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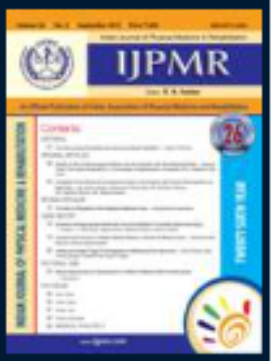

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PG Forum

BOOK NEWS

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Review Article

The Role of Physiatrist in the Diabetes Healthcare Team

Ramamoorthy Veyilmuthu

Abstract

The physiatrist is an important member of the diabetes healthcare team. His/her primary role is to provide physical activity counselling for the diabetics after thorough evaluation. Exercise is a primary component of diabetes management together with diet plan and antidiabetic drugs. The physiatrist, with his/her background knowledge in exercise physiology, is the most appropriate person for this job. Regular exercise has been shown to improve blood glucose control, reduce cardiovascular risk factors, contribute to weight loss and improve well being. Furthermore, regular exercise may prevent type 2 diabetes in high risk individuals. Thus physical activity plays a pivotal role in health promotion and diabetes control. People with diabetes should be advised to perform at least 150 minutes per week of moderate intensity aerobic physical activity using 'FITT Principle'. In the absence of contra-indications, people with type 2 diabetes should be encouraged to perform resistance training three times per week. DeLorme resistance exercise improves power, strength, endurance and burns more calories. Cross training is the term used to describe the use of a variety of activities during exercise. Interval training is the variation of intensity during one or more aerobic activities. Circuit training is the combination of aerobic exercise and weight training (anaerobic exercise). Before recommending a programme of physical activity, the physiatrist should assess diabetics for coronary artery disease and other complications of diabetes.

Key words: **'FITT Principle', DeLorme resistance exercise, cross training, interval training, circuit training.**

Introduction:

Exercise is an important part of the diabetes management plan[1]. Regular exercise has been shown to improve blood glucose control, reduce cardiovascular risk factors, contribute to weight loss, and improve well-being[2]. Furthermore, regular exercise may prevent type 2 diabetes in high-risk individuals [3-5]. Structured exercise interventions of at least 8 weeks' duration have been shown to lower glycosylated haemoglobin (HBA1C) by an average of 0.66% in people with type 2 diabetes[6]. Higher levels of exercise intensity are associated with greater improvements in A1C and in fitness [7]. Other benefits include increased sensitivity to insulin and improved blood glucose control[8] and potential for decrease in diabetes medications needed to control blood glucose. Exercise may reduce blood lipids (total cholesterol, LDL cholesterol and triglycerides) and increase HDL cholesterol. Exercise may lower blood pressure [9]. Weight loss and maintenance can occur when exercises are combined with healthy eating habits. Exercise may be as effective as some psychotherapies in treating depression [10,11]. Another study showed positive effects of exercise on hospitalized depressed patients, with regular aerobic exercise having an antidepressant effect[12]. The collateral benefits of exercises are the patients often notice energy throughout the day, improved stress management, promotion of psychological well-being and increased quality of life. Thus physical activity is considered as a therapeutic tool in a variety of patients with or at risk for diabetes. The physiatrist, with his background knowledge in exercise physiology, is the most appropriate person for evaluating and prescribing exercises to the diabetics [13].

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Review of Literature:

Review of literature reveals that diet and exercise interventions in those with impaired glucose tolerance may delay the development of type 2 diabetes [1]. There are 3 studies to support this statement. They are FINNISH Study [14], Diabetes Prevention Program (DPP) [15] and Da

Qing Study [16]. In all these studies, the individuals with impaired glucose tolerance (IGT) were randomised to either a usual care control group or intensive life style intervention group. The control group received general dietary and exercise advice at baseline and had an annual physician's examination. The subjects in the intervention group received additional individualised dietary counselling from a nutritionist. They were also offered circuit-type resistance training sessions and advised to increase overall physical activity. The intervention was most intensive during the first year, followed by a maintenance period. The intervention goals were to reduce body weight, reduce dietary and saturated fat, and increase physical activity and dietary fibre. At the end of these studies it was found out that the intervention group showed significantly greater improvement in each intervention goal like weight reduction, glycaemic control and improved lipid level[2]. So it was concluded that the intensive lifestyle intervention produced long-term beneficial changes in diet, physical activity, and clinical and biochemical parameters and reduced diabetes risk.

Evaluation of the Diabetics before recommending an Exercise programme - before recommending an exercise programme, the individual with diabetes mellitus should undergo a detailed medical evaluation and appropriate diagnostic studies by the physiatrist. This examination should carefully screen for the presence of macro- and micro vascular complications that may be worsened by the exercise programme. Identification of areas of concern will allow the design of an individualised exercise prescription that can minimise risk to the patient.

Exercise prescription :

After thorough evaluation, exercise is prescribed for the diabetics. "FITT Principle" is being followed to prescribe exercise to the diabetics[17]. The acronym 'FITT' stands for Frequency, Intensity, Time and Type of exercises.

The U.S. Surgeon General's report[18] recommended that most adults accumulate at least 30 minutes of moderate intensity activity on most, ideally all, days of work (frequency). They must carry out physical activity at the intensity of 50 - 70 % of maximum heart rate[19]. This is called target heart tate (intensity). The target heart rate is the rate at which the diabetics should aim to exercise. To calculate the maximum heart rate, the diabetic has to subtract his/her age from 220. Multiply the maximum heart rate by 0.50-0.70 will give the target heart rate[20]. The physical activity period must be approximately 60 (time) minutes which includes a warm up period of 5-10 minutes before the exercise session and a cool-down period of 10 minutes towards the end of the exercise session. The physical activity recommended is aerobic exercises (type).

Warm up (preliminary exercise)[21]: A standard recommendation for diabetic patients, as for nondiabetic individuals, is that physical activity includes a proper warm-up and cool-down period. A warm-up should consist of 5 – 10 minutes of aerobic activity (walking, cycling, etc,) at a low intensity level. The warm-up session is to prepare the skeletal muscles, heart, and lungs for a progressive increase in exercise intensity. After a short warm-up, muscles should be stretched for another 5-10 minutes, primarily, the muscles used during the active physical activity session, but warming up all muscle groups is optimal. The active warm-up can either take place before or after stretching.

There are two types of warm-ups.

General warm up: This type of warm up includes stretching, and general body movements or loosening up exercises that are unrelated to the specific neuromuscular actions of the anticipated performance.

Specific warm up: This type of preliminary exercise provides a skill rehearsal for the actual activity in which the participant is preparing. Practising tennis, playing football and performing preliminary lead up in the high jump or pole vault are examples of specific warm-up.

Cool down [22, 23] : Moderate exercise, the practice of continuing to walk or jog at a slow pace, after strenuous exercise is called cool down. This facilitates blood flow through the vascular circuit including the heart and prevents venous stasis in the lower extremities during recovery and thereby prevents syncope. The cool-down period gradually brings the heart rate down to its pre-exercise level. The cool down period should last about 5 – 10 minutes.

Resistance exercise :

Resistance exercise improves insulin sensitivity to about the same extent as aerobic exercise[24]. Clinical trials have provided strong evidence for the A1C lowering value of resistance training in older adults with type 2 diabetes[25, 26] and for an additive benefit of combined aerobic and resistance exercise in adults with type 2 diabetes[27]. If resistance is applied to a muscle as

it contracts, the muscle will adapt and become stronger over time. As the strength of a muscle increases, the cardiovascular response of the muscle improves so that muscular endurance and power also increases. To determine how much weight a person should use when beginning a resistance exercise programme, first the person's repetition maximum (RM) is found out. A RM is the greatest amount of weight (load) a muscle can move through the range of motion a specific number of times. For beginners, it is recommended a base line of 6 RM to 10 RM.

DeLorme developed the term Progressive Resistive Exercise to describe his approach to strengthen exercise[28-30].

- First determine the 10 RM
- The person then carries out
  - 10 repetitions at one half of the 10 RM
  - 10 repetitions at three-fourths of the 10 RM
  - 10 repetitions at the full 10 RM

The person has to perform all three bouts at each exercise session with a brief rest between bouts. The amount of weight is increased weekly as strength increases. The resistance exercise increases the strength, endurance, power and burns more calories.

General Exercise Tips to the Diabetics :

The diabetics are instructed to wear medical identification while exercising. They are advised to consider exercising with another person or a group, in case they need assistance. They have to keep blood glucose, food and exercise records to determine their overall response to exercise. The use of silicone gel or cotton polyester socks to prevent blisters and keep the feet dry is important for minimising trauma to the feet. Proper foot wear is essential and must be emphasised for people with peripheral neuropathy. Individuals must be taught to monitor closely for blisters and other potential damage to their feet both before and after physical activity. They have to drink plenty of water before, during and after exercise to avoid dehydration - 250 ml every 15 minutes or one litre of fluid per hour is recommended [31]. They have to set aside the same time each day for the exercise and make it a habit. They have to choose a time that does not coincide with the peak action of insulin. They are instructed not to inject insulin into an area that is contracting during the exercise. For example, if running, do not inject into the legs; if weight lifting, do not inject into the arms.

Insulin Adjustments :

The risk for hypoglycaemia is less when the level of insulin in the body is lower. The diabetics must avoid planning exercise programme for the time when the insulin is peaking. They have to avoid exercise for 1-2 hours after injecting rapid or short acting insulin. They have to plan exercise session before the morning insulin dose or 1-3 hours after eating. The risk for nocturnal hypoglycaemia is greater when exercise is performed in the evening. Reducing the evening insulin dose helps decrease the risk. Reducing insulin doses is particularly helpful for those who exercise routinely as part of weight management and or to improve control.

Carbohydrate Adjustments :

The diabetics are instructed to exercise around the same time each day that will make planning insulin and snack changes easier and more consistent. The decision about whether to eat additional food or adjust medication is based on the goal for exercise. Carbohydrate replacement is most useful for unplanned exercise or exercise of long duration.

Exercise in the Presence of Non-optimal Glycaemic Control:

Hyperglycaemia: When people with type 1 diabetes are deprived of insulin for 12–48 hours and are ketotic, exercise can worsen hyperglycaemia and ketosis; therefore, vigorous activity should be avoided in the presence of ketosis.

Hypoglycaemia: In individuals taking insulin and/or insulin secretagogues, physical activity can cause hypoglycaemia if medication dose or carbohydrate consumption is not altered. For individuals on these therapies, added carbohydrate should be ingested if pre-exercise glucose levels are 100 mg/dl. Hypoglycaemia is rare in diabetic individuals who are not treated with insulin or insulin secretagogues, and no preventive measures for hypoglycaemia are usually advised in these cases.

Exercise in the Presence of Specific Long Term Complications of Diabetes:

Retinopathy: In the presence of proliferative diabetic retinopathy (PDR) or severe non-proliferative diabetic retinopathy (NPDR), vigorous aerobic or resistance exercise may be contra-indicated because of the risk of triggering vitreous haemorrhage or retinal detachment[32].

Peripheral neuropathy: Decreased pain sensation in the extremities results in increased risk of skin breakdown and infection and of Charcot joint destruction. Therefore, in the presence of severe peripheral neuropathy, it may be best to encourage non-weight bearing activities such as swimming, bicycling, or arm exercises [33,34]. All individuals with peripheral neuropathy should wear proper footwear and examine their feet daily to detect lesions early.

Autonomic neuropathy: Autonomic neuropathy can increase the risk of exercise induced injury or adverse event through decreased cardiac responsiveness to exercise, postural hypotension, impaired thermoregulation, impaired night vision due to impaired papillary reaction, and unpredictable carbohydrate delivery from gastroparesis predisposing to hypoglycaemia[35]. Autonomic neuropathy is also strongly associated with CVD in people with diabetes[36,37].

Albuminuria and nephropathy: Physical activity can acutely increase urinary protein excretion. However, there is no evidence that vigorous exercise increases the rate of progression of diabetic kidney disease, and there is likely no need for any specific exercise restrictions for people with diabetic kidney disease [38].

Cross training- The term cross training refers to a training routine that involves several different forms of exercise. For example, using both biking and swimming each week to improve overall aerobic capacity, build overall muscle strength and reduce the chance of an overuse injury. Cross training is a great way to condition different muscle groups, develop a new set of skills, and reduce boredom that creeps in after months of the same exercise routines.

Interval training- Interval training is a type of physical training that involves bursts of high-intensity work interspersed with periods of low-intensity work. The high-intensity periods are typically at or close to near-maximum exertion, while the recovery periods may involve either complete rest or activity of lower intensity. It is a technique particularly employed by runners. However, it is also applicable to exercisers as it helps improve exercisers' aerobic capacity to exercise longer at varying intensities .This method of training may be more effective at inducing fat loss than simply training at a moderate intensity level for the same duration. A significant amount of high intensity work can be achieved with interval or intermittent work if there is appropriate spacing of the work relief intervals. The total amount of work than can be completed with intermittent work is greater than the amount of work that can be completed with continuous training [39-41].

Circuit training [42, 43] is a form of combining resistance training and high-intensity aerobics. It is designed to be easy to follow and target strength building as well as muscular endurance. An exercise "circuit" is one completion of all prescribed exercises in the programme. When one circuit is complete, one begins the first exercise again for another circuit. Traditionally, the time between exercises in circuit training is short, often with rapid movement to the next exercise.

Physical Activity Recommendations [44]:

- People with diabetes should be advised to perform at least 150 minutes/week of moderate-intensity aerobic physical activity (50–70% of maximum heart rate).
- In the absence of contra-indications, people with type 2 diabetes should be encouraged to perform resistance training three times per week.

Conclusions:

The recent Surgeon General's Report on Physical activity and Health underscores the pivotal role physical activity plays in health promotion and disease prevention. It recommends that individuals accumulate at least 30 minutes of moderate physical activity on most days of the week. In the context of diabetes, it is becoming increasingly clear that the epidemic of type 2 diabetes sweeping the globe is associated with decreasing levels of activity and an increasing prevalence of obesity. Thus, the importance of promoting physical activity as a vital component of the prevention as well as management of type 2 diabetes must be viewed as a high priority. Ultimately, all persons with diabetes should have the opportunity to benefit from the many valuable effects of physical activity prescribed by the physiatrist.

What We Already Knew:

- Exercise is a primary component of diabetes management together with diet plan and anti - diabetic drugs.

What We Learn from This Article:

- Exercise prescription method to diabetics – "FITT Principle"
- DeLorme's Progressive Resistive Exercises – Repetition Maximum (RM)

- Exercises to be avoided in presence of long term complications of diabetes
- Cross training, interval training, and circuit training

References:

1. Bird SR, Hawley JA. Exercise and type 2 diabetes: new prescription for an old problem. Maturitas. 2012 Aug; 72(4):311-6. doi: 10.1016/j.maturitas.2012.05.015.Epub 2012 Jun 27. Review. PubMed PMID: 22748760.
2. Brodsky JW. Outpatient diagnosis and care of the diabetic foot. Instr Course Lect. 1993; 42: 121-39. Review. Pub Med PMID: 8463660.
3. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, *et al:* Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002; **346:**393-403.
4. Tuomilehto J, Lindstorm J, Eriksson JG, Valle TT, Hamalainen H, Ilanne- Parikka P. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001; **344:**1343-50.
5. Pan XR, Li GW, Hu YH ,Wang JX, Yang WY, An ZX, *et al* . Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance : the Da Qing IGT and Diabetes Study. *Diabetes Care* 1997; **20:**537-44.
6. Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a mata-analysis of controlled clinical trials. *JAMA* 2001; **286:**1218-27.
7. Boule NG, Kenny GP, Haddad E, Wells GA, Sigal RJ. Meta-analysis of the effect of structured exercise training on cardio-respiratory fitness in type 2 diabetes mellitus. *Diabetologia* 2003; **46:**1071-81.
8. Ozdemir BA, Brownrigg J, Patel N, Jones KG, Thompson MM, Hinchliffe RJ. Population based screening for the prevention of lower extremity complications in diabetes. *Diabetes Metab Res Rev* 2012; Dec 25. doi: 10.1002/dmrr.2383. [Epub ahead of print] PubMed PMID: 23280992.
9. Aguilar R. Managing type 2 diabetes in men. *J Fam Pract* 2012; **61**(6 Suppl):S16-21. Review. Pub Med PMID: 22670243.
10. Klein MH, Greist JH, Gurman AS, Neimeyer RA, Lesser DP, Bushnell NJ, *et al.* A comparative out-come study of group psychotherapy vs. exercise treatments for depression. *Int J Ment Health* 1985;**13:**148-77.
11. Greist JH, Klein MH, Eischens RR, Faris J, Gurman AS, Morgan WP. Running as treatment for depression. *Comp Psychiatry* 1979; **20:**41-54.
12. Martinsen EW, Medhus A, Sandvik L. Effects of aerobic exercise on depression: a controlled study. *BMJ*1987; **291:**109.
13. Lippmann HI. Physiatrist's role in care of diabetes. *N Y State J Med* 1966; **6:** 1649-50. Pub Med PMID: 5219801
14. Care.diabetesjournals.org/content/26/12/3230.
15. National Institute of Diabetes and Digestive and Kidney diseases: Diabetes prevention program: Available from: URL:Diabetes.niddk.nih.gov/dm/pubs/prevention program
16. Diabetes care 1997 Apr: 20 (4): 537-44 Available from: URL: www.ncbi.nlm.nih.gov/pubmed/9096977
17. Sports fitness advisor: The FITT Principle of Training: Available from: URL:www.sport-fitness-advisor.com/fitt-principle.html
18. US Department of Health and Human Services: Physical activity and Health: A Report of the Surgeon General, Atlanta, GA, Centers for Disease Control and Prevention, 1996
19. American Diabetes Association: Standards of Medical Care for patients with diabetes mellitus (Position Statement). Diabetes Care, Volume 32, Supplement 1, S 26, January 2009.
20. 2004 American diabetes association, Instructor's Guide, Physical Activity and exercise,120.
21. Diabetes care, volume 26, supplement I, January 2003, S74-S75.
22. American Heart Association, The Committee on Exercise: Exercise Testing and Training of Apparently Healthy Individuals. A Hand book for Physicians. American Heart Association, 1972.
23. Wenger, NK, Hellerstein HK. Rehabilitation of the Coronary Patient. New York, Wiley, 1984.
24. Ivy JL. Role of exercise training in the prevention and treatment of insulin resistance and non-insulin-dependent diabetes mellitus, *Sports Med* 1997;**24:**321-36.
25. Dunstan DW, Daly RM, Owen N, Jolley D, de Court, Shaw J, *et al*. High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. *Diabetes Care* 2002; **25:**1729-36.
26. Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, *et al* A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care* 2002; **25:**2335-41.
27. Sigal RJ, Kenny GP, Boule NG, Wells GA, Prud'homme D, Fortier M, *et al* Effects of aerobic training , resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. *Ann Intern Med* 2007; **147:**357-69.
28. Delorme TL, Watkins A. Progressive Resistance Exercise. New York: Appleton-Century, 1951.

29. Delorme T, Watkins A.Technics of progressive resistance exercise. *Arch Phys Med Rehabil* 1948 ; **29:** 263.

30. Sanders M, Sanders B. Mobility: Active-resistive training. In: Gould, J, and Davies, editors. G (eds): Orthopedic and Sports Physical Therapy. St Louis: CV Mosby, 1985.

31. International Diabetes Center, Safe and Healthy Exercise for Patients with Diabetes. 2001.

32. Aiello LP, Wong J, Cavallerano J, Bursell SE, Aiello LM. Retinopathy. In: Ruderman N, Devlin JT, Kriska A, editors. Alexandria, VA, Handbook of Exercise in Diabetes. 2nd ed. American Diabetes Association, 2002: 401-13

33. Vinik A, Erbas T. Neuropathy. In 2nd ed. Ruderman N, Devlin JT, Kriska A editors Hand-book of Exercise in Diabetes. . Alexnadria VA: American Diabetes Association, 2002: 463-96.

34. Levin ME. The diabetic foot. In Ruderman N, Devlin JT, Kriska A editors. Hand-book of Exercise in Diabetes. 2nd ed. Alexandria VA: American Diabetes Association, 2002: 385-99.

35. Vinik A, Erbas T Neuropathy. In Hand-book of Exercise in Diabetes. Ruderman N, Devlin JT, Kriska A , editors. 2nd ed. Alexnadria VA: American Diabetes Association, 2002: 463-96.

36. Wackers FJ, Young LH, Inzucchi SE, Chyun DA, Davey JA, Barrett EJ, *et al*. Detection of silent myocardial ischemia in asymptomatic diabetic subjects: the DIAD study. *Diabetes Care* 2004; **27:**1954-61.

37. Valensi P, Sachs RN, Harfouche B, Lormeau B, Paries J, Cosson E. Predictive value of cardiac autonomic neuropathy in diabetes patient with or without silent myocardial ischemia. *Diabetes Care* 2001;**24:**339-43.

38. Mogensen CE: Neuropathy. In: Ruderman N, Devlin JT, Kriska A, editors. Hand-book of Exercise in Diabetes. 2nd ed. Alexandria VA: American Diabetes Association, 2002: 433-49

39. Astrand P Rodhal K. Text book of Work Physiology, Physiological basis of Exercise. 3rd ed. New York: Mc-Graw-Hill, 1986.

40. McArdle WD, Katch FI, Katch VL. Exercise Physiology: Energy, Nutrition, and Human Performance. 3rd ed. Philadelphia: Lea & Febiger, 1991.

41. Wenger. NK, Hellerstein HK. Rehabilitation of the Coronary Patient. New York: Wiley, 1984.

42. Butler RM, *et al*. The cardiovascular response to circuit weight training in patients with cardiac disease. *J Cardiopulm Rehabil* 1987; **7:**402.

43. McArdle WD, Katch FI, Katch VL. Exercise Physiology: Energy, Nutrition, and Human Performance, 3rd ed. Philadelphia: Lea & Febiger, 1991.

44. American Diabetes Association. Standards of Medical Care for patients with diabetes mellitus (Position Statement). Diabetes Care, **32:** 2009.

Editorial

Can Interventional Physiatry Overrule Conventional Physiatry?

Over last one decade there is a boom of Interventional Physiatry all over India. Specially the younger generation of Indian Physiatry community is showing tremendous inclination towards different types of interventions either blind or guided (USG/ C arm etc) procedures in different centres of PMR or different workshops during CME or conferences. This is very healthy, specific bold approach to practise the subject. The question is that "Are we neglecting the strength of conventional approach at the same time?"

From the very beginning the approach of Physiatry is primarily based on team work. We have plenty of non-pharmacological weapons in the armamentarium of PMR to tackle different clinical impairments. Those are our strength not weaknesses of our subject. Can we mingle up the other modes of treatment options with interventions?

Spinal pain due to PIVD is one of the most common conditions in consideration for an interventional Physiatrist. But as per different literature any decision regarding intervention should not be taken before practising Mackenzie's principle for at least forty-eight hours apart from the red flag signs. Not only that but also intervention has no role in mild pain or moderate pain of PIVD who are maintaining their ADL. If we want to treat spondylosis or spondylolisthesis then we cannot ignore the role of braces, flexion exercises, hamstring stretching exercise until today. Is there any scope of intervention in management of most common aetiology of non-inflammatory LBP like lumbosacral strain in younger patients? Even there is some role of conservative care in facet arthropathy or spinal canal stenosis etc. As per the evidences nobody can deny the role of exercise therapy in spinal pain remission and prevention of recurrence. Most importantly restoration of normal biomechanics by postural care is the key element to manage spinal pain.

If we consider the regional pain management then also interventions cannot defy conservative care. Simple practice of exercise therapy, orthoses and workstation modification can actually eliminate lots of local infiltrations in tennis or Golfer's elbow. Modifications of workstation and posture care are the key elements in management of any cumulative trauma disorder. Actually most of the non-inflammatory joint pain can actually better dealt by different modalities and pharmacological means. Is there any scope of intervention in knee pain due to patellofemoral joint pain syndrome (most common aetiology of anterior knee pain), soft tissue injuries like ACL, PCL, menisci injuries not amicable by surgery.

Lastly the decision regarding interventions like chemoneurolysis of botulinum toxin block should not be undertaken before a proper trial of orthoses, antispaticity exercise therapy and medications. Not only that but also the role of tone inhibiting orthoses and exercises cannot be ignored after a block.

Hence we should not underestimate the strength of conservative care like posture modification, orthoses, modalities or exercise therapy. Lots of other specialists are doing similar types of interventions. If we can mix up those procedures with our appropriate modalities the much superior results may be achievable. Timely appropriate intervention is much specific treatment option. So interventional Physiatry cannot overrule conventional approach but both are complementary to each other.

Rajesh Pramanik

Original Article

Evaluation of the Effects of Lumbosacral Corset on the Patients with Chronic Non-specific Low Back Pain

Md. Abdus Shakoor[1], Mohammad Tariqul Islam[2], Md. Muhibbur Rahman[3], Md. Shahidur Rahman[4], Md. Moyeenuzzaman[5]

Abstract

A randomised clinical trial was conducted in the Department of Physical Medicine & Rehabilitation (PMR), Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. A total of 81 patients having chronic LBP were included according to the selection criteria. Out of them, 31 (38.3%) were male and 50 (61.7 %) were female in a ratio of 1: 1.61. The mean age of the patients in study was 41.65 ± 8.41years. Female persons were affected in their earlier ages (between 30 and 45 years) than male. Most of the patients were housewives (54.3%). The patients were divided randomly into two groups by the way of lottery for the clinical trial. Group-A patients were treated with NSAIDs, activities of daily living instructions (ADLs) and lumbosacral corset and group-B patients were treated with NSAIDs and ADLs. The patients were followed up weekly for five weeks and significant improvement was recorded after the treatment in both the groups (p=0.001). In comparison between two groups, it was found that there was no significant improvement in pre-treatment, after 1st week and after 3rd week. A little bit improvement was found in group-A patients than group-B after 4th week (p= 0.06). But finally, there was significant improvement in group-A than group-B patients after 5th week (p=0.005). So, it may be concluded that both the treatment is effective for the patients with chronic non-specific LBP. But the patient may be more benefited if lumbosacral corset is used as an adjunct to NSAIDs.

Key words: **Low back pain, lumbosacral corset.**

Introduction:

Low back pain (LBP) is most common, experienced at some time by up to 80 % of the population[1]. Pain in the area between the lower rib case and gluteal folds is called low back pain[2]. LBP is the most common medical cause of inability to work in the western countries[3] and the leading cause of disability in people under the age of 45 years [4,5] specially most prevalent medical disorders in industrialised societies[6]. Disability related to back pain has increased exponentially over the past 20 years due, at least in part, to psychological and social factors that influence adaptation to back pain early in the process[7]. Defining LBP is difficult, but it refers to a symptom complex in which pain is localised to the lumbar spine or referred to the leg or foot and majority of cases of the backache is associated with some abnormality of the intervertebral discs at the lowest two levels of the spine[8-9]. Abnormalities in the lumbar spine are common and degenerative changes virtually be found in all older people[10]. Despite its high prevalence, LBP remains poorly understood and inadequately treated. This is due to the heterogeneity of the patients' population, and the lack of a simple and easy to apply, clinically useful system for characterisation of patients[11]. Non-specific LBP of mechanical origin is second only to the common cold as a cause of self-limiting symptoms and disability in the community[12], 70% of patients with an episode of LBP recover within one month, and 90% within 3 months. Only 4% patients will have symptoms larger than 6 months. This

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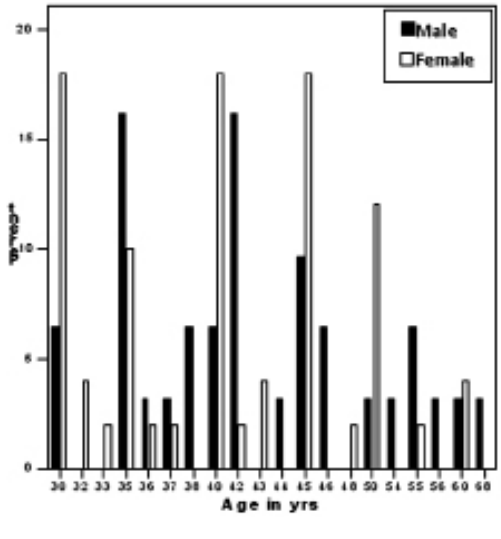
relatively small number of patients' account for 85% to 90% of funds spent on the treatment and compensation for LBP. Only 50% of these chronically symptomatic patients return to work, according to one study[6]. LBP affects 60%-80% of US adults at some times during their lives, up to 50% have pain within a given year, in 5%-10% of patients with LBP become chronic[13]. Another study[14] in the USA it is found that LBP is the most common single musculoskeletal complaint and a major cause for being out of work, resulting in billions of dollars in lost wages and compensations payment annually. It was estimated in 1997 that the financial cost of LBP accounting for medical bills compensation and forfeited productivity, was somewhere between $38 billion and $ 50 billion in the United States[15]. The treatment and management of LBP is not simple. There are many divergent ways of management of LBP. Chronic LBP is resistant to treatment, and patients are often referred for multidisciplinary treatment. Current multidisciplinary bio-psychosocial rehabilitation regards disabling chronic pain as the result of multiple interrelating physical, psychological, and social or occupational factors[16]. Bangladesh is a developing and densely populated country with very limited resources and poor management. So, for various reasons we cannot manage a huge number of disabled patients with low back pain with our present resources and management system. Lumber corset is used to support the lumber spine as a physical modality which is used to fix the lower back and abdomen. Fixation of the lower back reduces LBP by 3 actions: (1) limiting the movement of painful muscles, intervertebral joints, intervertebral discs, and fractured vertebral bodies, (2) maintaining good posture and (3) reducing the mechanical load on the lower lumbar spine[17].

Several studies of pain and instability of the lower extremities indicated that the wearing of elastic lumbar orthoses improved the patients' feeling of joint stability and improved the subject's joint position sense by increasing afferent proprioceptive input *via* the mechanoreceptors of the skin and it can affect LBP and instability or can improve restricted proprioception[18]. But still the efficacy is not been established in case of chronic non-specific LBP. So, the aims of this study is to find out the effects of lumbosacral corset on the patients with chronic non-specific LBP.

Materials and Methods:

This randomised clinical trial was conducted in the Department of Physical Medicine & Rehabilitation (PMR), Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from April 2008 to March 2009. Patients having chronic non-specific LBP were selected according to the following clinical criteria: patients of both sexes, age ≥ 30 years and ≤70 years, having complaints of LBP for more than three months excluding any specific chronic cause, having no evidence or history of nephropathy or peptic ulcer diseases. Before starting the study ethical clearance was given by the institute and informed consent was taken from the patients properly. A total of 100 patients were selected for the study according to the selection criteria. Selection was done randomly by the way of lottery and divided into two groups. Group-A (n=50) patients were treated with NSAIDs, ADLs and lumbosacral corset and group-B (n=50) patients were treated with NSAIDs and ADLs. Naproxen (250mg) twice daily orally was prescribed in both the groups with capsule omeprazole (20mg) twice daily. All the drugs were given from the same company to avoid any discrepancy of efficacy. Activities of daily living (ADL) instructions were given to protect the back from pain in both the groups. Lumbosacral corset was given to the group-A only and advices were given to these patients to use corset during journey and activity and not to use it during sleeping. The patients were followed up weekly for five weeks and the outcome were recorded accordingly. Assessment of pain intensity, disability, and physical impairment were done by using visual analogue scale, Schober's test, Oswastry Disability Index and Modified Zung Index. The numerical data were analysed statistically by using the SPSS-package program (verson-18) for Windows. Student's 't' test was done to see the level of significance.

Fig I: *Distribution of Sex in Relation to Age of the Study Subjects (n= 81)*



Results:

A total of 100 patients were included but [19] patients were dropped out from the study because they cannot follow the instructions of daily living properly. So, 81 patients followed the treatment allocated for them properly. Out of them, 31 (38.3 %) were male and 50 (61.7%) were female and male: female ratio was 1: 1.61. The mean age of the patients was 41.65 ± 8.41years. Female persons were affected in their earlier ages ie, between 30 and 45 years, (Fig1) than male. Before admission into the clinical trial, baseline characteristics of the patients of the two groups were compared and found identical (Table 1). All patients are married except one who was unmarried, maximum patients gave the history of gradual onset of the pain (80.2 %), some had history of sudden onset (13.6%) and a few gave the history of pain after trauma (6.2 %). Pain of most of the patients relieved by rest (56.8 %) and lying flat (43.2 %) and aggravated by activity. Maximum patients had the pain intermittent in character (84 %) but 16% patients had the pain of constant in character. There was significant improvement after treatment in group-A. In respect to time point improvement, marked improvement was started to occur after one week (p = 0.001, 95% CI = 2.89 to 5.77, Table 2). Improvement gradually increased day by day and after the end of treatment, highly

Table 1- *Baseline Clinical and Investigation Criteria of the Patients with LBP*

Group	Age in years	Height in cm	Weight in kg	Pulse/ minute	SBP in mm Hg	DBP in mmHg	ESR after 1st hour (mm)	Blood sugar (2HP-PBS in mmol/l)	Serun. uric acid
A (n=42)	42.81 ± 9.05	155.91 ± 8.92	57.90 ±10.01	80.88 ±5.49	122.57±14.28	76.98 ±7.77	23.90 ± 13.04	6.47 ± 2.16	4.36 ± 1.13
B	40.41	160.07	55.61	79.44	123.85±12.95	75.64	22.15	6.76	4.22
(n=39)	± 7.59	±4.53	±5.97	±563		±10.46	± 16.27		± 0.94
p-value	0.19	0.01	0.21	0.24	0.67	0.51	0.59	0.51	0.55
95% CI	-1.28 to 6.08	-7.27 to -1.05	-1.33 to 5.91	1.23to -1.02	-7.29 to 4.74	-2.77 to 5.44	- 4.81 to 8.31	-1.17 to 0.59	-0.32 to 0.60

The results are expressed in mean ± standard deviation (SD). n = Number of the patients participated in the clinical trial, SBP-Systolic BP, DBP-Diastolic BP.

Table 2- *Treatment Responses on Different Time Points in Group-A (n = 42)*

Time-point score	Mean ± SD	P - value	95% CI
Pre-treatment Vs W1	30.71 ±7.19 Vs 26.38	0.001	2.89 to 5.77
Pre-treatment Vs W2	30.71 ± 7.19 Vs 23.84 ± 4.96	0.001	5.46 to 9.01
Pre-treatment Vs W3	30.71 ± 7.19 Vs 20.69 ± 4.67	0.001	7.72 to 12.31
Pre-treatment Vs W4	30.71 ± 7.19 Vs 18.19 ± 5.18	0.001	10.08 to 14.96
Pre-treatment Vs Post-treatment	30.71 ± 7.19 Vs 14.88 ± 4.73	0.001	13.41 to 18.25

The results are expressed in mean ± standard deviation (SD).
n = Number of the patients participated in the clinical trial.
W= weak.

Table 3 - *Time-point Treatment Response in Group-B (n = 39)*

Time-point	Score in mean ± SD	p-value	95 % CI
Pre-treatment Vs W1	30.41± 7.35 Vs 27.74 ± 7.95	0.001	1.17 to 4.17
Pre-treatment Vs W2	30.41± 7.35 Vs 24.62 ± 7.64	0.001	4.04 to 7.54
Pre-treatment Vs W3	30.41± 7.35 Vs 22.67 ± 7.19	0.001	5.85 to 9.63
Pre-treatment Vs W4	30.41 ± 7.35 Vs 20.62 ± 6.16	0.001	7.55 to 12.03
Pre-treatment Vs Post-treatment	30.41± 7.35 Vs 18.44 ±6.03	0.001	9.88 to 14.06

The results are expressed in mean ± standard deviation (SD).
n = Number of the patients participated in the clinical trial.
W=weak

significance improvement was found (p=0.001, 95% CI = 13.41 to 18.25, Table 3). Significant improvement also found after treatment in group-B. In respect to time point improvement, marked improvement was started to occur after one week (p = 0.001, 95 % CI = 1.17 to 4.17, Table 3). Improvement gradually increased day by day and after the end of treatment, there was highly significance of improvement (p= 0.001, 95 % CI= 9.88 to 14.06, Table 3). In comparison between two groups, it was found that there was no significant difference in improvement up to third week. But more improvement was found in group-A than group-B after 4th week (p= 0.06). Finally, there was more improvement in group-A than group-B after 5th week (p= 0.005, Table 4). This indicates that NSAIDs are effective for the improvement of the patients with chronic LBP but when lumbar corset is used as an adjunct to NSAIDs, more improvement was found than only NSAIDs receiving group.

Table 4 - *Comparative Improvement of Symptoms between Group-A and Group-B in Different Time Points*

Group	Score at W0	Score at W1	Score at W2	Score at W3	Score at W4	Score at W5
A (n=42)	30.71±7.19	26.38 ± 5.45	23.48 ± 4.96	20.69 ± 4.67	18.19 ± 5.16	14.88 ±4.73
B (n=39)	30.41±7.35	27.74 ± 7.95	24.62 ± 7.64	22.67 ± 7.19	20.62 ± 6.16	18.44 ±6.03
95 % CI	-2.91 to-3.52	-4.41 to 1.68	-4.02 to 1.74	-4.69 to-0.73	-4.95 to 0.10	-5.97to –1.13
p-value	0.85	0.37	0.43	0.15	0.06	0.005

The results are expressed in mean ± standard deviation (SD).,n = Number of the patients participated in the clinical trial. W=weak

Discussion:

In this study, significant improvement of symptoms in both the groups began to appear at the end of first week (p= 0.001) and it was increasing day by day and finally there was highly significant improvement found (p= 0.001). But, in comparison between groups no significant difference of improvement was found at up to third week. After 4th week, more improvement was found in group-A than group-B (p= 0.06). In group-A, we prescribed lumbosacral corset and found more improvement, but in group=B, we prescribed NSAIDs without lumbosacral corset. At the end of treatment, there was more significant improvement in group-A than group-B patients after 5th week (p= 0.005). This indicates that NSAIDs are effective for the improvement of the patients with chronic LBP but when lumber corset is used to protect the back with NSAID, more improvement was found than the patient receiving only NSAID. Chard and Dieppe[19] reported in a review that aids and bracing is moderately effective in osteo-arthritis. This is in favour of this study. In our study, patients were also advised to protect the back by maintaining activities of daily living (ADLs). In some other studies [20,21] it was also found that ADL has beneficial effects on chronic LBP. These all findings support the results of the present series, although lumbar orthosis has shown biomechanical effects on trunk performance, including stiffening of the torso. First, wearing a corset can restrict the hyperextension of the lumbar spine, which may be a pain generating manoeuvre associated with spondylosis or facet syndrome. Second, wearing an hard corset can reduce the magnitude of lumbar rotation and increase hip rotation, changes that may benefit patients with conditions of lumbar disc degeneration[22]. A study[23] over 102 patients showed 64 were females (62.7%) and 76 (74.5%) patients had worn the lumbar corset for more than 1 year of duration where ninety (88%) patients normally wore the corset all day or most part of the day and there was more improvement in lumbar corset user group. This also suggests that wearing lumbar corset is useful to reduce chronic LBP.

Actually, lumbar support in the form of lumbar corset is used as an adjunct to NSAIDs therapy for pain management and in the maximum study, there is better tolerability and better improvement is found. In some another reviews, it was found that exercise is as effective intervention[19] and some reported that exercise is as effective as NSAIDs [24,]. In a review[25] it was reported that exercises seemed not to be better than bed rest and other conservative treatments like traction, manipulation, hot packs, or corsets. So it may be said that conservative treatment like lumbosacral corset is as effective as exercise.

Conclusions:

It may be concluded that both the treatment (NSAID and lumbosacral corset) is effective for the treatment of chronic non-specific LBP. But the patient may be more

benefited if lumber support in the form of lumbosacral corset is used as an adjunct to NSAIDs.

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References:

1. Hellmann DB, Stone JH. Arthritis and musculoskeletal disorders. In: Tierney Jr LM, McPhee SJ, Papadakis MA, editors. Current Medical Diagnosis & Treatment. New York: McGraw –Hill, 2002; **20:** 833-89.
2. Dixit R. Low back pain. In: Firestein GS, Budd RC, Gabriel SE, Mcinnes IB, O'Dell JR, editors. Kelley's Text Book of Rheumatology. Philadelphia: Elsevier Inc, 2013; **47:** 665.
3. Allen CMC, LeuckmCJ. Diseases of the nervous system. In: Haslett C, Chilvers ER, Hunter JAA, Boon NA, editors. Davidson's Principles and Practice of Medicine. Edinburgh: Churchill Livingstone, 1999; **14:**923-1023.
4. Wilson DJ, Hickey KM, Gorham JL, Childers MK. Lumber spinal moments in chronic back pain patients during supported lifting: a dynamic analysis. *Arch Phys Med Rehabil* 1997; **78:** 967-72.
5. Engstrom JW. Back and neck pain. In: Braunwald E, Hauser SL, Fauci AS, Longo DL, Kasper DL, Jameson JL. editors. Harrison's Principles of Internal Medicine. New York-London: McGraw-Hill, 2001; **16:**79-90.
6. Levine DB, Leipzig JM. The painful back. In: McCarty DJ, Koopman WJ, editors. Arthritis and Allied Conditions. Philadelphia-London: Lea & Febiger, 1993; **2:** 1583-600.
7. Moore JE, Korf MV, Cherkin D, Sounders K, Lorig K. A randomized trial of a cognitive behavioural program for enhancing back pain self care in a primary care setting. *Pain* 2000; **88:** 145-153.
8. Frank A. Low back pain, education and debate. *BMJ* 1993; **306:** 901-9.
9. Solomon L, Warwick DJ, Nayagam S. Back pain. In: Apley's System of Orthopaedics and Fractures. Arnold, A member of the Hodder Heading Group, London. Co-published in the USA by Oxford University Press Inc., New York, 2001; **18:** 371-404.
10. Porter RW. Management of Back Pain. 2nd ed. Edinburgh-Tokyo: Churchill Livingstone, 1993; **2:**13-27.
11. BenDebba M, Torgerson WS, Long DM. A validated, practical classification procedure for many persistent low back pain patients. *Pain* 2000; **87:** 89-97.
12. Nuki G, Ralson SH, Luqmani R. Diseases of the connective tissues, joints and bones. In: Haslett C, Chivers ER, Hunter JAA, Boon NA. editors. Davidson's Principles and Practice of Medicine. Edinburgh: Churchill Livingstone, 1999; **12:** 801-76.
13. Lahad A, Malter AD, Berg AO, Deyo RA. The effectiveness of Four interventions for the prevention of Low Back Pain. *JAMA* 1994; **272:**1286-91.
14. Milhous RL, Hough LD, Frymyer JW, Ruess JM, Gallagher RM, Wilder DG, *et al*. Determinants of vocational disability in patients with low back pain. *Arch Phys Med Rehabil* 1989; **70:** 589-98.
15. Casey PJ, Weinstein JN. Low back pain. In: Ruddy S, Jr EDH, Sledge CB. editors. Kelley's Textbook of Rheumatology. Philadelphia- London: WB Saunders, 2001; **36:** 509-24.
16. Guzman J, Esmail R, Karjalainen K, Malmivaara A, Irvin E, Bombardier C. Multidisciplinary rehabilitation for chronic low back pain: sytematic review. *BMJ* 2001; **322:**1511-6.
17. Hayashi Y. Physical therapy for low back pain. *JMAJ* 2004; **47:** 234-9.
18. Vogt L, Pfeifer K, Portscher M, Banzer W. Lumbar corsets: their effect on three-dimensional kinematics of the pelvis. *J Rehabil Res Dev* 2002; **37:**495-9.
19. Chard J, Dieppe P. The case for nonpharmacologic therapy of osteoarthritis. In: Cronstein BN editor. Current Rheumatology report. Philadelphia: Current Science Inc, 2001; **3:** 88-94.
20. Ahmed MS, Shakoor MA, Khan AA. Evaluation of the effects of shortwave diathermy in patients with chronic low back pain. *Bangladesh Med Res Counc Bull* 2009; **35:** 18-20.
21. Rahman MM. Low back pain – clinical analysis 342 cases. *Bangladesh Med Coll J* 1999; **4:** 67-71.
22. Hashimoto K, Miyamoto K, Yanagawa T, Hattori R, Takaaki A, Matsuoka T. Lumbar corsets can decrease lumbar motion in golf swing. *J Sports Sci Med* 2013; **12:** 80-7.
23. Khan AM, Salih M, Levack B. Lumbar corsets: compliance and effectiveness for lower back pain. *Hong Kong J Orthop Surg* 2002;**6:**34-8.
24. Brandt KD. Nonsurgical management of osteoarthities, with an emphasis on nonpharmacologic measures. *Arch Fam Med*1995; **4:** 1057-64.
25. Koes BW, van Tulder MW, Peul WC. Clinical review - diagnosis and treatment of sciatica. *BMJ* 2007; **334:**1313-7.

Pictorial CME

Missed Opportunity for Thrombolysis in a Patient of Massive MCA Territory Stroke

Pramanik R

A 45-year old male presented to PMR OPD with left sided complete hemiplegia (Day 3) due to stroke. Then he was admitted in PMR Indoor for neurorehabilitation. On initial evaluation he was haemodynamically stable. Motor power of his left upper limb and lower limb was 0/5. He was on catheter and IV fluid on the day of admission. When we reviewed the CT scan of his brain on Day 1 of his presentation we saw a right MCA thrombus (Fig 1). Unfortunately nobody sent the patient for thrombolysis. Eventually the patient landed up to PMR OPD for further rehabilitative management for dense hemiplegia. This was well corroborated by a repeat CT scan done in our ward which picked up a massive right MCA Infarct (Fig 2)

Over next 3 weeks patient was treated with all forms of physiatric management. On 4th week his FIM score improved from 30/126 to 94/126. Patient was still dependent for his mobility (Fig 3) and toilet care. Muscle power improved to only grade 2/5. Then the patient was discharged home and advised to carry on supervised therapy at home.

Hence it was unfortunate for the patient to carry on his daily life with some amount of expected residual disability which possibly might be well prevented by a proper timely referral for neurothrombolysis.

Fig 1

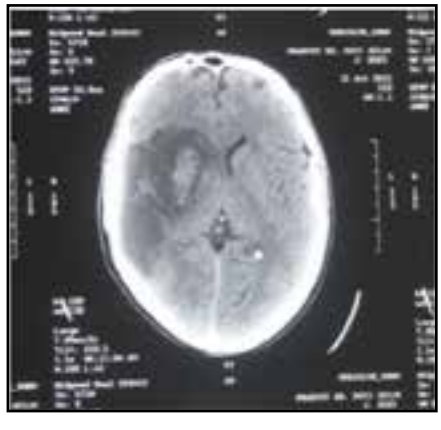

Fig 2

Fig 3

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Original Article

Quality of Life in Postmenopausal Women and Its Correlation with Bone Mineral Density

Minggam Pertin[1], Romi Singh Nongmaithem[2], C Zonunsanga[3], Hmingthanmawii[4] , Chongreilen Chiru[5], Yengkhom Jotin Singh[6]

ABSTRACT

Aim: To evaluate the quality of life in postmenopausal women and its correlation with bone mineral density.

Study design: Cross-sectional study.

Duration of the study: October 2012 to September 2014.

Settings: Physical Medicine and Rehabilitation Department, Regional Institute of Medical Sciences, Imphal.

Study population: Postmenopausal women who attended the department during the study period.

Materials and Methods: Quality of life was assessed using WHOQOL-BREF questionnaire, a validated brief version of the WHOQOL-100. Bone mineral density (BMD) in the lumbar spine, femoral neck and trochanter were measured using dual energy x-ray absorptiometry (DEXA) scan – GE Lunar model.

Results: A total of 125 patients were studied. The mean t-scores in lumbar spine, femoral neck and trochanter were -2.550 ± 1.209, -1.831 ± 0.921 and -1.621 ± 1.064 respectively. The mean BMD (g/cm2) in lumbar spine, femoral neck and trochanter were 0.867 ± 0.144, 0.789 ± 0.131 and 0.682 ± 0.139 respectively. The mean overall WHOQOL score was 57.68±10.07. There were statistically significant positive association of WHOQOL score with the BMDs in lumbar spine, femoral neck and trochanter ($p < 0.05$). Multivariate regression showed significant relation of overall WHOQOL score with BMD lumbar spine (b=0.229; R2=0.119), BMD femoral neck (b=0.285; R2=0.129), and BMD trochanter (b=0.245; R2=0.119).

Conclusion: BMDs in the lumbar spine, femoral neck and trochanter had a positive correlation with quality of life scores. BMD also had a good predictive value in determining the quality of life in postmenopausal women.

Key words: **Quality of life, postmenopausal women, osteoporosis, WHOQOL-BREF, bone mineral density.**

Introduction:

Osteoporosis is a disease of impaired mineralisation of bone leading to a decreased bone mineral density (BMD), low bone mass and deterioration in the bone micro-architecture particularly trabecular bone thereby making the bone more fragile and prone to fractures[1]. Due to peri-menopausal decreased level of oestrogen, osteoclastic activity is increased due to increase level of tumour necrosis factor (TNF-alpha) and interleukins (IL-1 and IL-6) and thereby causing increased bone resorption. Low BMD along with many other risk factors such as poor nutrition, reduced lean and fat tissue, reduced physical activity, general frailty, poor balance and slowed gait speed have been known to increase the risk of fractures[2].

Quality of life is defined as individual's perceptions of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns[3]. It refers to a subjective evaluation which is embedded in a cultural, social and environmental context and focuses on the respondent's perceived quality of life.

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Low BMD has been correlated with the degree of skeletal pain pertaining to the daily activities of living. Postmenopausal women experience back pain both of lumbar and thoracic vertebral origin and experience sleep disturbance, frailty, weakness, difficulty with balance and difficulty in activities of daily living. These factors thus have a great impact on the overall quality of life[4]. Primary osteoporosis is a health issue with multi -dimensional involvement in the daily life activities due to pain, fatigue, depression or self image. These factors affect self dependence, and limit the walking ability and the activities of daily living and thereby largely affecting the quality of life[5]. Osteoporosis constitutes a problem not only for patients with osteoporosis but also for their families and the society as a whole. The approach to a patient suffering from osteoporosis has always been targeting symptomatic treatment of complaints and fracture prevention. Identifying and understanding the right factors which contribute to the disabilities will enable us make a management plan that would target not only on symptomatic management but on improvement of the overall quality of life of a person.

Studies on quality of life measures in postmenopausal women have been conducted elsewhere. Only few studies are available on the correlation between bone mineral density and quality of life in postmenopausal women. The present study was an attempt to find out the quality of life in postmenopausal women and its correlation with bone mineral density.

Materials And Methods:

The study was a cross-sectional study and subjects were recruited from consenting postmenopausal women coming with various complaints of pain, attending the Department of Physical Medicine and Rehabilitation, Regional Institute of Medical Sciences, Imphal between October 2012 and September 2014. All the patients recruited for the study were subjected to clinical, laboratory, radiological examinations and questionnaires on quality of life. Approval of the Institutional Ethics Committee was taken before starting the study.

Exclusion criteria:

Patients with history of secondary causes of osteoporosis like hyperthyroidism, hyperparathyroidism, mal-absorption, malnutrition and chronic liver diseases, patients currently on medications for osteoporosis and on corticosteroids, premenopausal hysterectomy, arthritic conditions like rheumatoid arthritis, patients with cognitive impairment, unwilling patients and with chronic comorbid conditions such as metabolic bone diseases, heart disease, metastasis, stroke and vascular disease were excluded from the present study.

Quality of life (QOL):

Besides clinical examination, quality of life of the patients was assessed using the WHOQOL-BREF, a brief version of World Health Organisation Quality of Life-100 questionnaire[6]. The WHOQOL-BREF is a set of 26 questions covering 24 facets of quality of life questions and the other 2 questions are of overall quality of life and general health. These 24 facets are incorporated into 4 domains – physical health, psychological health, social health and environmental health. A time frame of two weeks is indicated in the assessment. The domain scores are scaled in a positive direction from 0 to 100 and higher the score represents higher the quality of life of the individual.

Bone mineral density measurement:

Bone mineral density measurements at lumbar spines (L1-L4 AP view), femoral neck and trochanter (in g/ cm^2) were assessed using DEXA scan (dual energy x-ray absorptiometry) – GE Lunar model. Evidence of osteoporosis and its severity according to WHO guidelines [7] were evaluated from the t-score values in the DEXA scan report in the following manner:

- Normal: ≥ -1
- Osteopenia: -1 to -2.5
- Osteoporosis: ≤ -2.5

Statistical analysis:

Data collected from the clinical examination, DEXA scan, laboratory investigations and WHOQOL-BREF scores were entered in microsoft excel and analysed using SPSS version 16. Independent sample t test was applied to compare the means of overall WHOQOL scores in relation to the t-scores in the lumbar spine, femoral neck and the trochanter. Pearson's correlation was applied to determine the correlation between the WHOQOL scores in each domain and BMD. Regression analysis of the WHOQOL scores and the BMD were done to find out the R2 value. A p-value of <0.05 was taken as statistically significant for all statistical tests.

Results And Observations:

There were a total of 125 postmenopausal women included in the study. Table 1 shows the demographic profile of the study population. The mean age of the

patients was 60.02 ± 9.223 years. Seventy-four (59.2 %) patients had reported with the complaints of low back pain followed by 22 patients (17.6 %) with knee pain. Back pain and knee pain comprised a total of 96 patients (76.8%). The mean duration of illness was 16.98±14.61 months (range 1-60 months). Fig1 shows the age group distribution; 53 women (42.4%) were in the 56-65 years age group, 36 (28.8%) women were in the 46-55 years and 32 (25.6%) women were in the age group more than 65 years. Fig 2 shows the occupation-wise distribution among the study population; 81 women (64.8%) were housewives, 21 (16.8%) had a sedentary lifestyle. Twenty-nine patients (23.2%) among the study population reported previous history of fracture.

Table 1- *Demographic Profile of the Study Subjects (n=125)*

Variables		Rank (mean ± SD)	No. of cases	Percentage
Age (in years)	60.02 ± 9.223			
Agewise distributions (in years)				
	≤ 45		4	3.2
	46-55		36	28.8
	56-65		53	42.4
	>65		32	25.6
Complaints	Back pain		74	59.2
	Knee pain		22	17.6
	Neck pain		17	13.6
	Others		12	9.6
Duration of complaints (in months)		1-60 (16.98±14.61)		

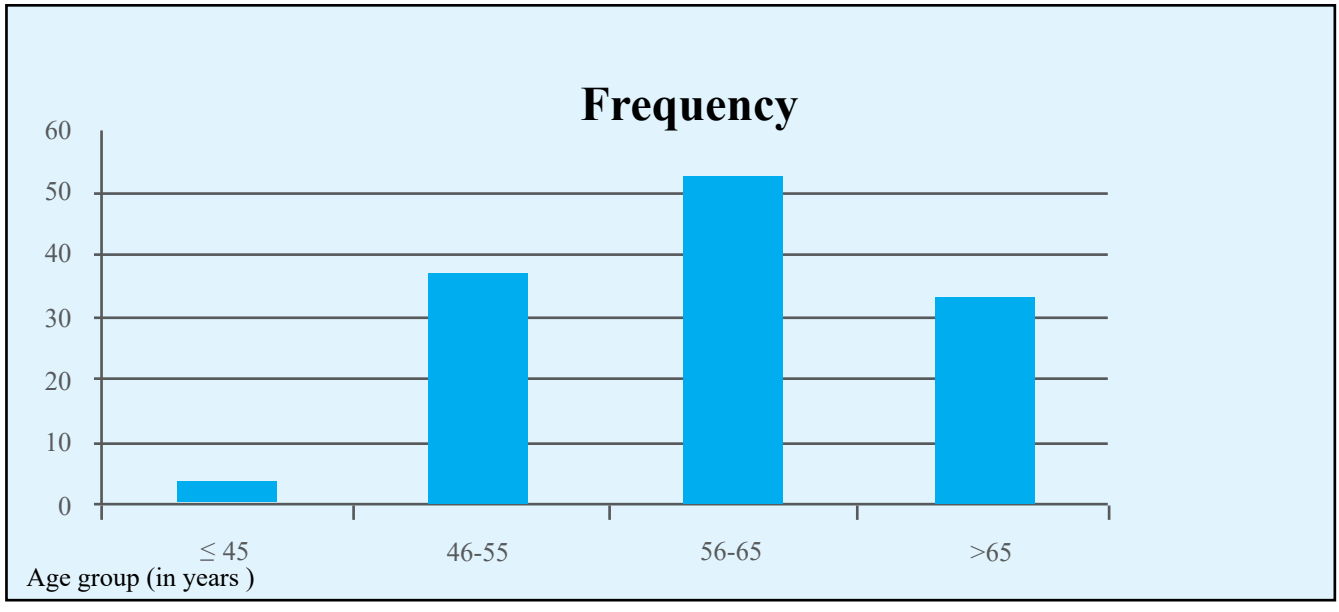

Fig 1 : *Agewise Distribution of Cases*

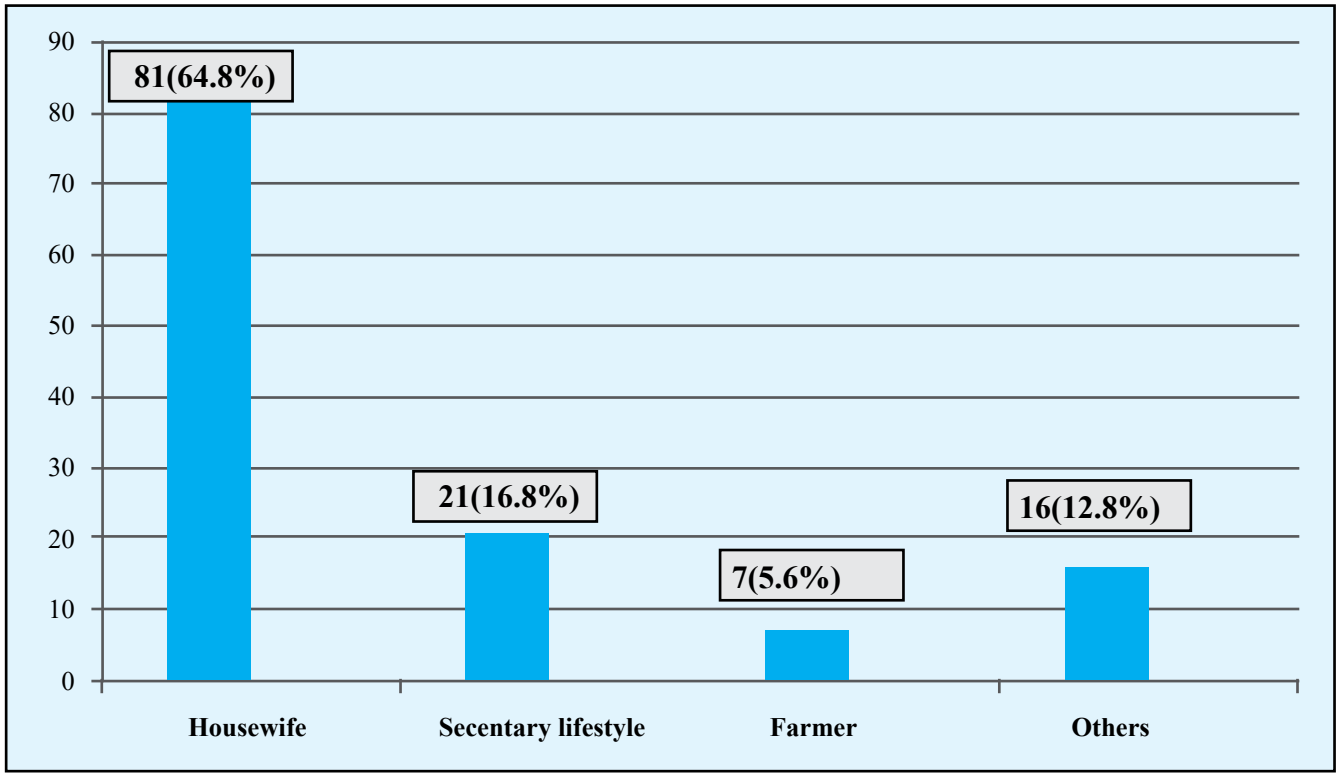

Fig 2: *Occupationwise Distribution of the Cases*

Table 2 - *Showing the Mean t-scores and Mean BMD at Lumber Spine (LS), Fenoral Neck (FN) and Trochanter (TR)*

Site	Mean t-score	Mean BMD (g/cm2)
LS	-2.550 ± 1.209	0.867 ± 0.144
FN	-1.831 ± 0.921	0.789 ± 0.131
TR	-1.621 ± 1.064	0.682 ± 0.139

Table 2 shows the mean t-scores and the mean BMDs (in g/cm2). The mean t-scores in the lumbar spine, femoral neck and trochanter were -2.550 ± 1.209, -1.831± 0.921 and -1.621 ± 1.064 respectively. Similarly, the mean BMD in the lumbar spine, femoral neck and trochanter were 0.867 ± 0.144, 0.789 ± 0.131 and 0.682 ± 0.139 in (gms/cm2) respectively.

Overall WHOQOL score and WHOQOL scores in different domains:

Table 3 shows the WHOQOL scores of the study group. The WHOQOL score was grouped into 2 sub-groups viz, those with score of < 50 and score of ≥ 50. All 4 domains were scored from 0 to 100. Domain 1, i.e. the physical domain had a mean score of 48.04±13.53 within a score range of 19 to 81. Out of the 125 women, 63 women (50.4%) scored <50. Domain 2, i.e. the psychological domain had a mean score of 58.50±10.02 within a score range of 38 to 88, 107 women (85.6%) had a score of ≥ 50. Domain 3, i.e. the social domain had a mean score of 59.38±9.59 within a score range of 31 to 75 and 119 (95.2%) women had a score of ≥ 50. Domain 4, i.e. the environmental domain had a mean score of 68.88±9.10 within a score range of 38 to 81 and 121 women (96.8%) had a score of ≥ 50. The overall WHOQOL score was computed by taking an average of scores of all the domains and overall WHOQOL mean score was found to be 57.68±10.07. Overall, 28 women (22.4%) had an overall WHOQOL score < 50 and 97 women (77.6%) had a score ≥ 50.

Relationship between mean overall WHOQOL score and t-scores in lumbar spine, femoral neck and trochanter:

Table 4 shows that the mean overall WHOQOL score was higher in the osteopenic group in comparison to the osteoporotic group in the lumbar spine, femoral neck and trochanter. In the osteopenic group, the mean WHOQOL score was 60.22±9.08, 58.61±8.64 and 58.93±8.72 in the LS, FN and TR respectively. In the osteoporotic group, the mean WHOQOL score was 54.64±10.43, 54.63±13.54 and 52.68±13.34 in the LS, FN and TR respectively. The associations were statistically significant in the lumbar spine and the trochanter only ($p < 0.05$).

Table 3 - *Overall WHOQOL Score and Domainwise WHOQOL Scores*

Variables		WHOQOL score			
		< 50	≥ 50		
	Mean	Frequency	Percentage	Frequency	Percentage
Overall WHOQOL	57.68±10.07	28	22.4	97	77.6
WHOQOL Domain 1	48.04±13.53	63	50.4	62	49.6
WHOQOL Domain 2	58.50±10.02	18	14.4	107	85.6
WHOQOL Domain 3	59.38±9.59	6	4.8	119	95.2
WHOQOL Domain 4	68.88±9.10	4	3.2	121	96.8

Table 4 - *Showing the Relationship of the Mean WHOQOL Score to the Mean t-scores in Lumbar Spine, Femoral Neck and Trochanter*

t-score	LS		FN		TR	
	Mean WHOQOL	p	Mean WHOQOL	p	Mean WHOQOL	p
≥ -2.5	60.22±9.08	0.02	58.61±8.64	0.06	58.93±8.72	0.005
< -2.5	54.64±10.43		54.63±13.54		52.68±13.34	

Table 5 - *Association between BMD in Lumbar Spine (LS), Femoral Neck (FN) and Trochanter (TR) with WHOQOL Scores in the Domain 1,2,3,4 and overall WHOQOL*

BMD	LS		FN		TR	
	r	p	r	p	r	p
WHOQOL Domain1	0.261	0.003	0.209	0.020	0.157	0.081
WHOQOL Domain 2	0.227	0.011	0.229	0.010	0.248	0.005
WHOQOL Domain 3	0.230	0.010	0.228	0.010	0.220	0.014
WHOQOL Domain 4	0.260	0.003	0.274	0.002	0.276	0.002
Overall WHOQOL	0.272	0.002	0.307	0.000	0.275	0.002

Table 6 - *Multivariate Regression Analysis of BMD Lumbar Spine, Femoral Neck and Trochanter with the Overall WHOQOL after Adjusting forBMI, Age and Fracture History*

Variable	B	p value	R2 (df); F
BMD LS	0.229	0.033	0.119 (4,120); 4.068
BMD FN	0.285	0.015	0.129 (4,120); 4.447
BMD TR	0.245	0.035	0.119 (4,120); 4.036

Relationship between BMD (g/cm2) in lumbar spine, femoral neck and trochanter with overall WHOQOL score and WHOQOL scores in different domains:

Table 5 shows the correlation between BMD in lumbar spine, femoral neck and trochanter with overall WHOQOL and WHOQOL scores in the domain 1, 2, 3 and 4 as depicted by the Pearson's correlation coefficient (r) and p-values. It clearly shows that there was a positive correlation of BMD in the lumbar spine, femoral neck and trochanter with overall WHOQOL and WHOQOL scores in all the 4 domains. The correlations were statistically significant (p-value < 0.05) except for the correlation between BMD in trochanter and the physical health domain.

As shown in Table 6, multivariate regression analysis of the overall WHOQOL score with the BMDs, after adjusting confounding factors like age, BMI and fracture history, showed statistically significant regression coefficients (p <0.05). BMD lumbar spine had a predictive value of 11.9% in contributing to the quality of life. Similarly, BMD femoral neck and trochanter had predictive values of 12.9% and 11.9% respectively.

Discussion:

In the study, WHOQOL (0-100) score of the WHOQOL-BREF questionnaire was taken as the score for reference. As per the scores, participants were grouped into poor score (< 50) and good score (≥ 50).

It was observed that most of the patients i.e. 97 patients (77.6%) had a score of ≥ 50 with a mean score of 57.68±10.07 in the overall WHOQOL score. Physical health domain was the most affected domain with a mean score of 48.04±13.53. Psychological health, social health and environmental health domains were less affected in the study population with mean scores of 58.50±10.02, 59.38±9.59 and 68.88±9.10 respectively. This finding was similar to the study done by Kotz *et al*[8], which was a longitudinal follow up study on a cohort of 1,171 aged women in an attempt to analyse the quality of life outcomes of osteoporosis. They observed that measures of physical health like greater risk for frailty (OR= 1.96), difficulty with frailty (OR= 2.77) and problems with ADL (OR= 3.37) were most affected.

Lai *et al*[9] studied the quality of life of 46 postmenopausal women with back pain and 42 postmenopausal women without back pain, using quality of life questionnaire of the European foundation for osteoporosis (QUALEFO). They observed that quality of life was significantly correlated with back pain (r range 0.50-0.90; p<0.0001). In our study also, it was found that the physical health was the most affected domain. Overall WHOQOL score was found to be better than the physical health score. It might be because of the effect of the other domains especially the social and environmental health domains, in contributing to the overall general well being of the individual.

On comparing the means of the WHOQOL scores in relation to the t-scores, it was observed that the mean WHOQOL score in all the domains as well as the overall WHOQOL score were higher in the osteopenic group in comparison to the osteoporotic group at all sites. This finding was similar to the study by Bruyere *et al*[10] in which they observed a better HRQOL (health related quality of life) scores in osteopenic group compared to the osteoporotic group. In the study by Berkemeyer *et al*[11] on 440 participants, they found that t-scores had a significant association with functional measures like activities of daily living (95.3 CI, 94.5 - 96.2; p < 0.05), instrumental activities of daily living (7.3 CI, 7.2 – 7.5; p < 0.05) and Timed Up and Go test (10.7 CI, 10.0 - 11.3; p < 0.05). The present study also found a significant association between overall WHOQOL score and t-scores in the lumbar spine and trochanter (p<0.05) though we did not find significant association of WHOQOL score with the t-score in femoral neck.

The study observed a significant association of the BMDs at lumbar spine, femoral neck and trochanter with the WHOQOL scores in all domains (r range 0.227 to 0.274; p <0.05) but we did not find any significant association between BMD trochanter and the WHOQOL score in the physical health domain (r = 0.157; p <0.081). We also observed a significant association of the BMDs at lumbar spine, femoral neck and trochanter with the overall WHOQOL score (r range 0.272 to 0.307; p <0.05). This finding was also in agreement with the study of Bruyere *et al*[10]. In a 3 years follow up study on a population of 1838 postmenopausal osteoporotic women to study the relationship of BMD and HRQOL, they observed a weak but significant positive association between lumbar BMD and HRQOL scores measured with QUALIOST.

Lindsey *et al*[12], also conducted a cross-sectional study on 116 healthy postmenopausal women who were not under any medication known to affect the bone including hormone replacement therapy. They observed that measures of physical performance like normal step length, brisk step length, normal gait speed, brisk gait speed, etc, were significantly correlated with BMDs at lumbar spine, femoral neck and trochanter (r range 0.19 to 0.38; p <0.05). In our study, we also observed that WHOQOL score in the physical health was significantly correlated with BMD at lumbar spine (r = 0.261; p<0.05) and femoral neck (r= 0.209; p<0.05).

The predictive value of BMDs at lumbar spine, femoral neck and trochanter in influencing or contributing to the quality of life were analysed using a multivariate regression after adjusting confounding factors like age, BMI and fracture history. The regressions were found to be statistically significant (R2 range 0.119 to 0.129; p <0.05). BMD lumbar spine had a 11.9% predictive value in contributing to the quality of life. Similarly, BMD femoral neck and trochanter had predictive values of 12.9% and 11.9% respectively. This was in contrast to the study by Bruyere *et al*[10], in which the multivariate regression after adjusting for age, BMI and prevalent fractures, showed no significant relationship of quality of life scores with the BMDs at both lumbar spine and femoral neck. This could probably be due to the reason that their study had excluded subjects with a history of fracture within three years and moreover all participants were prescribed a good calcium diet. In the present study, we had neither taken into consideration fracture duration nor the dietary calcium intake records.

The present study also showed similar observations with the study by Lindsey *et al*[12]. Multiple regression model

analysis, after adjusting for BMI, hours of total activity, total calcium intake and age of menarche, showed significant association of the physical performance measures and BMD at lumbar spine, femoral neck and trochanter (R2 range 0.11 to 0.24; $p < 0.05$). The findings were in agreement with the present study findings (R2 range 0.119 to 0.129; $p < 0.05$). The study indicates that the BMDs, with their positive associations with the WHOQOL scores, in fact have a part in predicting the quality of life of postmenopausal women.

There are some limitations which may be inherent to this study. One limitation was that this study had a small sample size (n=125). Another limitation of this study was of it being a cross-sectional study which was conducted on patients who had consulted a tertiary health institute and it might not have included the rural population at large and the results cannot be generalised to the whole ethnic population. There was also a problem regarding answering on certain questions especially on the social health issues viz, on personal relationships and sexual activity. As all the participants were postmenopausal women and most of them had some degree of reluctance in answering questions pertaining to personal / sexual relationship which may be due to personal or social reasons.

Conclusions:

From the present study it was observed that quality of life in postmenopausal women had a positive association with bone mineral density. The predictive values of the bone mineral density measured at the lumbar spine, femoral neck and trochanter in determining the quality of life were found to be significant. Management of osteoporosis mainly targets on symptomatic treatment, fracture risk prevention, minimisation of functional disabilities and improvement of the quality of life. Addressing the measures to improve the bone mineral density would improve the physical health in postmenopausal women and thereby may improve the quality of life in such population.

Properly designed population based studies which will represent the whole ethnic community would be desirable to confirm our findings. Further studies are required in this regard but nevertheless, this study has been successful in observing that though osteoporosis is a disease of low bone mineral density, there are also other important factors that might have influence on the quality of life of an individual.

References:

1. Graham P, Adler RA, Bonner FJ, Kasturi G. The prevention and treatment of osteoporosis. In: Frontera WR, Delisa AJ, Gans BM, Walsh NE, Robinson LR, editors. Delisa's Physical Medicine and Rehabilitation. Vol 1. 5th ed. Philadelphia: Lippincott Williams and Wilkins, 2010: 979-1014.
2. Fink FA, Ensrud KE, Nelson DB. Disability after clinical fracture in postemenopausal women with low bone density: the fracture intervention trial. *Osteoporos Int* 2003; **14:** 69-76.
3. Harper A, Orley J, Power M, Kuyken W, Sartorius N, Bullinger M, *et al.* WHOQOL-BREF- Introduction, administration, scoring and generic version of the assessment. 1996; [18 screens]. http://www.who.int/mental_health/media/en/76.pdf (accessed August 10, 2012).
4. Ryan PJ, Blake G, Herd R, Fogelman I. A clinical profile of back pain and disability in patients with spinal osteoporosis. *Bone* 1994; **15:** 27-30. http://www.sciencedirect.com/science/article/pii/8756328294908877(accessed August 18, 2012).
5. Lindsay R, Felica C. Osteoporosis. In : Longo DL, Fauci AS, Lasper DL, Hauser SL, Jameson JL, Loscalzo J, ediors. Harrison's Principles of Internal Medicine. Vol 2. 18th ed. New York: McGraw-Hill, 2011 : 3120-35.
6. Skvington SM, Lofty M, O'connell KA. The World Health Organisation's WHOQOL-BREF quality of life assessment: psychometric properties and results of international field trials - a report from the WHOQOL group. *Qual Life Res* 2004; **13:** 299-310.
7. Woolf AD, Akesson K, editors. An Atlas of Investigation and Management of Osteoporosis. New Delhi: Jaypee brothers, 2009.
8. Kotz K, Deleger S, Cohen R, Kamaigaki A, Kurata J. Osteoporosis and health related quality of life outcomes in Alameda county study population. *Prev Chronic Dis* 2004; **1:** 1-9.
9. Lai PSM, Chua SS, Chan SP, Low WY. Validation of the english version of QUALEFFO in Malaysia. *Int J Rheum Dis* 2008; **11:** 421-9.
10. Bruyere O, Cock CD, Deroisy R, Reginster JY. Bone mineral density and health related quality of life: a 3 year follow up study of osteoporotic women. *Open Geriatr Med J* 2009; **2:** 44-52.
11. Berkemeyer S, Schumacher J, Thiem U, Pientka L. Bone t-scores and functional status: a cross sectional study on German elderly. *PLoS One* 2009; **4:** 1-8.
12. Lindsey C, Brownbill RA, Bohannon RA, Ilich JZ. Association of physical performance measures with bone mineral density in postmenopausal women. *Arch Phys Med Rehabil* 2005; **86:** 1102-7.

PG Forum

REHAB CHALLENGE

A 5-year-old boy from poor socio-economic status presented to PMR OPD with difficulty in standing and inability to walk since 2 years of his age due to spastic diplegic cerebral palsy. On assessment in indoor there was significant untreated lower limb spasticity which made him almost bed bound (Fig 1). Initial response with exercise therapy and antispasticity medications was very poor. That's why phenol chemoneurolysis was done on bilateral post tibial nerve. Subsequently diagnostic followed by therapeutic motor point block of bilateral hamstring muscles were done.

At that stage patient was able for independent supported standing with bilateral ankle foot orthosis though mild bilateral knee flexion deformity was persisting (Fig 2). That's why a gaiter was planned for standing balance and supported walking (Fig 3). Now the patient is keen for community mobility and to take part in outdoor games.

Please opine regarding further rehabilitative management plans for this young boy for further mobility and independence.

Fig 1

Fig 2

Fig 3

Medical Philately

2007 Obligatory Tax Red Cross Week. Generations

Country	Slovenia
Date	2007
Disability	Elderly || Stick
Meta	philately, stamps, Digital Disability, theme, imagery of disability on postage stamps, Outside Centre, 2007, Obligatory Tax, Red Cross Week, elderly people, children, stick
Number	SG760
Theme	Charity || Red Cross

PG Forum

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Country	Slovenia
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Disability	Elderly || Stick
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Number	SG760
Theme	Charity || Red Cross

PG Forum

REHAB QUIZ

1. O'Brien's test detects all except

A) Acromioclavicular joint sprain
B) Rotator cuff impingement
C) SLAP lesion
D) None of these

2. Clean intermittent catheterisation was first proposed by

A) Guttmann & Frankel
B) Crede et al
C) Lapides et al
D) Peritz et al

3. 'Charlie Chaplin' gait seen in

A. Hip abductor weakness
B. Tibial torsion
C. Hip adductor weakness
D. Pes cavus

4. In 2015 ACR/EULAR Gout classification criteria, if sufficient criterion not met, serum urate value <4mg/dl denotes a point of

A. 0
B. -2
C. 2
D. -4

5. Sensitivity and specificity of MRI in confirming arthrography-proven rotator cuff tear

A. 96 & 82
B. 88 & 92
C. 100 &88
D. 92 &100

6. Jaipur foot weighs

A. 400g B. 800g
C. 1000g D. 2000g

7. Beneficiaries under 1995 PWD act are all except

A. Blind
B. Multiple disability
C. Mental retardation
D. Leprosy-cured

8. According to ACR 2012, orthoses for medial tibiofemoral OA

A. Lateral heel wedge
B. Lateral heel & sole wedge
C. Lateral wedge subtalar strapped insole
D. Varus correcting orthoses

9. According to the Caryle formula, in bilateral transradial amputee

A. the distance from the apex of the lateral epicondyle to thumb tip (forearm) is the patient's body height X 0.21
B. the distance from the apex of the lateral epicondyle to thumb tip (forearm) is the patient's body height X 0.19
C. the distance from the apex of the medial epicondyle to thumb tip (forearm) is the patient's body height X 0.21
D. the distance from the apex of the medial epicondyle to thumb tip (forearm) is the patient's body height X 0.19

10. In pulmonary rehabilitation exercise should be discontinued for O2 saturation below

A. 85%
B. 90%
C. 92%
D. 88%

ANSWERS

Answer keys to June 2015

1C, 2A, 3C, 4B, 5A, 6D, 7A, 8D, 9A, 10D

Case Report

Syndrome of Inappropriate Antidiuretic Hormone (SIADH) in Traumatic Spinal Cord Injury

Chethan. C[1], Vinay Goyal[2], Diganta Borah[3], Rajendra Sharma[4], Nonica Laisram[5]

Abstract

Hyponatraemia is a known complication associated with neurosurgical conditions including acute spinal injury. The prevalence of hyponatraemia in acute spinal cord injury has been known to be much higher than in the general population. Hyponatraemia is a marker of different underlying diseases and it can be a cause of morbidity itself; this implies the importance of a correct approach to the problem. The syndrome of inappropriate antidiuretic hormone (SIADH) is one of the most common causes of hyponatraemia; it is a disorder of sodium and water balance characterised by urinary dilution impairment and euvolaemic/hypotonic hyponatraemia, in the absence of renal disease or any identifiable non-osmotic stimulus able to induce antidiuretic hormone (ADH) release. It is a diagnosis of exclusion. We are reporting a case of hyponatraemia in a patient with lumbar spinal cord injury who was initially managed as any other hyponatraemia and was later diagnosed as suffering from SIADH.

Key words: **SIADH, Hyponatraemia, spinal cord injury.**

Introduction:

Hyponatraemia is the most common electrolyte disorder encountered in clinical medicine[1]. The prevalence of hyponatremia in spinal cord injury has been known to be much higher than in the general medical or surgical patient population. The prevalence of hyponatraemia in the neurosurgical population has been reported as high as 50%[2]. Uncontrolled hyponatraemia may lead to lethargy, seizures, coma, cardiac arrhythmia and death. Therefore, the complication of hyponatremia should be paid attention after the spinal cord injury. Balance of sodium in blood is complicated and is influenced by many factors. Autonomic nervous system, neuroendocrine dysfunction and haemodynamic changes after spinal cord injury (SCI) play a key role in occurrence of electrolytes abnormality[3].

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Case Report:

In October 2011, a 25-year-old married female suffered SCI following an accidental fall from roof. She temporally lost consciousness and was immediately shifted to a tertiary care trauma centre, where she was diagnosed to have fracture L1 with traumatic SCI, paraplegia and neurogenic bowel and bladder. She was managed surgically with pedicle screw fixation and was discharged subsequently. On 13/02/2012 she presented to PMR OPD of Safdarjang Hospital with complaints of weakness and loss of sensation in lower limbs, bowel and bladder incontinence and pressure ulcers on both trochanteric and sacral region. Patient was subsequently admitted to rehab ward for comprehensive management.

On examination her cardiorespiratory (PR- 94 beats/minute regular in rhythm, BP - 106/60 mm Hg, RR - 12 breaths/minute, regular) and gastro-intestinal system showed no abnormality. Pallor was present. There were grade IV pressure ulcers on bilateral trochanteric and sacral region; measuring 10 X 10 cm on right, 7 X 5 cm on left trochanter and 18.5 X 9 cm on sacrum. On neurological examination her higher mental functions and cranial nerves were normal, she had complete SCI with AIS – A. Motor, sensory and neurological levels were D11.

Management :

Baseline investigations showed Hb to be 10.8g/dl and serum sodium - 141meq/l. SCI rehabilitation consisting

of two hourly position changes, ROM exercises of bilateral lower limbs and strengthening exercises of bilateral upper limbs, gradual training in tilt table along with daily pressure ulcer dressing was started. Culture and sensitivity of sacral pressure ulcer showed pseudomonas, sensitive to ciprofloxacin, hence she was started on IV ciprofloxacin in view of long standing ulcer with copious discharge, inflanned and indurated surrounding skin.

On twelfth day she developed nausea, vomiting (4 episodes, non-projectile, non-bile stained containing undigested food particles) and weakness.Injection ondansetron 4mg and 75mg injection ranitidine were administered. Her pulse rate was 98 beats/ minute regular in rhythm, BP - 96/60 mm Hg. Vomiting stopped but nausea and weakness persisted, hence she was started on IV fluids with 0.9% saline and Ringer lactate (1500 ml IV fluids) and 800 ml of oral fluids. Next morning her serum electrolytes, KFT and LFT were obtained, her serum sodium level was decreased to 122meq/l (Table1). She was treated with isotonic fluids (2500ml of IV fluids and 1000ml oral intake) and oral sodium chloride in the form of table salt. Along with persistent nausea and weakness she developed headache and dizziness, serum electrolytes showed her serum sodium level had dropped further to 114meq/l along with decrease in serum potassium level to 3.2meq/l. ECG and chest x-ray was normal.

Since there were no signs of dehydration or fluid overload a diagnosis of euvolaemic hyponatraemia was made. The commonest cause of euvolaemic hyponatraemia is SIADH, hence blood and urine analysis were carried out for the same which revealed serum hypoosmolality (240m osnn ol/kg), urine hyperosmolality (510m osnn ol/kg), increased urine sodium excretion (46meq/L) and decreased blood urea nitrogen 8mg/dl (Table 2). Based on her clinical findings and investigation reports she was diagnosed to have SIADH as per Bartter and Schwartz criteria[4] (Table 3). She was treated accordingly with fluid restriction 1000ml/day, 3% saline infusion - 2 ml/kg body weight per hour, (0.5mmol/l/hour), increased oral salt intake and intravenous 40mg injection furosemide. Repeat serum electrolytes were done after 12 hours, her serum sodium level had improved to 120meq/l and potassium was 4.1meq/l and her symptoms improved. Further on day 16 her serum sodium increased to 131meq/l and potassium was 4.8meq/l (Table 1) and symptoms disappeared.

Discussion:

Hyponatraemia is frequently found in both acute and chronic SCI. The aetiology of hyponatraemia in SCI patients is multifactorial and includes not only general factors such as the use of diuretics and the intravenous infusion of hypotonic fluids, but also certain mechanisms which operate in the spinal cord injured: decreased renal water excretion due to both intrarenal and arginine vasopressin dependent mechanisms (resetting of the osmostat), coupled with habitually increased fluid intake, and the ingestion of a low salt diet[5]. Frisbie[6] reported that higher levels of SCI correlate with reduced sodium conservation, hypotension, and hyponatraemia. While Peruzzi *at el*[7] suggested that the most significant predictor of hyponatraemia is Frankel class A (loss of sensory and motor function below the level of injury) to E (no motor or sensory deficits associated with spine fracture), defined by the neurological classification of SCI, rather than the level of SCI.

Table 1: *Blood Investigation Values on Daily Basis*

Baseline	Day - 13	Day - 14	Day - 15	Day - 16
Hb: 10.8g/dl	T. BILI: 0.2	T. BILI: 0.2	T. BILI: 0.2	T. BILI: 0.2
TLC: 6500	S. ALP: 302	S. ALP: 288	S. ALP: 288	S. ALP: 270
DC: P66, L30,M2,E2	SGPT: 14	SGPT: 14	SGPT: 14	SGPT: 16
ESR: 25mm/hour	SGOT: 45	SGOT: 42	SGOT: 42	SGOT: 48
Creatinine: 0.3mg/dl	Creatinine: 0.6 mg/dl	Creatinine: 0.5 mg/dl	Creatinine: 0.5 mg/dl	Creatinine: 0.4 mg/dl
Blood urea: 12mg/dl	Blood urea: 12 mg/dl	Blood urea: 14 mg/dl	Blood urea: 16 mg/dl	Blood urea: 14 mg/dl
Sodium: 141 meq/l	Sodium: 122 meq/l	Sodium: 114 meq/l	Sodium: 120 meq/l	Sodium: 131 meq/l
Potassium: 4.9meq/l	Potassium: 3.8 meq/l	Potassium: 3.2 meq/l	Potassium: 4.1meq/l	Potassium: 4.8 meq/l
RBS: 121mg/dl	RBS: 108mg/dl	RBS: 102mg/dl	RBS: 114mg/dl	RBS: 120mg/dl
Total protein: 5.9g/dl			Total protein: 6.1g/dl	
Albumin: 3.2g/dl			Albumin: 3.2g/dl	
Globulin: 2.7g/dl			Globulin: 2.9g/dl	
CRP - 2 mg/l	CRP - 6 mg/l			

Table 2 - *Blood and Urine Parameters of Patient on 14th Day*

Lab test	Blood / Urine parameter	
	PATIENT	SIADH
Serum sodium	114meq/l	<135meq/l
Serum osmolality	240mosmol/kg	<275mosmol/kg
Urine sodium	46meq	> 20meq/l
Urine osmolality	510mosmol/kg	>100 mosmol/kg
BUN	8mg/dl	<10mg/dl

BUN - Blood urea nitrogen

Hyponatraemia is classified into three types - hypovolaemic hyponatraemia, hypervolaemic hyponatraemia and euvolaemic hyponatraemia. Our patient had euvolaemic hyponatraemia. Commonest cause of euvolaemic hyponatraemia being SIADH and other causes are excessive drinking, renal failure, hypothyroidism, extreme exercising during which over hydration has occurred, low solute intake[8].

The syndrome of inappropriate antidiuresis (SIADH) is one of the most common causes of hyponatraemia. It is a disorder of sodium and water balance characterised by urinary dilution impairment and hyponatraemia, in the absence of renal disease or any identifiable non-osmotic stimulus capable of inducing antidiuretic hormone (ADH) release. According to its definition, it is diagnosed through an exclusion algorithm. The pathophysiological basis of SIADH is an absolute increase in body water. This increase depends on an excessive water intake that overwhelms the restricted renal ability of diluting urine and mounting compensatory diuresis due to ADH dysregulation[9]. In our case we excluded other causes of euvolaemic hyponatraemia and narrowed on to SIADH as per Bartter and Schwartz criteria and managed appropriately, we attribute multiple factors (antibiotics, pressure ulcers, stress, long term immobilisation and hospitalisation) interacted in initiation of SIADH and increased fluid administration as possible cause of worsening of hyponatraemia. Patient's sodium levels were improved after tapering infusion volume and water intake restriction in this case. Treatment of the case is the same as that for other causes of SIADH, which have suggested that the reduced extracellular volume is important to improve the hyponatraemia after traumatic SCI.

Conclusions:

Hyponatraemia is common in traumatic SCI, especially in cervical injuries. As physiatrists, we deal with SCI patients in our daily practice this situation can be encountered and one should be vigilant in presence of symptoms described above. SIADH is a frequent cause of hyponatraemia; this condition can be misdiagnosed and mismanaged. Early diagnosis and prompt management of SIADH is of paramount importance as it can be fatal.

Table 3- *Bartter and Schwartz criteria for SIADH:*

1.	Decreased plasma osmolality (<275 mosmol/kg)
2.	Inappropriately concentrated urine (>100 mosmol/kg)
3.	Euvolaemic
4.	Elevated urine Na (>20 meq/l)
5.	Euthyroid, eucortisolemic and no diuretic use.
6.	Chest x-ray and in selected cases, computed tomography (CT) scan of head may be appropriate to reveal an underlying cause.
Supplemental features	
Uric acid < 4 mg/dl	
BUN < 10 mg/dl	
Failure to correct hyponatraemia after NS infusion	
Correction of hyponatraemia after fluid restriction	

References:

1. Boscoe A, Paramore C, Verbalis JG. Cost of illness of hyponatremia in the United States. *Cost Eff Resour Alloc* 2006: 4 - 10.
2. Rahman M, Friedman WA. Hyponatremia in neurosurgical patients: clinical guidelines development. *Neurosurgery* 2009; **65:**925–36.
3. Kageyama K, Suda T. A case of hyponatremia after cervical spinal cord injury. *Endocrine* 2011; **58**: 369-72.
4. Schwartz WB, Bennett W, Curelop S, Bartter FC. A syndrome of renal sodium loss and hyponatremia probably resulting from inappropriate secretion of antidiuretic hormone. *Am J Med* 1957; **23:**529–42.
5. Soni BM, Vaidyanthan S, Watt JWH, Krishnan KR. A retrospective study of hyponatremia in tetraplegic/paraplegic patients with a review of the literature. *Paraplegia* 1994; **32:**597-607.
6. Frisbie JH. Salt wasting, hypotension, polydipsia, and hyponatremia and the level of spinal cord injury. *Spinal Cord* 2007; **45:**563-8.
7. Peruzzi WT, Shapiro BA, Meyer PR Jr, Krumlovsky F, Seo BW. Hyponatremia in acute spinal cord injury. *Crit Care Med* 1994; **22:**252-8.
8. Haralampos JM, George LL, Moses S E. The hyponatremic patient, a systematic approach to laboratory diagnosis. *CMAJ* 2002; **166:**1056-62.
9. Alan GR, Joseph GV. Chapter 10 - Posterior Pituitary. William's Textbook of Endocrinology. 12th ed. Philadelphia: Elsevier, 2011:291 - 315.

Case Report

Acquired Flat Foot due to Tibialis Posterior Rupture, a Known Yet Missed Cause

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Abstract

Rupture of tibialis posterior tendon is a known cause of acquired flat foot but the diagnosis is missed or delayed in most of the cases. It may lead to significant morbidity. We present a case of 13 years old boy with history of blunt trauma to his left foot one year back and presented with pain medial aspect of right foot and difficulty in prolonged walking and running. Clinically he had a flat and hyperpronated foot. His x-rays were normal and MRI revealed partial tear of posterior tibial tendon. He was recommended medial arch support, shoe modification, NSAIDs and referred to orthopedic surgeon for repair. Posterior tibialis tendon dysfunction is one of the concealed injuries that require earliest diagnosis and immediate attention, failing which the outcomes can have debilitating effects on patient's quality of life. This is important to prevent foot deformities and long term disability.

Key words: **Acquired flat foot, tibialis posterior, diagnosis.**

Introduction :

Acquired flat foot is a rare condition which can be due to neuropathic, degenerative and traumatic causes[1]. Rupture of tibialis posterior is a known cause of acquired flat foot but the diagnosis is delayed in most of the cases[2]. The likelyhood of tibialis posterior involvement as a potential cause of flat foot deformity is less emphasised in clinical practice, even though it has an established association with foot trauma. Missed or delayed diagnosis leads to significant morbidity especially in active individuals due to chronic pain[3]. Late complications include a rigid deformity with forefoot abduction and valgus heel. Rupture of tibialis posterior muscle is diagnosed on clinical evaluation and can be confirmed by ultrasound, bone scintiogrphy and MRI[4]. Conservative treatment includes, rest, ice, immobilisation and orthosis[5].For refractory or advance cases, surgical options may be considered, which include tendon debridement, tendon transfer, corrective osteotomy and orthodesis[6].

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Case Report :

A 13-year-old male presented to our clinic with complaint of pain on medial aspect of left foot and sole. His pain started one year ago after sustaining a blunt trauma to the undersurface of right foot while he was running barefooted. He described it as a sharp pain of acute onset. He could not recall any ankle twisting but had temporary ankle swelling for which he did not seek any medical advice initially. His swelling subsided gradually while a dull pain on the medial border and sole of the foot persisted. His symptoms were aggravated with standing, walking and running which was limiting his mobility and sporting activities. He remained under follow-up with general physician and orthopaedician, and had multiple x-rays of his foot. His condition did not improve with use of topical analgesics and short courses of NSAIDs. His symptoms progressively got worse and were later referred to rehabilitation medicine for further management. There was no history of any metabolic or inflammatory disorder. On examination he had pesplanovalgus deformity on left side with flattening of medial longitudinal arch while the right foot was normal. There was hyperpronation and

abduction of left forefoot (Fig 1). Localised tenderness was present over the navicular bone area. Medial arch was lower on the affected side and was unable to perform left heel raise. Active range of motion of ankle and foot were normal. He had an antalgic gait pattern. X rays of the left foot did not reveal any bony abnormality. His baseline lab investigations including blood complete picture, erythrocyte sedimentation rate, C-reactive protein, serum uric acid, blood sugar random and rheumatoid factor were within normal limits. MRI of the left foot revealed a partial tear to the tibialis posterior tendon at its insertion into the navicular bone (Fig 2). A diagnosis of acquired flat foot secondary to partial tear of tibialis posterior was established. The patient was recommended orthotic medial arch support, shoe modification and physiotherapy and was referred to orthopaedic surgeon for tendon repair.

Discussion:

Acquired flat foot has a number of causes which include degenerative changes secondary to osteo-arthritis, inflammatory arthritis and fractures of the foot and ankle[1]. Neuropathic conditions include diabetes mellitus, severe peripheral neuropathies and leprosy while acquired causes include tear of the spring ligament, tibialis posterior rupture and rarely tear of tibialis anterior[7]. Patients with acquired flat foot secondary to a generalised medical condition are diagnosed early as compared to those patients developing it after a traumatic event. There can be a delay of months to years before a correct diagnosis is reached as most patients presenting with posterior tibialis tendon dysfunction are misdiagnosed as ankle sprain or arthritis[8].

Tibialis posterior is plantar flexor and invertor of the foot. It is a dynamic stabiliser of medial longitudinal arch of the foot. It lies posterior to the medial malleolus and inserts into the navicular tuberosity and plantar aspect of tarsal bones. The tendon is at greatest stress immediately after heel strike when foot moves into increased inversion[9]. Foot inversion after heel strike predisposes the tendon to greatest stress[9]. Tibialis posterior rupture is a documented cause of acquired flat foot deformity but it is often missed on clinical examination leading to a delay in diagnosis. Patients are insufficiently treated and are likely to have further complications. In a case series of 17 patients with posterior tibial tendon rupture, the average time to treatment was found to be 43 months[10]. In all but two of the patients, incorrect diagnosis had been made on initial assessment. In almost all patients who sustain a rupture of the tibialis posterior tendon, there is a history of ankle trauma[1].Typically the mechanism of injury is forced foot eversion. Younger patients and athletes, tend to sustain a traumatic avulsion of the tendon at its insertion into the navicular, while middle aged or elderly individuals often have inflammatory or degenerative type tear.

Its prevalence increases with age and is found to be up to 10% among elderly. Diabetes mellitus, seronegative arthritis, hypertension, steroid injection around the joint are associated risk factors of tibialis posterior rupture[11]. The tibialis posterior dysfunction progression has been classified in four stages which facilitates its management[12]. Stage I ; is tendon inflammation, stage II; the tendon is elongated and there is acquired flat foot deformity, stage III ; fixed foot deformity with degenerative changes at subtalar joints and stage IV; additional degenerative changes at ankle joints.

The diagnosis is mainly clinical. It usually presents with tenderness and swelling at the medial aspect of the foot with difficulty in inversion of foot and inability to stand on forefoot. The arch gradually collapses, the heel goes into valgus and the forefoot is abducted leading to ‘too many toes’ sign[13]. Radiographs are recommended to rule out other causes and to stage the rupture depending on the degenerative changes in the ankle and subtalar joints. Conservative treatment is focused on rest, icing, immobilisation, orthotic supports, NSAIDs and modified shoeware[14].

The surgical management includes tendon debridement, tendon transfer and osteotomy for stage I, tendon transfer and reconstruction for stage II and subtalar and pan talar orthodesis for stage III and stage IV respectively[6]. Closed fractures of ankle have been associated with posterior tibialis tendon ruptures. Hence soft-tissue injuries must be considered in the management of unusual or complex fractures around ankle.[15]Though it remains one of the commonest ruptured tendon in adult foot injuries, non-specific symptoms of foot pain and complexity of foot dynamics may distract the physicians to consider ligamentous and joint injuries as their initial diagnoses. Initial x-rays are usually focused on fractures and dislocations and low arches can be overlooked as an obscure sign of an underlying soft tissue injury. MRI may appear to be an aggressive modality in the initial phases of investigating a painful foot. Hence it is usually reserved for refractory cases as it is expensive and not readily available, especially in developing health systems.

Conclusions:

Posterior tibialis tendon dysfunction is one of the concealed injuries that requires earliest diagnosis and immediate attention, failing which, the outcomes can have debilitating effects on patient's quality of life. Clinical and radiological evidence of flat foot post -trauma, should raise a high suspicion of posterior tibialis tendon rupture as it occurs more frequently than anticipated, and has tremendous therapeutic implications. This is important to prevent foot deformities and long term disability.

Fig1: *Hyperpronation of Left Foot*

Fig 2: *MRI Left Foot Showing Partial tear of Tibialis Posterior*

References

1. Mendicino SS. Posterior tibial tendon dysfunction. Diagnosis, evaluation and treatment. *Clin Podiatr Med Surg* 2000;**17:**33–54.
2. Kohls-Gatzoulis JA, Singh D. Tibialis posterior dysfunction as a cause of flatfeet in elderly patients. *Foot* 2004;**14:**207-9.
3. Mizel MS, Temple HT, Scranton PE II, Gellman RE, Hecht PJ, Horton GA, *et al*. Role of the peroneal tendons in the production of the deformed foot with posterior tibial tendon deficiency. *Foot Ankle Int* 1999;**20:**285-9.
4. Hogan J. Posterior tibial tendon dysfunction and MRI. *J Foot Ankle Surg*1993;**32:**467–72.
5. Chao W, Wapner KL, Lee TH, Adams J, Hecht PJ. Non-operative treatment of posterior tibial tendon dysfunction. *Foot Ankle Int*1996;**17:**736-41.
6. Wacker JT, Hennessy MS, Saxby TS. Calcaneal osteotomy and transfer of the tendon of flexor digitorum longus for stage-II dysfunction of tibialis posterior. Three- to five-year results. *J Bone Joint SurgBr* 2002;**84:**54-8.
7. Basmajian JV, Stecko G. The role of muscles in arch support of the foot. *J Bone Joint. Surg Am*1963;**45:**1184–90.
8. Mosier SM, Pomeroy G, Manoli A II. Pathoanatomy and aetiology of posterior tibial tendon dysfunction. *Clin Orthop Relat Res* 1999;**365:**12-22.
9. Pomeroy GC, Pike RH, Beals TC, Manoli A II. Current concepts review. Acquired flatfoot in adults due to dysfunction of the posterior tibial tendon. *J Bone Joint SurgAm* 1999;**81:**1173-82.
10. Mann FA, Thompson FM. Rupture of the posterior tibial tendon causing flatfoot. *J Bone Joint SurgAm*1985;**67:**556–61.
11. Myerson M, Solomon G, Shereff M. Posterior tibial tendon dysfunction: its association with seronegative disease. *Foot Ankle* 1989;**9:**219-25.
12. Johnson KA, Strom DE. Tibialis posterior tendon dysfuncion. *Clin Orthop*1989;**239:**196–206.
13. Johnson KA. Tibialis posterior tendon rupture. *Clin Orthop* 1983;**177:**140-7.
14. Van Wellen PA, De Boeck H, Opdecam P. Non-traumatic dislocation of tibialis posterior tendon in a child. A case report. *Arch Orthop Trauma Surg* 1993;**112:**243-4.
15. Kelbel M, Jardon OM .Rupture of Tibialis Posterior Tendon in a Closed Ankle Fracture. *J Trauma* 1982; **22:**1026-7.

Case Report

Ultrasound Guided Trigger Point Injections in Myofascial Pain Syndrome

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Abstract

Trigger points as a cause of musculoskeletal or myofascial pain syndrome is well documented. Trigger points (Tr Ps) are tender and hypersensitive nodules seen in skeletal muscles which develop as a result of sudden or repetitive trauma to the muscles. They cause contractile state of a muscle with local or radiating pain. Active trigger points cause intense pain with limitation of movements of the muscles. The treatment involves deactivating the trigger points, usually done by various methods. Most common practice is myotherapy which involves deep tissue massage which is painful and time consuming. Dry needling and needling with anaesthetic injaection have been successfully used by many. Recently, ultrasound guidance is used to locate the trigger points and to accurately place the needle in to them to deactivate, thus preventing complications of blind procedures.

Key words: **Ultrasound, trigger point, needling, myofascial pain.**

Introduction:

Myofascial pain syndromes are known to be secondary to active trigger points (Tr Ps) in skeletal muscles. Most common form of deactivation Tr Ps has been deep tissue massage, which is painful and time consuming. Tr P dry needling or needling with local anaesthetic injection is being used as a successful alternative. Blind needling has few complications, like wrongly placed needles, haematoma and pneumothorax.

High frequency transducers have helped in successful localisation of trigger points. Present case report is to identify, prove and characterise trigger points in ultrasound and pave the way for understanding them and utilise the knowledge for pain relief.

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Case Report:

A 44-year-old man presented with generalised neck and shoulder pain for more than two years. The patient has had degenerative changes in the spine with no obvious disc herniation or nerve root compression. Patient was diagnosed to have repetitive stress injury (RSI) from his work. Musculoskeletal and sports medicine specialist had found multiple trigger points and had advised trigger point therapy (deep tissue massage or myotherapy). He had undergone regular myotherapy and had intermittent relief with recurrence. Finally ultrasound guided needling was suggested.

Patient was taken up for ultrasound guided trigger point injection. Patient was examined and individual muscles were palpated to locate trigger points eliciting tenderness, which are known to occur in specific locations in each muscle.Trigger points are extremely painful on pressure. Ultrasound is used to locate the point over the palpated nodule. The skin mark was done from a needle cap. After sterile preparation, the nodule was palpated and held between two fingers and needle was inserted with ultrasound guidance. Colour Doppler was used to rule out any vascular interposition. Needle was progressed till there was twitch response and patient felt pain. Patient's feedback in terms of altered pain sensation and referral pain confirmed accurate placement of the needle (Fig 1). Trigger point had unusual characteristic feel while being entered. Needle was seen to glide on the point and slip. While being pierced, there was a

feeling of giving in after little pressure, which felt like it had a well formed capsule. Once confirmed, needle was used to mechanically disrupt the nodule in multiple points. Ultrasound monitoring was also used to avoid further unwanted penetration of the needle into deeper structure (Philips HD 11 XE Ultrasound system, Linear probe 12 – 3 MHz, Needle.60 X 25mm 23 G).

Three types of trigger points were noticed in this report. The first one was an ill defined hyperechoic nodule, which was most elusive (Fig 2). The second type was a well defined hypoechoic nodule with a thin hyperechoic wall (Fig 3). Third was well-defined hypoechoic to anechoic nodule with a thin hyperechoic wall and subtle acoustic enhancement. It also showed a tiny central hyper echoic ring or linear dot (Fig 4). However we feel further studies are needed to confirm our findings. The smallest Tr P we could locate on ultrasound was 1.6 mm and the largest was 4mm.

At the end of the procedure 1ml of lignocaine was injected and needle was withdrawn. Soon after the procedure, there was immediate relief of pain, with release of stiffness in that particular muscle and improved range of movements.

Patient was asked to stretch that particular muscle after the procedure regularly. Other muscles were treated in the same way. Post procedure no complications were seen.

Discussion :

Trigger points as the cause of musculoskeletal and myofascial pain syndrome have been well established by Trevell and Simons [1]. Trigger points form as a result of acute or chronic trauma to skeletal muscles and are known to form in specific locations in all the muscles [1]. Cause of formation of Tr P is still unknown. Biochemistry of the contents studied by Shah *et al* [2] have showed pain causing chemicals in the Tr Ps. Active and latent form of Tr Ps are described, where active Tr Ps cause local pain and taut bands in a muscle with shortening. Active Tr Ps are painful on compression and cause local and radiating pain.

The treatment options involve deactivating the active Tr Ps. Deactivation of Tr Ps are done by various methods which involve stretch and spray, deep tissue massage, soft tissue mobilisation, dry needling and injection therapy among others. Trigger point injection has been described as the most effective treatment by Alvarez *et al*[3].

We adopted dry needling [4, 5], injection therapy [6] and a combination of both methods for deactivating TrPs. Dry needling involves insertion of needle into the Tr P and puncture in multiple locations to mechanically disrupt the point [3]. Injection therapy involves injection of a local anesthaetic in to the Tr P to get an effective pain relief.

Blind needling has few complications, like accidental syncope, skin infection, haematoma and pneumothorax[7].

Trigger point injections have been proven to be effective in deactivating them, thereby reliving the pain. Ultrasound guidance [8,9] has been used recently to locate them for advancing the needles. Accurate placement and guided puncture of the points have helped in eliminating complications of blind needling. We are of the opinion that prior knowledge of the trigger points in muscles and palpation are important for localising them on ultrasound. Further studies are needed with musculoskeletal higher frequency transducers to confirm our findings.

Though blind techniques are quite safe and using expensive ultrasound machine may not be feasible in all the cases. Further studies are needed to identify quick and easily for the inaccessible Tr Ps.

Post procedure follow-up had shown the trigger point to be smaller in size with heterogeneous echotexture break in the wall of the trigger point, in the initial days. In the long duration trigger point sites did not show any problems. However looking for the passive trigger point would be difficult without tenderness.

Finally we would like to confirm that the study has helped in proving, identifying and characterising the Tr P in the myofascial pain syndrome. Further studies with high definition imaging probes may be needed to study the Tr Ps and ultrasound does not hold superior all the time over the blind dry needling, as they are cheaper and less time consuming in the general therapy other than the inaccessible areas.

Conclusions:

Ultrasound guided needling and injections have been used recently in treating trigger points. This has helped in accurate placement of needles with real time visualisation of the procedure. Complications involved in blind procedures have been eliminated. Significant pain relief is seen after the procedure without post procedure complications. Prior knowledge of the trigger points in specific muscles is of utmost importance in locating them on ultrasound.The use of ultrasound guided localisation definitely reduces the complications

Fig 1: *Needle Tip*

Fig 2: *Hyperechoic Nodule*

Fig 3: *Hypoechoic Nodule*

Fig 4: *Central Dot*

associated with needling and possibly enhances the efficacy of dry needling. Ultrasound guided needling helps in cutting short the time in deactivation of the trigger point than manual therapies in inaccessible areas. Manual therapies are cheaper and less time consuming for the general therapy.

Further and larger studies are needed to help in imaging the trigger points on ultrasound.

References :

1. Tavell JG, Simons DG. Myofascial pain and dysfunction: The Trigger Point Manual. Philadelphia PA: Lippincott Williams & Wilkins,1983.
2. Shah P, Elizabeth A,Gilliams BA. Uncovering the biochemical milieu of myofascialtrigger points using in vivo microdialysis: an application of muscle pain concepts tomyofascial pain syndrome. *Bodywork and Movement Therapies* 2008; **12:** 371–84.
3. Alvarez DJ, Pamela G, Rockwell, University of Michigan Medical Scholl, Ann Arbor, Michigan. *Am Fam Physician* 2002; **65:** 653-1.
4. Bubnov RV. The use of trigger point "dry" needling under ultrasound guidance for the treatment of myofascial pain (technological innovation and literature review). *Liksparva* 2010 ; **6:** 56-64.
5. *Lewit K:* The needle effect in the relief of myofascial pain. *Pain* 1979; **6:**83-90.
6. Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point. The importance of the local twitch response. *Am J Phys Med Rehabil* 1994;**73:**256–63.
7. Kenneth P, Sharma K, Botwin: Ultrasound-guided trigger point injections in the cervicothoracic musculature: a new and unreported technique. *Pain Physician* 2008; **6:**885-9.
8. Niraj G, Collet BJ, Bone M. Ultrasound-guided trigger point injection: first description of changes visible on ultrasound scanning in the muscle containing the trigger point. *Br J Anaesth* 2011; **107** : 474-5.