Serum Lipid Changes after Short Term SIPC Therapy for Lower Limb Lymphedema

Mostafa Mehrabi Bahar · Mohammad-Hadi Saeed Modaghegh · Ehsan Soltani

Abstract Lymphedema is a ubiquitous chronic disease with various suggested treatment options, but no definite treatment. Using (Sequential) Intermittent Pneumatic Compression (SIPC) is one of the most appropriate non-surgical treatments without any noticeable complications. In this study, we evaluated the serum lipids changes following SIPC. Participants included 40 lower limb lymphedema patients who underwent High Pressure SIPC for a period of 48 hrs. Pre and Post SIPC serum lipids changes were evaluated. Though, there was some increase in the serum level of cholesterol and triglyceride, none of the patients had the values above the normal range. We concluded that, the fluid entering the serum during SIPC, contain large molecules such as lipids, which increases serum lipid levels. However this phenomenon does not have any significant complication for the patients.

Keywords Lymphedema · Postprandial hyperlipidemia · Intermittent pneumatic compression complication

Introduction

Lymphedema is regarded as chronic accumulation of lymph in the soft tissues.

Over 19 million people are involved with the ubiquitous disease [1]. Among secondary subtype which is more common than the primary ones, surgical lymph node dissection in industrial countries and post infectious lymphedema in developing countries are the most common causes [2]. Various complications such as skin dryness and thinning, abnormal and deep folds of the skin, soft tissue recurrent infections and malignancies were identified for the patients, especially in prolonged disease.

Despite various suggested treatment options, there is no definite medical or surgical treatment for the disease.

The Sequential Intermittent Pneumatic Compression (SIPC) is one of the most common and safest modality of medical treatment for driving out lymph from the extremity.

Although, no significant complications have yet been identified for this method [1, 3–5], there is some concern about the hemodynamic and biochemical changes, as well as soft tissue damage after SIPC usage. Previous studies on hemodynamic changes indicated that in patients with congestive heart failure or renal problems, there is the possibility of pulmonary edema following SIPC therapy due to resulting volume over load. Therefore, in these patients it is necessary to use lower pressures for a short period of time [1]. Also, study of the modality on muscle tissue damage did not show any significant increase in muscle enzyme levels [3].

Since lymph fluid contains high concentration of lipids, entrance of high quantity of lymph to the serum during SIPC therapy can be theoretically considered as a complication of the modality. However, as far as we know, no particular study has yet been performed on the matter. In this study we evaluated serum lipid changes following SIPC in lymphedema patients.

Methods

The study was conducted after ethics committee approval (Mashhad University of Medical Sciences, Iran) from
January 2004 to December 2005. Sample size included 40 sequentially selected lymphedema patients with the age range of 17 to 71 years (mean: 31). 24 patients had primary involvement and the other remaining 16 suffered from secondary lymphedema.

8 male (6 patients with primary involvement: 25%) and 32 females (18 cases with primary involvement: 75%) were evaluated in this study. Surgery (37.5%) and trauma (31%) were the commonest causes of the secondary lymphedema.

Four patients with a less than one year history of lymphedema, 18 patients suffered from the disease for 1 to 4 years, 3 patients for 4 to 10 years and the remainder 11 for more than 10 years (there was no reliable information for the remaining 4). Thirty patients (75%) have got unilateral involvement (16 cases with primary involvement: 66%) while, bilateral involvement was seen in 10 (25%) patients (8 cases with primary involvement: 34%). All of the patients had lower extremity involvement.

Patients with congestive heart failure, as well as patients known to suffer from renal dysfunction, hepatic failure and lower limbs venous or arterial insufficiency were excluded from the study.

None of them had an experience of using the SIPC devices formerly.

Patients were hospitalized for 48 hrs. Before beginning the therapy, a baseline fasting serum lipid levels were evaluated as a basic criterion in all patients.

During hospital stay, using the Pneumolymph® device the involved limb was subjected to a standard pressure for 2 consecutive days (included 8 hours daily). The patients used regular diet during the hospitalization and post treatment sampling time for evaluation of serum lipid level [1] A SIPC device manufactured in Iran was arranged so as to be at least 8 hours after the meal [6]. For this purpose during SIPC using, the patients didn’t have anything.

Finally, pre and post treatment serum lipid levels were compared and analyzed.

Among the studied patients, at initial evaluation 8 cases suffered from hypertriglyceridemia and 4 cases from hypercholesterolemia. Since pre and post SIPC levels of serum lipid changes (not its absolute amount) was to be evaluated, above patients were not excluded from the study.

After data collection, statistically analysis was performed using SPSS (version 16), by means of pare t-test ±2SE for determination of confidence interval of 95%.

Results

The pre SIPC mean serum triglyceride levels was 119.3 mg/dl (SD: 95.3), and the mean of serum cholesterol levels was 176.25 mg/dl (SD: 59), whereas post SIPC mean of serum triglyceride levels was 121.3 mg/dl (SD: 64.85) and mean of serum cholesterol levels was 189.75 mg/dl (SD: 56.11).

In 87.5% of individuals, post SIPC triglyceride levels showed an increase ($p=0.031$). Also, for 85% of patients, there was an increase of cholesterol levels ($p=0.000$).

In all of the patients with normal serum triglyceride and cholesterol levels, the rising did not exceed the upper limit of the normal range.

During the treatment session, no complications attributed to the use of SIPC were observed.

There is no relation between post SIPC increase of serum lipid levels and the number of involved limb as well as the etiology of the disease.

Discussion

As previously mentioned, although absolute count of triglyceride and cholesterol were increased 2 and 13.5 mg/dl after intervention, there is a statistically significant difference between pre and post SIPC serum levels. Also all parameters that could change the lipid levels (such as diet) were fixed in our study, so the cause of these changes only can be due to the intervention. Specially these changes were seen in many of patients (85–87.5%) not in some of them. It suggests that large molecules such as lipoproteins can pass into the circulatory system during SIPC usage which contradicts some researchers’ belief indicating that it is only water molecules that enter into the circulatory system which makes transient and unreliable decrease in limb size during SIPC therapy [7]. In fact, increase of serum lipids levels shows that proper use of SIPC in early stages of the disease could prevent chronic accumulation of the lymph, and its subsequent complications, especially fibrosis formation. The question that is raised, is whether lymph entrance as a lipid rich fluid into the serum following SIPC therapy, may have complications of transient hyperlipidemia? Since lymphedema patients should be frequently undergone the treatment, so the subsequent hyperlipidemia shows a similar pattern to that of postprandial hyperlipidemia. In the postprandial hyperlipidemia phenomenon, following a high fat meal, lipoproteins which contain high quantity of triglycerides enter into the serum. Many studies indicate the role of the postprandial hyperlipidemia in accelerating atherosclerosis and its relation with the cardiovascular events [6, 8–12], but this is due to increasing the lipids level above the normal range.

Our study suggested increased serum lipid levels following SIPC therapy, but the values were not out of normal ranges. It means that in spite of the increase observed in postprandial hyperlipidemia, the episodic
increase of the serum lipids following SIPC therapy is not clinically significant and the subsequent risks do not matter. So, the therapy is safe in the management of lymphedema.

We concluded that, despite the increase of lipid levels in serum following SIPC therapy which proves entrance of large molecules from interstitium to the blood vessels, the levels remain in normal range, which indicates safety of the this treatment with no worry about the episodic hyperlipidemia.

**Acknowledgment** The authors extended sincere gratitude to Maássoumeh Hassanpour for her critical suggestions and helpful comments in regard to editing the manuscript.

**References**

1. Gogia SB (2007) Role of VPL therapy for filarial lymphoedema in India [monograph on the internet]. India: Amla mediquip [cited May 20]. Available from: http://business.vsnl.com/vipel/LymphBook.html
2. Cheville AL, McGarvey CL, Petrek JA, Russo SA, Taylor ME, Thiadens SR (2003) Lymphoedema management. Semin Radiat Oncol 13(3):290–301
3. Richmand DM, O’Donnell TF Jr, Zelikovski A (1985) Sequential pneumatic compression for lymphoedema: a controlled trial. Arch Surg 120(10):1116–1119
4. Szuba A, Achalu R, Rockson SG (2002) Decongestive lymphatic therapy for patients with breast carcinoma-associated lymphedema. A randomized, prospective study of a role for adjunctive intermittent pneumatic compression. Cancer 95:2260–2267
5. Manjula Y, Kate V, Ananthakrishnan N (2002) Evaluation of sequential intermittent pneumatic compression for filarial lymphoedema. Natl Med J India 15:192–194
6. Jackson KG, Armah CK, Minihane AM (2007) Meal fatty acids and postprandial vascular reactivity. Biochem Soc Trans 35:451–453
7. Miranda F Jr, Perez MC, Castiglioni ML, Juliano Y, Amorim JE, Nakano LC, de Barros N, Jr LWG, Burihan E (2001) Effect of sequential intermittent pneumatic compression on both leg lymphoedema volume and on lymph transport as semi-quantitatively evaluated by lymphoscintigraphy. Lymphology 34:135–141
8. Elmas E, Külsch T, Suvačac N, Leweling H, Neumaier M, Dempfle CE, Borggreve M (2007) Activation of coagulation during alimentary lipemia under real-life conditions. Int J Cardiol 114:172–175, Epub 2006 May 30
9. Alipour A, Elte JW, van Zaanen HC, Rietveld AP, Cabezas MC (2007) Postprandial inflammation and endothelial dysfunction. Biochem Soc Trans 35:466–469
10. O’Keefe JH, Bell DS (2007) Postprandial hyperglycemia/hyperlipidemia (postprandial dysmetabolism) is a cardiovascular risk factor. Am J Cardiol 100:899–904, Epub 2007 Jun 26
11. Nitenberg A, Cosson E, Pham I (2006) Postprandial endothelial dysfunction: role of glucose, lipids and insulin. Diabetes Metab 32:28–33
12. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM (2007) Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA 298:309–316