Silicone gel sheet dressing for sclerodermatous type chronic graft-versus-host-disease (cGVHD)

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ABSTRACT

Systemic sclerosis is an autoimmune disease characterized by endothelial cell injury, fibroblast activation and immunological aberrations. Generalized form of the disease involves skin and other organs. Progressive sclerodermatous type cGVHD is the difficult type to treat. Immunosuppressors are the most commonly used treatment regimens.

Topical silicone gel sheet (SGS) were first used in the treatment of burn wound and following their initial successes have begun to be used in the treatment of hypertrophic scars and keloids. To best of our knowledge, this is the first patient with extensive sclerodermatous type cGVHD in whom SGS was applied on to the skin of the antecubital region. After a six months application of SGS, the skin of this region was remarkably soft and thick compared to other regions of the arm. The result indicate that SGS may be an useful tool for the treatment of extensive sclerodermatous type cGVHD.

Key Words: Graft-versus-host-disease, Dressing, Silicone.

ÖZET

Sklerodermatöz tip kronik graft-versus-host-hastalığının tedavisinde silikon jel kaplama uygulaması

Sistemik skleroz endotel hücre hasar, fibroblast aktivasyonu ve immünolojik bozukluklar ile karakterize bir otoimmün hastalıktr. Jeneralize şekilli cilt ve diğer organları tutar. Progresif tip sklerodermatöz kronik GVHH’nin
INTRODUCTION

Patients with cGVHD may manifest with a spectrum of problem including skin, sweat gland, hair, eyes, mouth, respiratory tract, liver, musculoskeletal, immune and hematopoietic system. For this reason cGVHD must be considered one of the major problems still facing the field of the blood and marrow transplantation. Especially, skin involvement of cGVHD patients is characterized by two types of clinical manifestation including sclerodermatous and lichenoid. One of the most disabling forms of cGVHD resembles systemic sclerosis (SSc) and shares various clinical and pathological features. Scleroderma-type cGVHD is a difficult complication to treat and if progressive, causes high risk of mortality and morbidity. In addition, progressive limitation of range of motion of joint may develop in patients with cGVHD. Immunosuppression with corticosteroids, anti-lymphocyte globulin, mycophenolate...
mophetil, extracorporeal photoimmunotherapy are the most commonly used treatment approaches with varying responses and limited success[1].

Topical SGS are widely used in plastic surgery as it has a high flexibility and has been found to be very effective in the treatment of contractures of hypertrophic scars and keloids[2]. SGS is a water-impermeable dressing material employed successfully in the management of burns. SGS also consistently liberates silicon oil in combination with antimicrobial agent ofloxacin. An antimicrobial agent or prostaglandin E1 releasing gel sheets have been utilized successfully for dermal burns, donor site or over the flap or graft without infection or hypersensitivity[3-5]. The addition of vitamin E to SGS has been reported to be particularly effective in short term prophylactic treatment of keloid and hypertrophic scars. We tried to treat a patients with sclerodermatous type cGVHD using SGS on the forearm. In this report, we present the first patient with extensive sclerodermatous type cGVHD treated with SGS.

A CASE REPORT

A 29-years-old male, diagnosed with Ph positive CML in 1st chronic phase, is transplanted within the second year of his diagnosis on May 1995. The patient was transplanted with unmanipulated PBSC, containing 4.94 x 10^6/kg CD34+ cells from his HLA identical ABO compatible 22-years-old female sibling. He did not develop acute GVHD and discharged from the hospital one month after the transplantation. One year later, he was diagnosed with cGVHD associated with complaints of oedema of the skin on the extremities, xerophthalmia and hepatic involvement. Skin biopsy revealed sclerodermatous type cGVHD. After the diagnosis CsA was reinitiated with oral corticosteroids. SGS was applied on his forearm for a duration of six month (Figure 1,2). We measured skin oedema by the ultrasonography.

The results of ultrasonographic measurements were shown on Table 1. The skin oedema improved considerably with SGS application and this region was remarkably soft and thick compared to other region of the arm (Figure 1,2).

**DISCUSSION**

The mode of the action of SGS in hypertrophic scar and keloid is not completely understood, but it has been suggested that hydration, not silicone, modulates the effects of keratinocytes on fibroblast. Various mechanisms by which SGS acts have been proposed. Perkins et al first reported in 1982 that silicone gel was an effective therapy in the prevention of scar contractures and hypertrophy due to burns. As opposed to compression garments used for the treatment of scars due to burns, the mechanism by which silicone gel acts has been thought not to be related to pressure[6]. Perkins et al have concluded that silicone gel treatment improves hypertrophic scars by releasing a

<table>
<thead>
<tr>
<th>Time (month)</th>
<th>Proximal part of SGS on the right forearm</th>
<th>SGS applied area on the right forearm</th>
<th>Control on the left forearm (SGS was not applied)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3.3 mm</td>
<td>3.4 mm</td>
<td>3 mm</td>
</tr>
<tr>
<td>1</td>
<td>3.3 mm</td>
<td>4.0 mm</td>
<td>3 mm</td>
</tr>
<tr>
<td>2</td>
<td>3.5 mm</td>
<td>4.5 mm</td>
<td>3.4 mm</td>
</tr>
<tr>
<td>3</td>
<td>3.5 mm</td>
<td>4.9 mm</td>
<td>3.4 mm</td>
</tr>
<tr>
<td>6</td>
<td>3.6 mm</td>
<td>5.4 mm</td>
<td>3.5 mm</td>
</tr>
</tbody>
</table>
low molecular weight silicone fluid and by hydrating the stratum corneum[2]. SGS are occlusive and cause an increase in hydration of the skin horny layer and in turn increase the permeability for water soluble compounds, including plasmatic proteins[7-9]. Based on the above fact, a logical hypothesis may be advanced that the hydrated horny layer that covers hypertrophic scars mobilizes diffusion exudate plasmatic proteins in the direction of the skin surface[8]. It has also been reported that fibroblast production and collagen proliferation improve the occluded wounds by decreasing oedema[9]. Reduction in hypertrophic scar formation by SGS treatment may be explained by the regulation of cytokine network[6]. It can also be explained by a mechanism in which SGS acts on stratum corneum and restores haemostatics by decreasing hyperemia and fibrosis, and thus ultimately alteration of the scar results. Hydration may account for softening and thickening of the skin of the patient. In addition, cytokines may cause healing through formation of fibroblast and collagen. It seems that the treatment of sclerodermatous type cGVHD using SGS may be an essential tool for improvement of joint range and motions. We believe that further prospective randomized trials are required to evaluate the clinical importance of this method.

REFERENCES

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