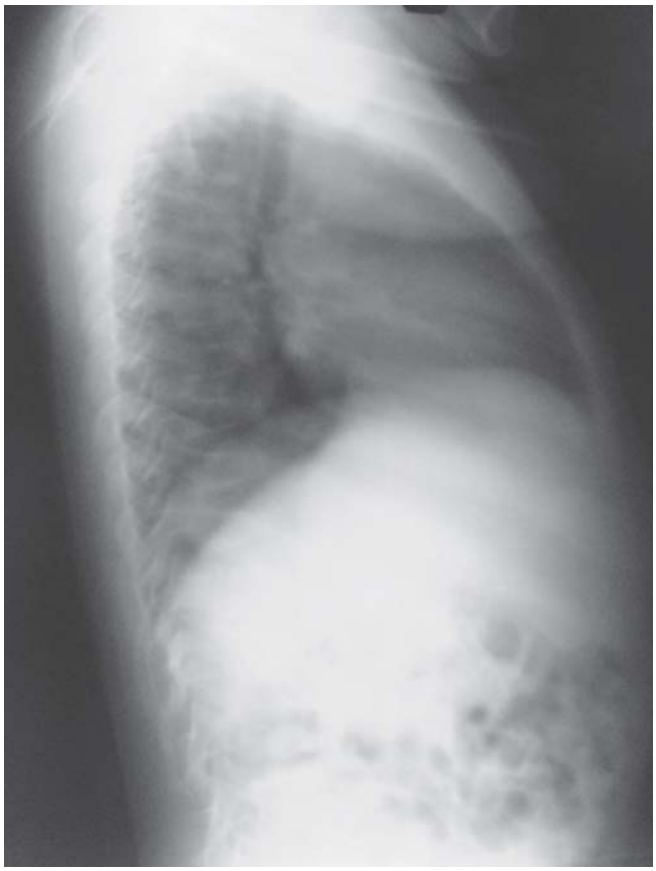

Fig 6

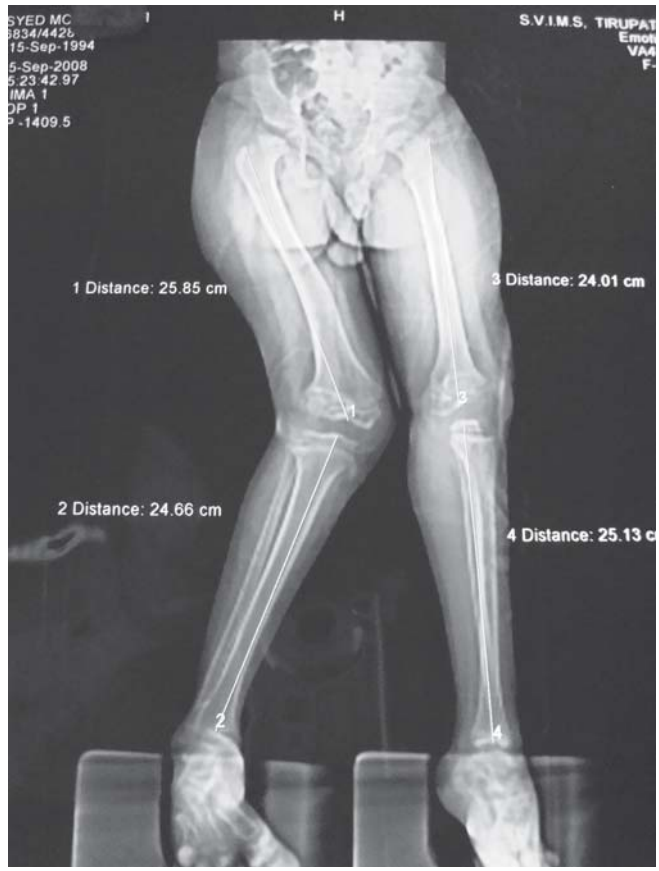

Fig 7

short metacarpals, widening of the distal radial metaphysis, the flattened humeral head with shallow glenoid fossa, and oblique acetabulum with flattened femoral head. In addition, a frequent feature was a slanting of the ankle joint mortise found in ten of twelve patients.

Treble *et al* (1990)[5] reported that the development of ossific nucleous of the femoral head was abnormal. The nucleus was small, misshapened and showed abnormal pattern of ossification. In our case the acetabular components of the hip joint were comparatively normal and the femoral head misshapened and flattened symmetrically.

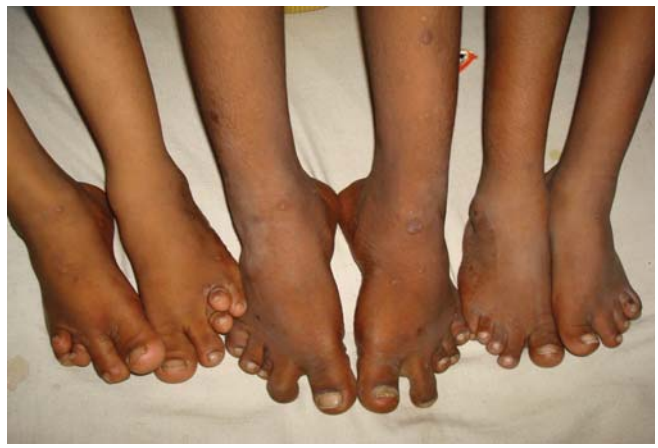

Fig 8

Conclusion:

These cases were presented to us as bilateral symmetrical involvement of all the epiphysis along with above findings. These cases were unusual form of osteochondrodystrophy with skeletal defects with hereditary involvement.

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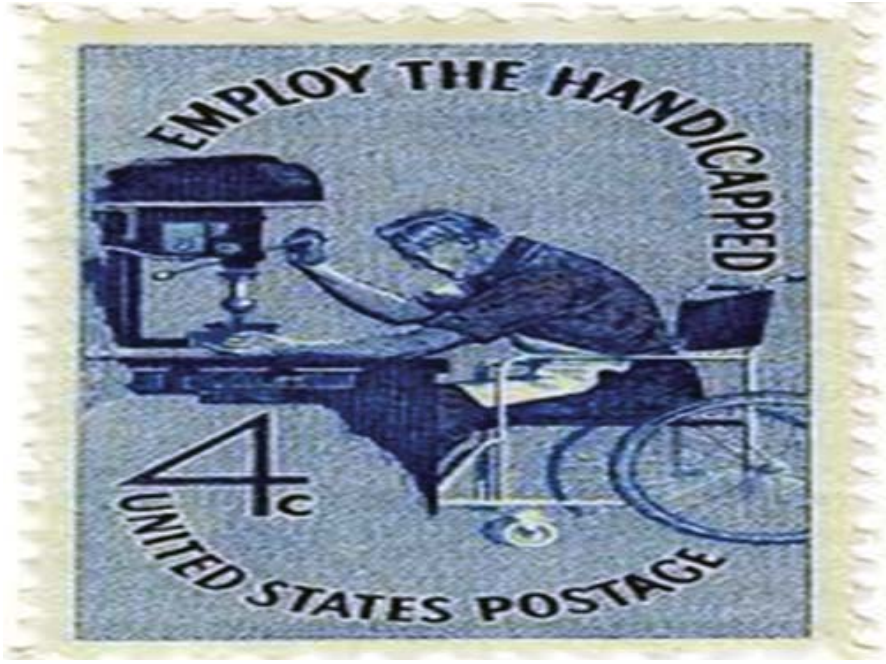
Medical Philately



US Stamp of 1960

The “Employ the Handicapped” issue commemorated the meeting of the 8th World Congress of the “International Society for the Welfare of Cripples” in New York City in 1960. Over 3,000 delegates attended the meeting. This society was later renamed the “International Society for the Rehabilitation of the Disabled” and more recently to “Rehabilitation International”.

Review Article

Role of Platelet-Rich Plasma (PRP) in Chronic Tendinopathy

Manoj Sivan[1], *James Brown*[2]

Abstract

Platelet-rich plasma (PRP) is increasingly being used in the treatment of chronic tendinopathy in both sporting and sedentary population. It is rich source of various growth factors and is believed to stimulate and enhance the tissue repair process in tendinopathy. The current literature has six clinical studies (excluding single case studies) which have investigated the effect of PRP in tendinopathy of various tendons. The evidence so far is inconclusive in demonstrating the superiority of PRP over placebo injection or eccentric loading exercises. Future research should focus on conducting randomised controlled studies to establish the clinical effect and support or refute the current widespread use of PRP in chronic tendinopathy.

Key words : **Platelet-rich plasma, tendinopathy.**

Tendon related disorders account for 30-50% of sport related injuries[1]. The term "tendinopathy" refers to a clinical triad of pain, swelling and decreased activity[2]. It was termed tendinitis in the past in the belief that there was an inflammatory component to the condition but it has been shown not to be the case in histological studies. The understanding now is that of collagen disruption with increase in ground substance matrix with abnormal tissue repair and degeneration[3].

Among all the available conservative treatment approaches, the best evidence so far is for eccentric loading exercises, which is shown to have positive effect on tendon collagen synthesis and accelerating the reparative process[4]. Platelet-rich plasma (PRP) is a relatively new treatment approach and is now being widely used in the treatment of chronic tendinopathy both in the sporting and sedentary population. The underlying hypothesis is that platelets derived from whole blood (using a centrifuge system) (Fig 1) is a rich source of various growth factors including platelet-derived growth factor, transforming growth factor-β, vascular-derived endothelial growth factor, epithelial growth factor, hepatocyte growth factor and insulin-like growth factor which can stimulate and hasten the tissue repair process in tendinopathy[5].

The evidence in this new treatment method has so far been inconclusive. Some recent review articles which included laboratory and clinical studies in tendinopathy

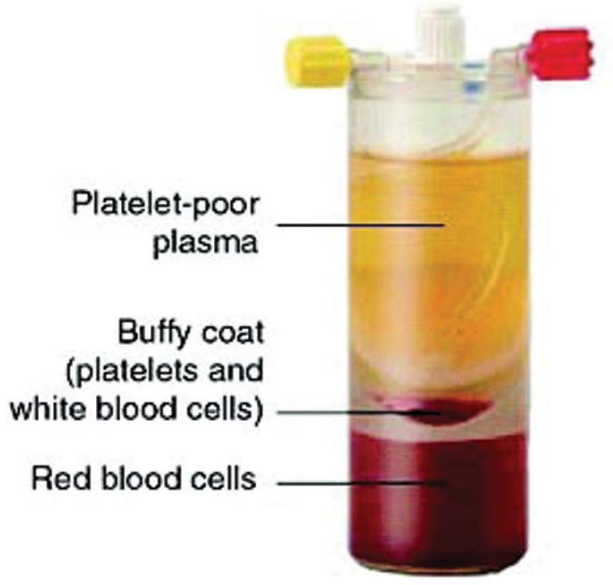


Fig 1 – *Post-centrifuge blood sample layers*

Author's affiliations:
[1] MBBS, D Ortho, MRCS, *Specialist Registrar in Rehabilitation Medicine, Leeds Teaching Hospitals NHS Trust, Leeds, United Kingdom*
[2] MBChB, MSc, FFSEM. *Consultant in Musculoskeletal and Sports Medicine, Leeds Primary Care NHS Trust, Leeds, United Kingdom*

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Correspondence:
Dr Manoj Sivan, Department of Rehabilitation Medicine, Leeds General Infirmary, Leeds LS1 3EX, United Kingdom.
Email: M.Sivan@leeds.ac.uk

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concluded significant improvement in pain and functional activity with use of PRP[6,7]. However, the studies included in these reviews lacked high-quality randomised controlled clinical trials (RCT). Since then, there has been one recent RCT in Achilles tendinoapthy with one-year follow-up data which showed no significant effect of PRP over saline injection[8]. The International Olympic Committee (IOC) consensus paper on the use of PRP in sports medicine concludes that there is a lack of convincing evidence to support its use in clinical setting and calls for more research in basic science and robust clinical trials to test efficacy[9].

This article aims to review the available clinical studies involving the use of PRP to treat tendinopathy. Clinical studies in both sporting and sedentary populations with well-defined outcome measures utilised to measure change have been included in this review. This review will help future research in terms of type of study, participant criteria, sample size, PRP type, imaging and outcome measurement needed to establish the real clinical effect of this treatment.

Summary of Studies:

Six studies were deemed suitable for inclusion in this review. The studies which were not included were single case reports and cases which have investigated the role of augmenting surgical repair of tendon with PRP injection. The methodology of each included study was analysed to derive level of evidence based on recommendations from the Oxford Centre for Evidence-Based Medicine[10]. The evidence levels are I: High- quality Randomised Controlled Trial (RCT) or systematic review of level-I RCTs; II: Lesser-quality randomised controlled trial (eg, <80% follow-up, no blinding, or improper randomisation) or Prospective comparative study; III: Case-control study or Retrospective comparative study; IV: Case series; V: Expert opinion. Table 1 summarises the methodology and results of the studies included in this review.

Discussion:

The results suggest the evidence so far is inconclusive for recommending use of PRP in routine practice. The best evidence so far is provided by the RCT by de Vos *et al*[8] which suggests no enhanced effect over saline injection in Achilles tendinopathy. However, the RCT by Peerbooms *et al* suggests that PRP had a significant enhanced effect when compared to steroid injection in lateral epicondylitis (tennis elbow). This is supported by another level II study in medial or lateral elbow epicondylitis (golfers or tennis elbow). Finally, two studies from same research group (one level IV study and the other level II study) suggest beneficial effect in patellar tendinopathy.

One could argue that PRP seems to be more effective in non-body weight bearing tendons (common wrist flexor/extensor tendon origin) than body weight bearing tendons (patellar/achilles). In fact, the effect sizes of the intervention have been large with 93% improvement in the study by Mishra *et al*[6] and 64% in the RCT study by Peerboons *et al*. The effect size in studies involving patellar tendon or achilles tendon have been smaller. The study in achilles tendinopathy showed no benefit over saline injection[8] and in patellar tendinopathy, only 54% improvement in EQ-VAS after repeated injections (three in a 6-week period) was observed[11].

There is considerable variation in the techniques used in preparation of PRP. There is no standardisation across trials in terms of centrifuge technique, speed and time of centrifuge, apparatus used, storage time and concentration of platelets in the injected PRP. This makes comparability between studies difficult. The IOC consensus statement suggests having a classification system for different PRPs as it might help comparing efficacy among different products[9]. The belief is that the amount and type of growth factors may vary in these different products.

The injection technique varies across the reviewed studies. Most authors used anatomical landmarks to inject PRP in the most tender areas of the tendon. The studies by de Vos *et al*[8] and Gaweda *et al* however used ultrasound guidance to inject PRP in areas of most hypoechogenecity within the tendon. de Vos *et al*[8] though used ultrasound for guiding injection, did not report on the follow-up ultrasound findings. This might have been beneficial to know as this is a level I study and it did not show any difference in outcome between the two groups. Thickness of tendon, echogenecity and neovascularity are key features in ultrasound appearance of tendinopathy and are believed to be related to the severity of the tendinopathy.

All the studies had similar post-procedure rehabilitation programme which included initial rest to gradual buildup of eccentrically loaded exercises. Combining injection with eccentric exercises was utilised in most of the studies. The authors rightly defend that the outcome

Table 1: ***Summary of Methods, Results and Conclusion of Included Studies***

Evid-ence level	Year	Author	No of patients	Tendon (I) PRP	Inter-vention group	Control (C)	Outcome measures months	Follow-up	Outcomes (% improvement of PRP	Conclusion on effect
I	2010	de Vos *et al*	54	Achilles	1 inj-ection	1 Saline injection	VISA-A (0-100)	6	Mean VISA-A change: 7.8 to 21.7 in I group, 4.6 to 20.5 in C group	Same effect
I	2010	Peerbooms *et al*	100	Elbow lateral	1 inj-ection	1 Steroid injection	VAS (0-100) DASH	12	Mean VAS change: 65.8 to 50.1 in C group (24%), 70.1 to 25.3 in I group (64%)	Better effect
II	2006	Mishra *et al*	20	Elbow medial and lateral	1 inj-ection	1 LA inj-ection	VAS (0-100)	25	Mean VAS change: 80.3 to 5.7 (93%) in I group, values not reported for C group	Positive effect
II	2010	Filardo *et al*	31	Patellar	3 inj-ection	Exercise therapy	EQ-VAS (0-100)	6	Mean EQ-VAS change: 52.7 to 78.3 (54%)	Better effect
IV	2010	Gaweda *et al*	14	Achilles	1 inj-ection	–	AOFAS VISA-A	18	Mean AOFAS change: 55 to 96, Mean VISA-A change: 24 to 96	Positive effect
IV	2009	Kon *et al*	20	Patellar	3 inj-ection	–	EQ-VAS (0-100) SF-36	6	Mean EQ-VAS change: 57 to 82 (58%)	Positive effect

cannot be attributed solely to the exercise component of the treatment as the included patients had failed treatment with eccentric exercises prior to the PRP injection. The role of orthosis in the rehabilitation protocol is debatable. The study by Gaweda *et al* had an additional orthosis (heel lift in shoe) to offload the achilles tendon which was not used in the RCT by de Vos *et al*.[8]

Another unknown factor is the ideal time for PRP treatment. It is difficult to conclude from these trials whether this treatment is better suited for treatment of refractory tendinopathy or as an add-on to eccentric exercise rehabilitation programme early in the treatment of the condition. Most of the included studies had patients with failed conservative treatment methods for at least 6 months prior to PRP treatment. It is not known whether including patients at an early stage will lead to improved benefit from the treatment. It might also be interesting to conduct a trial comparing PRP and eccentric exercises, however the trial cannot be blinded and would be biased by the placebo effect of the injection. Such a trial would have to include a crossover design to overcome such a treatment bias.

There is lack of uniformity in outcome measures used in the studies. The VAS has been used in few studies to capture change in pain intensity. The tendon specific outcomes like VISA-A have been used by de Vos *et al*[8] and Gaweda *et al*. SF-36 has been used in the study by Kon *et al*, to demonstrate change in general well being. Only one study reports on ultrasound appearances of tendon. In the study by Gaweda *et al*, the changes seen with improvement in pain were reduced thickness of tendon and resolution of hypoechoic areas within the tendon. Interestingly the study noted increased neovascularity within and around the tendon with reduction of pain, which is normally not the case with successful treatment of tendinopathy.

Recommendations for Future Research:

In summary, there is lack of substantial evidence to support use of PRP in routine clinical practice. There is need for high-quality RCTs to establish the clinical effect and support the current widespread use.

Two study designs could be considered for future research. First, a double-blind placebo-controlled RCT comparing PRP and saline injection. This will add strength to the findings observed in study by de Vos *et al*[8]. Second, a cross-over randomised controlled trial comparing PRP and eccentric exercises, as there is no study so far which compares PRP to the well proven treatment of eccentric exercises.

The participants should be a homogenous population with similar duration of symptoms and clinical diagnosis. This can include the ultrasound appearance of tendon in terms of presence or absence of discontinuous areas (or defects) in the tendon architechture. The influence of tendon anatomy and biomechanics (upper versus lower limb) on response to PRP needs to be explored.

Using ultrasound to record tendon architectural changes and guiding the injections can help better understanding the tissue response to PRP. No study so far has robust follow-up ultrasound appearance of tendon response to PRP injections.

The preparation and concentration of PRP must be standardised to enable comparability across different studies. Particular emphasis must be placed on platelet concentration, recovery and activation time.

The rehabilitation protocol must be standardised and evidence-based. The type and duration of exercises and equipment used needs to be tendon-specific and uniform for all patients in the study.

Appropriate outcomes to capture pain, functional limitation and return to sport with a long-term follow-up are desirable. A combination of VAS, VISA-A (AOFAS for non-sporting population) and return to sport (SF-36 for non-sporting population) would be ideal to capture change in all domains of the health condition (achilles tendinopathy).

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Abbreviations

VAS	Visual Analogue Scale
VISA-A	Victorian Institute of Sports Assessment-Achilles
DASH	Disabilities of the Arm, Shoulder and Hand
AOFAS	American Orthopedic Foot and Ankle Society
EQ-VAS	EuroQol 5D VAS component
SF-36	Short Form- 36
LA	Local Anaesthetic

Original Article

Prevalence of Hemiplegic Shoulder Pain in Post-stroke Patients – A Hospital Based Study

Joy AK[1], Ozukum I[2], Nilachandra L[3], Khelendro Th[4], Nandabir Y[5], Kunjabasi W[6]

Abstract

Objectives: To study the prevalence of hemiplegic shoulder pain (HSP) and its association with other factors like age, sex, side of paralysis, type of brain lesion, muscle tone, degree of functional recovery in upper limb and glenohumeral subluxation (GHS).

Methodology: Prospective study based on all the hemiplegic 140 patients admitted in the physical medicine and rehabilitation ward in two consecutive years.

Tools: Assessment of HSP was done by using a structured questionnaire known as "Shoulder Q". Modified Ashworth scale (MAS) was used for spasticity assessment and functional independence measure (FIM) to document the severity of disability.

Follow-up at intervals of 1, 3 and 6 months from the date of discharge for all cases were attempted and even cases with at least one follow-up around 3 months were also included in the study. Analysis was done on 109 patients as 31 patients lost to follow-up.

Results: Out of the 109 patients, 61.5% were males with a mean age of 58.9 ± 10.9 years. Cerebral infarct represents 53.2% of patients. HSP was present in 47.7% (n= 52) of patients. The prevalence of HSP on left and right sides was comparable though involvement was more on the left side (58.8%). Glenohumeral subluxation was present in 32.7% (n=17) of 52 cases with HSP and 33.3% (n=19) of 57 cases without HSP. Mean FIM score at admission for patients with HSP was 54.5 ± 17.6 and 56.6 ± 19.5 among cases without HSP. Again, mean FIM scores at last follow-up were 80.0 ± 16.4 and 79.9 ± 18.9 respectively for both cases with HSP and without it. Among the compliers, patients with tone more than MAS=1 were more likely to develop HSP.

Conclusion: Prevalence rate of HSP among post-stroke hemiplegic patients admitted during two years was 47.7%. There was no association of HSP with factors like age, sex, side of paralysis, type of lesion and GHS. Correlation between HSP and muscle tone or degree of functional recovery was significant.

Key words : **Post-stroke hemiplegia, hemiplegic shoulder pain, glenohumeral subluxation, functional independence measure.**

Author's affiliations:
[1] MD, DNB, DSM, PhD, Professor
[2] MD, Medical Officer, Nagaland State Health services
[3] MD, Senior Registrar
[4] MBBS, Postgraduate Student
[5] MS, Associate Professor
[6] MS, DNB, Professor (Head)
Department of Physical Medicine & Rehabilitation
Regional Institute of Medical Sciences, Imphal

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Correspondence :
Dr. AK, Joy Singh, Professor
Department of Physical Medicine & Rehabilitation
Regional Institute of Medical Sciences, Imphal
Email: joyakoijam2@yahoo.com

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Introduction:

Stroke[1] is a world-wide health problem; with incidence ranging from 0.2 to 2.5 per thousand per year according to WHO Collaborative Study in 12 countries. It accounts for 20% of neurological admissions. Till date, in India there have been only a few community based studies for either prevalence or incidence of stroke; with one reporting a prevalence rate of 334/100,000 and an incidence of 73/100,000 in 1990[2]. Post-stroke hemiplegia is one of the most common causes of disability in adults. Prevalence of hemiplegia in South India is 56.9 per 100,000; as compared to 150 to 186 per 100,000 in the USA and Europe. Hemiplegic shoulder pain (HSP) is one of the commonest complications, occurring in about 20-72% of such patients with average figures ranges from 43 to 64%[3-10]. Kalichman and Ratmansky[11] reported

prevalence of HSP is approximately 22%-23% in the general population of stroke survivors and approximately 54%-55% among stroke patients in rehabilitation settings. It interferes with effective rehabilitation programme of upper limb in hemiplegia, thereby, compromising functional recovery and prolonging hospital stay. Good management of patients can reduce both the frequency and intensity of shoulder pain, improving functional outcome. There are not enough studies done to establish incidence of HSP with various risk factors[12].

The primary cause of HSP is not fully understood. According to involvement of anatomical structures, the causes of HSP may be due to; (i) rotator cuff tear, (ii) over-stretching of ligaments and muscles, like supraspinatus and deltoid, (iii) spasticity, (iv) muscle trigger points, (v) subacromial bursitis, (vi) tendinitis of long head of biceps tendon, (vii) adhesive capsulitis, (viii)impingement syndromes, (ix) reflex sympathetic dystrophy, (x) brachial plexopathy and (xi) central pain syndromes[13]. Shoulder subluxation, occurs at an early stage after stroke and is associated with subluxation of the shoulder joint and spasticity (mainly subscapularis and pectoralis). Dromerick *et al*[14] also implicate 2 vertical stabilisers of the humerus namely the long head of the biceps and the supraspinatus in early onset hemiplegic shoulder pain. Further, Huang *et al*[15] reported that the frequency of shoulder soft tissue injuries (85%) and HSP (67%) was higher in patients with hemiplegic shoulder with impaired sensation, spasticity, subluxation, and restricted rotation. Frequency of abnormal sonographic findings and shoulder pain and visual analogue scale score of HSP before discharge were significantly higher in the poor motor function group than in the good motor function group.

This study was aimed to find out the prevalence of HSP, degree of association between HSP and other factors like age, sex, side of paralysis, type of brain lesion, muscle tone, degree of functional recovery in upper limb and glenohumeral subluxation (GHS).

Materials and Methods:

A prospective study which included all the new hemiplegia patients in the age group of 40-80 years admitted in two years was conducted in the department of physical medicine & rehabilitation, Regional Institute of Medical Sciences, Imphal. Out of the total 140, analysis was done on 109 patients as twenty-seven were lost to follow-up and another four expired during the study period. Informed consent was taken before inclusion in the study.

Comatose patients, recurrent stroke, thalamic pain syndrome, comorbid conditions like diabetes, chronic obstructive lung disease, coronary artery disease, malignancy, severe arthritis of shoulder and recent fracture of humerus, clavicle etc, were excluded from the study.

Clinical diagnosis of stroke was confirmed by CT scan of brain in all the cases. Range of motion of the affected shoulder joint was measured by using a goniometer. Other important clinical tests for impingement, laxity of joint, tendinitis and rotator cuff lesions were also performed in appropriate cases. Modified Ashworth scale (MAS) was used for spasticity assessment and functional independence measure (FIM) to document the severity of disability as well as the outcomes of the rehabilitation treatment.

Assessment of severity of HSP was done by using a structured questionnaire developed by Turner-Stokes and Jackson[16] called the “Shoulder Q” which consists of verbal rating scale and visual graphic rating scale designed to assess even in those subjects with language and visuospatial deficits. Shoulder Q[16] is a simple and practical tool for evaluation of shoulder pain. Changes on visual graphic rating scale (VGRS) were associated with verbal reports of improvement ($p < 0.001$). Summed VGRS score of 3 showed 77% sensitivity and 91.3% specificity for identifying the responders to the treatment, with a positive predictive value of 93.3%. Summed VGRS scores of =2 had a negative predictive value of 73.3%.

Follow-up at intervals of 1, 3 and 6 months from the date of discharge for all cases were attempted and even cases with at least one follow-up around 3 months were also included in the study.

Those patients who attended the department within a period of 12 weeks after stroke were defined as “early cases” and after that they were labelled as “late cases”. Again, for the convenience of assessing effectiveness of rehabilitation intervention of HSP, patients were grouped into “complier” if they attended at least one follow-up within 12 weeks after admission and another within 6 months of the first follow-up. Whereas, “non-complier” was labelled to those patients who had irregular follow-up and was also assumed to have received inadequate rehabilitation intervention.

The data was processed by using SPSS (Version 12). Chi-square test and logistic regression analysis were used.

The study was undertaken after getting ethics approval from the Institutional Ethics Committee.

Results:

Out of the 109 patients, 61.5% (n=67) were males while 38.5% (n=42) were females. Mean age group was 58.9 ± 10.9 (range 41-80 years). Maximum number of patients belonged to the age group 51-60 years (n=42), while minimum was in the age group 71-80 years (n=18).

Cerebral infarct was more common than haemorrhage (53.2% vs 46.8%). HSP was present in 47.7% (n= 52) of patients of the 109 patients evaluated. It was more prevalent among cerebral infarct patients (n= 30 of 58 patients, 51.7%) than those with haemorrhage (n=22 of 51 patients, 43.2%).

Left sided hemiplegia was seen more than the right (58.8% vs 42.2%). However, the prevalence of HSP on left and right sides were comparable (47.8% vs 47.6%).

Mean post-stroke duration at admission was 15.03 ± 32.39 weeks (range between 2 days to 154 weeks). Out of the 84 early patients, 41.7% (n=35) developed HSP and 68% (n=17) developed HSP out of the 25 late cases. Prevalence between late and early cases were statistically significant (p=0.021). Again, *the prevalence of HSP among compliers was 11.1%, while that of the non-compliers was 35.7% at the end of the follow-up. The difference was found to be statistically significant.*

Glenohumeral subluxation was present in 32.7% (n=17) of 52 cases with HSP and 33.3% (n=19) of 57 cases without HSP. Their association was not statistically significant.

Reflex sympathetic dystrophy was found in 15.6% (n=17) of the total cases and in 32.7% of HSP cases.

Mean FIM score at admission for patients with HSP was 54.5 ± 17.6 and 56.6 ± 19.5 among cases without HSP. Again, mean FIM scores at last follow-up were 80.0 ± 16.4 and 79.9 ± 18.9 respectively for both cases with HSP and without it.

Association of HSP with tone of upper extremity according to MAS, side of paralysis and recovery measured by FIM etc, were tested by logistic regression analysis. Subjects were categorised into complier who is regular in follow-ups (n=36) and non-complier who is not regular in follow-ups (n=73) to remove the effect of confounding factors of compliance to treatment.

Logistic regression analysis was done to find out relation of HSP with tone, side and recovery among compliers and non-compliers (Tables 1 and 2).

Odd's ratio was calculated from values among the compliers and patients with tone more than MAS=1 were more likely to develop HSP. Similarly, patients having good recovery as assessed by FIM (score of >72) had 52% less chance of developing HSP. The degree of association between HSP and side of paralysis was weak.

Table I: ***Logistic Regression Analysis of HSP with Tone, Recovery, GHS and Side of Paralysis in Compliers***

	B	S.E	Sig	Exp(B)	95% C.I. for Exp (B)(Lower)	95% C.I. for Exp (B)(Upper)
FIM2 CAT (1)	-0.680	1.246	0.586	0.507	0.044	5.830
Tone CAT (1)	0.548	1.251	0.661	1.730	0.149	20.094
GHS II (1)	7.145	43.732	0.870	1267.231	0.000	2.126455036399467 E+40
Side	-0.084	0.409	0.838	0.920	0.412	2.052

FIM 2 CAT(1) represents FIM assessments at second follow-up where 0 = persons having FIM score ≤ 72 and "1" = those with FIM score > 72.

Tone CAT(1) represents tone of the patients at second follow-up where '0'= FIM persons having MAS score ≤ 1 and "1" = those with tone MAS score > 1.

GHS II represents persons with GHS at the second follow-up where '0' = absent GHS and '1'= present GHS.

Side represents the side of paralysis where '1'= right and '2'= left.

Table 2: ***Logistic Regression Analysis of HSP with Tone, Recovery, GHS and Side of Paralysis in Non-compliers***

	B	S.E	Sig	Exp(B)	95% C.I. for Exp (B)(Lower)	95% C.I. for Exp (B)(Upper)
FIM2 CAT(1)	-0.444	0.559	0.428	0.642	0.214	1.920
Tone CAT (1)	0.119	0.514	0.817	1.126	0.412	3.083
GHS II (1)	-0.339	0.589	0.565	0.713	0.225	2.259
Side	0.229	0.495	0.643	1.258	0.476	3.322

Again among the non-compliers, odd's ratio value for those with more spasticity and better motor recovery were just the reverse of that of compliers. It explains that rehabilitation intervention had an overriding influence over the occurrence of HSP in relation to variables like tone and degree of motor recovery.

Discussion:

The prevalence rate of HSP among post-stroke hemiplegia patients in the present study is similar with the finding of Poulin de-Courval *et al*[6] (47.9%). No relation between age groups and HSP was also reported by Griffin[17] and Cheng *et al*[10].

A study by Davis *et al*[18] had found predilection of HSP among right sided hemiplegia, while HSP was more prevalent among left sided hemiplegia as reported by Pauline de Courval *et al*[6]. Present study showed more cases of left sided hemiplegia (57.8%) and a similar prevalence of HSP on both sides. Cheng *et al*[10] also did not find any relationship between HSP and side of paralysis.

There were two common factors associated with patients with HSP; loss of range of motion of shoulder, especially external rotation, and subluxation of glenohumeral joint.

Present study showed a positive correlation between HSP and increased tone which is similar to reports of studies by Van Ouwenaller *et al*[7] and Poulin de Courval *et al*[6]. However, studies done by Bohannon and Andrew[5] and Joynt[19] have not found relationship between spasticity and HSP.

No association was found in the study between HSP and GHS, which was similar to studies done by Bohannon and Andrews[5], Wanklyn *et al*[8] and Zorowitz *et al*[9]. However, a positive correlation was also reported by Van Ouwenaller *et al*[7] and Najenson *et al*[20].

There was a window of opportunity to observe two types of post-stroke hemiplegic patients which was categorised into early and late cases. The median time for development of HSP was within the first 12 weeks of post-stroke. Hence, twelve weeks was taken as the limit to differentiate between early case who sought rehabilitation within 12 weeks and late case who reported after 12 weeks. Early cases were assumed to have learned and received the rehabilitation early.

It was found that HSP was more prevalent and persisted among the late cases at follow up which was found statistically significant (p=0.021). This showed that institution of early rehabilitation was effective in prevention and improvement of HSP.

Another method of assessing the effectiveness of the rehabilitation intervention was by comparing its prevalence in compliers and non-compliers which was found significant (p=0.042). It may also be presumed that the rehabilitation intervention practiced in RIMS was effective in the prevention and management of HSP syndrome.

A similar study was done by Wanklyn *et al*[8] where the followed-up 108 patients over a period of 6 months for prevalence of HSP and correlation with other factors. It was reported that HSP developed in 63.8% patients and its prevalence increased in the first few weeks post discharge. They also found a strong association between HSP and poor motor recovery.

Conclusion:

Prevalence rate of HSP among post-stroke hemiplegia patients admitted during two years was 47.7%. There was no association of HSP with factors like age, sex, side of paralysis, type of lesion and GHS. Correlation between HSP and tone or degree of functional recovery was significant.

It was also found that the rehabilitation intervention practiced in the management of HSP was effective as was evident from the prevalence of HSP among early and late cases and compliers and non-compliers.

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PG Forum

REHAB QUIZ

1. All are electrophysiological diagnostic criteria of carpal tunnel syndrome except

A) Distal median motor latency > 4.4 Ms
B) Difference between distal motor latency of median and ulnar>0.4 Ms
C) Difference between distal sensory latency of median and ulnar>0.2 Ms
D) Inching technique: latency jump > 0.2 Ms/cm

2. Commonest inspiratory muscle training method prescribed in patients with COPD

A) Threshold loading technique
B) Flow resistive technique
C) Purse lip breathing technique
D) Diaphragmatic breathing technique

3. Brandt-Daroff exercise is used for

A) In hospital treatment for cervical vertigo
B) Home exercise for cervical vertigo
C) In hospital treatment for BPPV
D) Home exercise for BPPV

4. Which artery is involved in a stroke patient presented with contralateral hemianaesthesia with visual special deficit and aprosody without affect disorder

A) Upper division of MCA – dominant hemisphere
B) Upper division of MCA – non-dominant hemisphere
C) Lower division of MCA – dominant hemisphere
D) Lower division of MCA – non-dominant hemisphere

5. Which lobe of lung is drained in supine position with foot end of bed elevation 18 – 20 inches?

A) Anterior basal segment of right lower lobe
B) Lateral basal segment of right lower lobe
C) Medial basal segment of right lower lobe
D) Lateral basal segment of left lower lobe

6. Both chin tuck and supraglottic swallow is helpful in

A) Delayed swallowing reflex and decreased unilateral pharyngeal paralysis
B) Delayed swallowing reflex and decreased bilateral pharyngeal paralysis
C) Delayed swallowing reflex and decreased laryngeal closure
D) Delayed swallowing reflex and decreased opening of cricopharyngeal region

PG Forum

7. All are energy storing prosthetic foot except
A) SAFE II foot
B) Seattle foot
C) Carbon copy II foot
D) Flex foot

8. Calf hypertrophy is not seen in
A) Duchene muscular dystrophy
B) Becker muscular dystrophy
C) Limb girdle muscular dystrophy
D) Emery- Dreifuss muscular dystrophy

9. Modified Amor criteria is diagnostic of
A) Bechet's disease
B) Undifferentiated spondylo-arthropathy
C) Juvenile onset spondylo-arthropathy
D) Electrophysiological criteria of GBS

10. All are done in speech therapy of aphasia except
A) Melody intonation technique
B) Retraining of articular pattern
C) Conversational coaching
D) Oral reading.

ANSWERS

June issue:

1-C; **2**-D; **3**-D; **4**-C; **5**-A; **6**-D; **7**-B; **8**-A; **9**-C; **10**-C

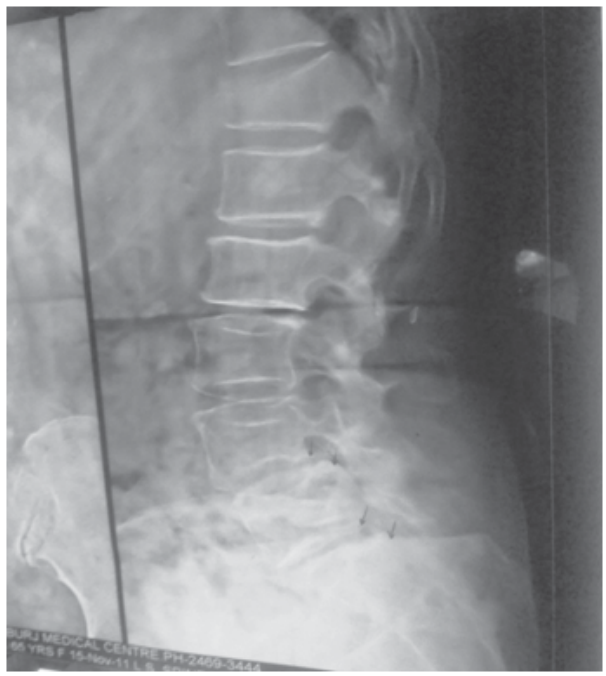
Right responder (10 out of 10): **Dr. Harleen Uppal,** Junior Resident, Dept. of PMR, AIIMS, New Delhi

Next right responder: **Dr. Prajna Ranjani M,** Junior Resident, AIIMS, New Delhi

PG Forum

REHAB CHALLENGES

A sixty-six-year lady presented to department of PMR with severe non-inflammatory backache. On clinical examination VAS score of pain was 7 out of 10 and step sign was positive. Initially she was put on NSAID, local superficial heat and static group of spinal exercise regimen. On routine investigations spondylolisthesis of L4 over L5 was quite obvious on x-ray.



X ray picture

Interestingly it was noticed that there were few collapsed vertebrae in lower dorsal region. In routine haemogram ESR was 26mm and haemoglobin was 12.3g/dl. Subsequently DEXA- Scan of her spine showed a T score of -3.2g/dl. Her back pain was controled by the medications and initial rehabilitation regimen.

Please opine regarding further exercise regimen and bracing options for this lady?

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Synovial Chondromatosis Misdiagnosed As OA Knee

R Pramanik[1], D K Khatua[2]

A 52-year old lady presented to PMR outpatient department with knee pain and swelling for last two months. At the time of first visit VAS score of pain was 7 out of 10. There was moderate tenderness on joint lines and significant effusion of her right knee joint (Figs 1 & 2).

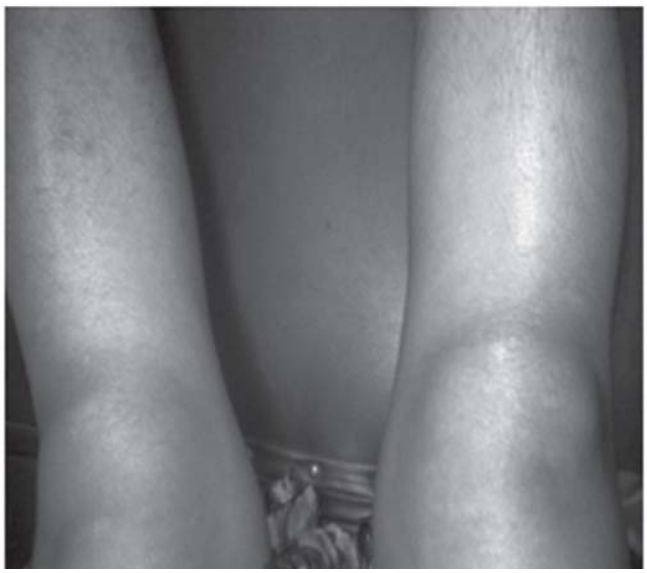

Fig: 1

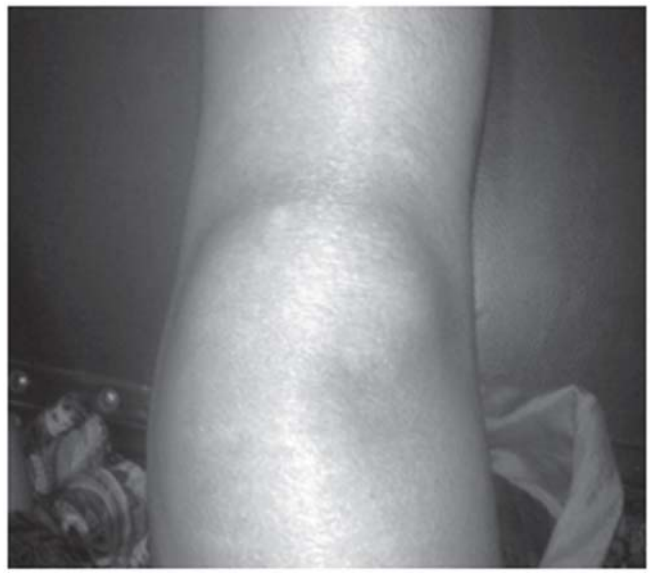

Fig: 2

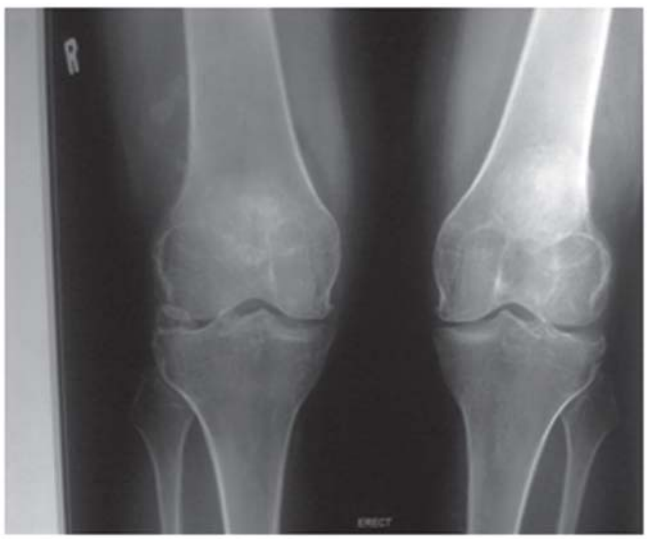

Fig: 3

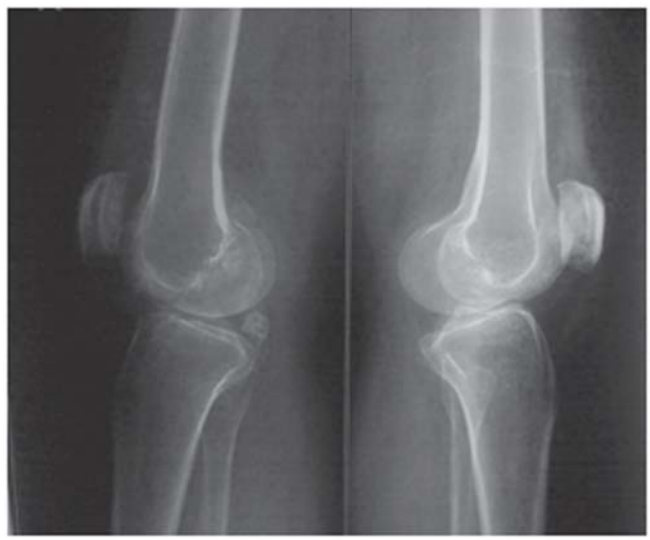

Fig: 4

Author's affiliations
[1] *MD, MRCP(UK), Assistant Professor, Dept. of PMR, IPGMER & SSKM Hospital, Kolkata.*
[2] *MD, Associate Professor, Dept. of PMR, BSMC, West Bengal*

Initially a provisional diagnosis of osteo-arthritis of knee joint was made and base line investigations like inflammatory markers and x-rays were advised. Initial rehabilitative management like joint protection, life style

modification, exercise regimen and paracetamol 1g four times a day was started. According to her blood reports Hb% 10g/dl (69%), WBC- 10,200 (neutrophil 68%, lymphocyte 27%), ESR 56, fasting blood glucose 90g/dl, creatinine 0.9, TSH 5.8. Considering significantly disproportionate involvement of right knee joint one diagnostic synovial fluid aspiration was done. The synovial fluid was inflammatory in nature without any Gram-stain response, AFB, malignant cell and crystals. Her x-rays findings are shown in Figs 3 & 4.

Interestingly we noticed lots of opacities in x-rays, looking like loose bodies due to osteo-arthritis of knee. But few spots are located little bit proximally (eg, superior lateral side of right knee). After a careful observation we were sure that these were unlikely to be in the suprapatellar pouch. At this juncture a MRI scan of knee was advised due these unexplained lesions.

In MRI scan Figs 5–8 mutiple focal calcifications were seen in peri and retro-articular region with such lesion also seen along the margins of the condyle. The MRI was reported as a case of synovial chondromatosis which is a benign but rare clinical condition of knee joint. Subsequently the patient was reffered to department of orthopaedics for sugical excision.

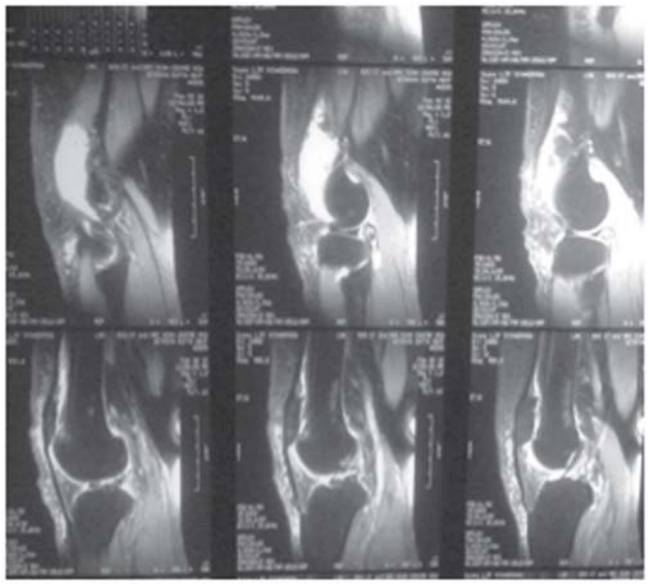

Fig: 5

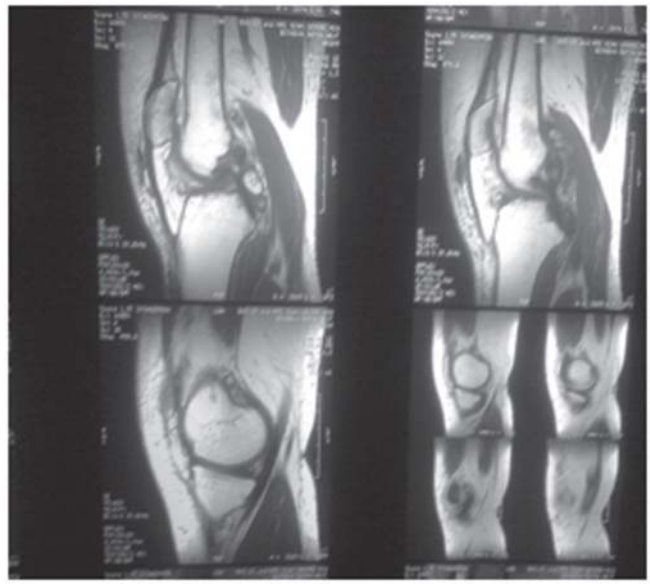

Fig: 6

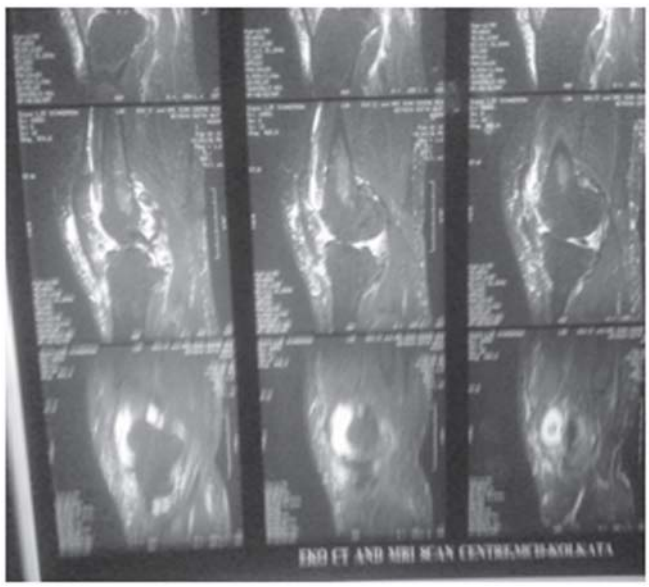

Fig: 7

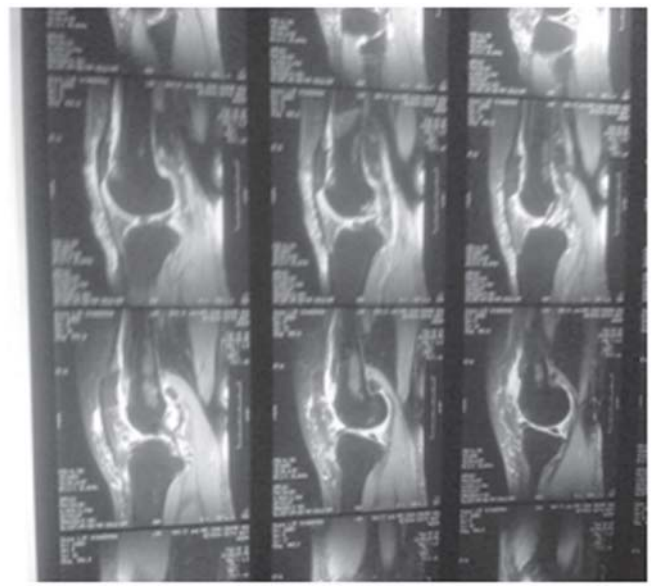

Fig: 8

Case Report

Unwanted Muscle Weakness following Botulinum Neurotoxin A Administration in Spinal Cord Injury with Literature Review

Tapan N Joshi

Abstract

Botulinum neurotoxin A (BoNTA) is rapidly gaining acceptance for management of spasticity secondary to spinal cord injury (SCI). Due to its increased usage, more undesirable effects and complications have come in light. Unwanted distant and/or generalised muscle weakness is possible following BoNTA administration in SCI population causing temporary neurological and functional decline. Physicians should carefuly perform a clinical assessment of every patient individually for risks stratification. Additional studies for adult population evaluating adverse-effects of high dose of BoNTA treatment for spasticity management are indicated.

Key words : **Botulinum neurotoxin A, adverse events, spasticity, spinal cord injury, distant muscle weakness.**

Introduction:

Spasticity is a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon responses.[1] It is one of the most common consequences of spinal cord injury (SCI). One year post-injury, about 78% of patients demonstrate spasticity with more than half requiring pharmacological interventions.[2] Botulinum neurotoxin is gaining rapid acceptance for spasticity management due to several advantages. It avoids sedation, common with oral antispasticity medications. No surgical intervention is required. It provides an option of focal spasticity management. It is equally effective as phenol[3] but technically simpler to administer, less painful, and without any side-effects like dysesthesia. Due to its increased usage, more undesirable effects have come in light. Here, I have described a case elucidating distant and generalised muscle weakness as a potential side-effect of botulinum neurotoxin A (BoNTA) in a patient with SCI with brief literature review.

Author's affiliations:
MD, MS, DABPMR, CIIRP Director, Division Chief of Spinal Cord Injury, VA Central Iowa Healthcare System, USA

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Correspondence :
Tapan N. Joshi,
Phone # - 515-699-5999 Ext: 4213, 4640,
Email : tapan.joshi@va.gov

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Case Report:

A 35-year-old man sustained C5 burst fracture secondary to a diving accident. His injury was little more than one year old when he was evaluated at our centre for outpatient neurorehabilitation. He was diagnosed C4 ASIA impairment scale C. During the rehabilitation course, his progress was hampered due to spasticity (3/4 on modified Ashworth scale) in bilateral hip adductor and ankle plantar-flexor muscles even after receiving oral baclofen, 80 mg/day. Therefore, it was decided to address spasticity with Botox®. A total of 800 units (~10 units/kg; 200 units in thigh adductors, 150 units in gastrocnemius – both heads, and 50 units in tibialis posterior in each extremity) were injected using an EMG-guided technique. Each 100 units were diluted with two ml of preservative-free normal saline. Patient tolerated the procedure well. A week later, he was admitted in a hospital for cellulitis followed by septic arthritis of the left elbow. He was treated with intravenous cephalosporin. His infection never progressed to cause compartment compression syndrome and his 2-week hospital stay was uneventful. One week post-discharge, upon his return to the clinic, patient complained for left wrist extension weakness and increased difficulty to transfer. It evolved over past one week. He denied for dysphagia or dysphonia. As per his hospital records, patient did not acquire left wrist weakness during his hospital stay. On physical examination, cranial nerves evaluation was normal. His left elbow infection was resolved but he developed

generalised truncal weakness along with noticeable deterioration in muscle strength of left wrist extensors. It deteriorated from 4/5 to 1/5 on Medical Research Council scale (Fig 1). There was no prominent change in strength of other left upper extremity muscles especially brachioradialis, supinator, and finger extensors. Sensory examination was also at baseline. Spasticity in hip adductors and ankle plantar-flexor muscles was decreased to 1+ on modified Ashworth scale. Patient demonstrated decreased sitting balance due to truncal weakness. His abilities for pressure relief and bed-to-wheelchair transfer were also markedly impaired due to wrist extension weakness. He was referred for laboratory, electrodiagnostic, and imaging studies. Electrodiagnostic study was performed three weeks after onset of left wrist weakness. It was positive for ulnar nerve entrapment at the right elbow. Nerve conduction study (NCS) of radial nerve did not show latency increase or decrease in amplitude or conduction velocity across the left elbow. Electromyography (EMG) was performed in left brachioradialis, supinator, extensor digitorum and extensor indicis. It showed normal activities at needle insertion as well as at rest along with no denervation potentials. Magnetic resonance imaging (MRI) with contrast of the cervical, thoracic and lumbar spine and laboratory work-ups demonstrated no acute pathology (Fig 2). He was continued on outpatient rehabilitation programme. He gradually regained functional strength of his left wrist extensors and trunk muscles. His functional ability to transfer, pressure relief, and sitting balance also improved.

Discussion:

Our patient developed generalised weakness with prominent weakness in left wrist extension, approximately three weeks after BoNTA administration. Multiple plausible aetiologies were considered including radial nerve injury at the elbow level, syringomyelia, cord tethering and cord/root compression secondary to infection, tumour or disc herniation. Since radial nerve

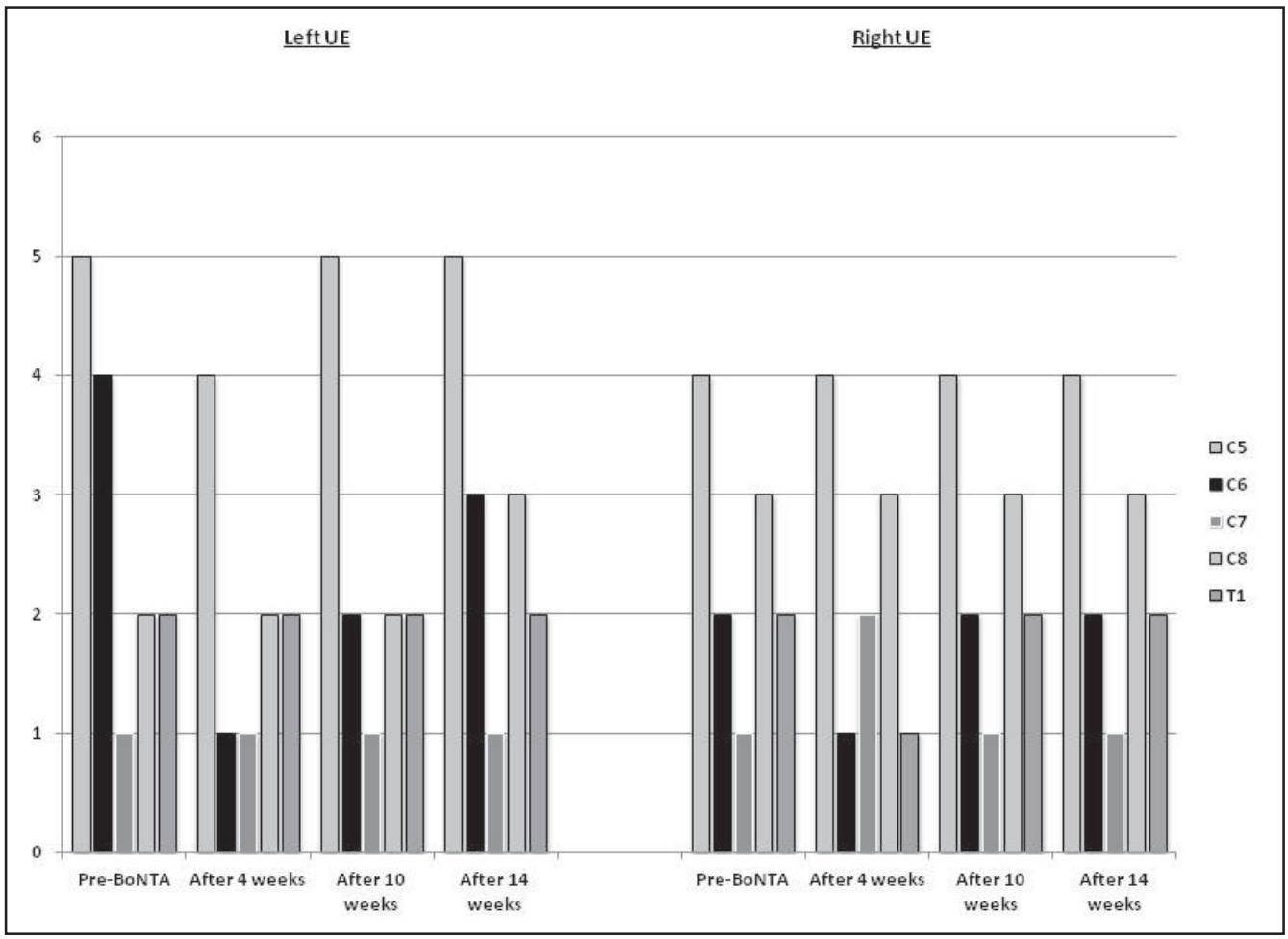

Fig 1: Muscle Strength in Upper Extremities as per American Spinal Injury Association (ASIA) Classification

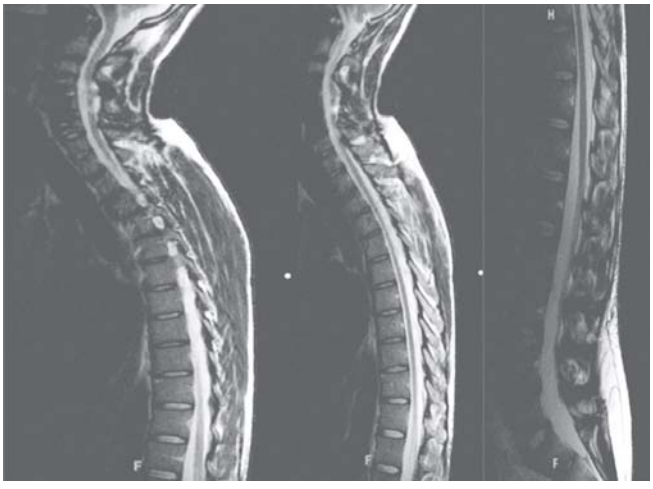

Fig 2: MRI of the Cervical, Thoracic and Lumbar Spine with Gadolinium Contrast Showing Chronic Changes due to C4-5, C5-6 Fusion after Old Cervical Spine Injury without New Pathology like Post-traumatic Syringomyelia, Cord Tethering, or Cord Compression Secondary to Infection, Tumor, or Disc Herniation

injury at the elbow level was one of the differential diagnoses, an electrodiagnostic study was ordered. Absence of conduction block at the elbow level as well as lack of denervation potentials effectively ruled out left radial nerve injury including neuropraxia or axonotmesis. Likewise, physical examination did not demonstrate prominent weakness in other muscles supplied by radial or posterior interosseous nerve except the wrist extensors. Unremarkable spine MRI and laboratory work-up exclude radiculopathy, myelopathy, or other acute aetiologies. Along with left wrist weakness, patient also developed generalised weakness which is a documented side-effect of BoNTA. His muscle strength improved within 14-16 weeks. All information can collaboratively deduce BoNTA as a causative factor for unwanted muscle weakness.

Reports suggesting unwanted muscle weakness following BoNTA injection are commonly published for cerebral palsy but sparsely for SCI. To this author's knowledge, only one report has been published till this date.[4] BoNTA has been utilised successfully since the late 1980s to treat limb spasticity.[5] It irreversibly binds to presynaptic neurons at the neuromuscular junction within hours of administration. It actively cleaves peptides necessary for membrane-bound acetylcholine release. The peripheral blockade of neuromuscular activity causing focal muscle weakness is a desirable therapeutic effect. Though BoNTA has good safety and tolerability profile,[6] one of the undesirable systemic effects is unwanted muscle weakness. Occasionally, the toxin tends to spread causing regional, distant or generalised muscle weakness. Regional weakness manifests in adjacent anatomical area to the injection site e.g. dysphagia/dysphonia after injections in neck muscles for cervical dystonia[7] or diplopia/facial palsy after BoNTA treatment for blepharospasm[8]. Distant muscle weakness occurs in anatomically separate and remote sites. In animal studies, BoNTA has been shown to spread 30-45 mm from the injection site.[9] Higher dose,[9] an incorrect injection technique, and higher dilution[10] may potentiate spread of the toxin. Its effects on neuromuscular transmission distant to the injected site have been demonstrated by single-fibre electromyography showing increased jitter and blocking.[11] Although these changes suggest disturbance in neuromuscular transmission, they do not explain the cause of the disturbance. Besides, clinically they are not always associated with muscle weakness.[11] Premorbid conditions like amyotrophic lateral sclerosis or neuromuscular junction disorders (e.g. myaesthenia gravis or Lambert-Eaton syndrome) may increase possibilities of unwanted muscle weakness.[12] It is possible that decreased function of alpha motor neurons makes these patients more susceptible to BoNTA. Patients treated concomitantly with agents interfering with neuromuscular transmission (e.g. aminoglycosides, curare-like agents) should also be observed closely for the same reason.[13] In the present case, the patient neither had above mentioned pre-existing conditions nor received agents impairing neuromuscular transmission.

There are several hypotheses regarding modes affecting BoNTA diffusion. Retrograde axonal spread is one of the proposed physiological modes of diffusion in literature but there are no solid evidences to support. Wiegand suggested a theory of retrograde axonal spread to corresponding spinal cord segments using radioactive BoNTA.[14] On the contrary, Koman *et al*[15] provided an indirect evidence to nullify that theory by demonstrating no change in 'pre- and post-BoNTA' H-reflex. The H-reflex is a well-standardised measure of central synaptic activity. Therefore, they concluded that BoNTA had no significant "central" effect. Systemic spread of BoNTA can also be possible by entering into a vascular system. Another mode of BoNTA diffusion to the distant sites can be systemic vascular spread. Nonetheless, it is a standard practice to aspirate before administration to prevent intravascular injections. Besides if the toxin enters the venous system, the manifestations will be much quicker; which did not happen in this case. Capillary uptake may be possible and needs to be explored further.

There may be many factors affecting BoNTA diffusion

causing systemic effect: lack of injection guidance, higher weight-adjusted dose, dilution (volume) and total cumulative dose. Underlying comorbidities may also make subjects predisposed to systemic involvement.[13,16] There is not a single factor that has emerged as a potential cause. Needle guidance ascertains a placement of a needle and an end-organ to be injected. Therefore, lack of guidance may increase chances of systemic spread. Higher weight-adjusted dose is another possible factor for systemic spread causing distant/generalised weakness. Crowner *et al*[17] demonstrated no relationship between high dose (21units/kg) and distant muscle weakness. The European consensus group has also suggested 30 units/kg of BoNTA as a safe upper limit for spasticity management.[18] Our patient received about 10 units/kg of his body weight which is considered very safe by most published studies and yet, he developed unwanted muscle weakness. A possible explanation can be 'an indirect overdose' of BoNTA. In chronic SCI, there is a decrease in lean muscle mass secondary to muscle atrophy and its replacement with fatty tissue.[19] Therefore the requirement of BoNTA can be less than the calculated weight-adjusted dosage, causing indirect overdose but its effect is not universally seen in every patient with chronic SCI. Higher dilution has shown to increase spread across the muscular plane[10] but at the same time, Lee *et al*[20] has shown no clinical effect in term of excessive weakness with different dilutions. Dilution used in our case is standard for spasticity management involving large muscle groups. Another possible reason could be the total cumulative dose of BoNTA injected.[13] The patient was injected with 800 units of Botox® which is arguably a high dose for the first time treatment and it could be a possible reason for distant and generalised weakness. Although, total dose of 800-1200 units of Botox® is safely used for spasticity management in young adults more than 45 kg of body weight.[21] In the present case, the dose was titrated based on the practitioner's experience and the patient's clinical condition. He was already on 80 mg/day of oral baclofen while receiving rehabilitation interventions and yet he had significant spasticity, 3/4 on modified Ashworth scale. Besides, many of our spinal cord injured subjects were treated safely and successfully with a high dose of BoNTA. This was our first case of unwanted distant muscle weakness along with generalised weakness in a patient with SCI. It remains unclear why only left wrist extensors became markedly weak. Recently, the European consensus group has also suggested further studies to identify the side-effects of high doses of BoNTA treatment in adults.[22]

Conclusion:

Botulinum neurotoxin has evolved as a widely used therapeutic measure for spasticity management. Serious side-effect like unwanted generalised and/or distant muscle weakness is uncommon but possible. Our understanding of risk factors as well as pathogenesis of distant muscle weakness is limited. It should be discussed prior to the treatment. Physicians should carefully perform a clinical assessment of every patient individually for the benefit-risk profile. Additional studies for adult population evaluating adverse-effects of high dose of BoNTA treatment for spasticity management are recommended.

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