Rare Complication of Myositis in Chronic Graft-Versus-Host Disease

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In bone marrow transplantation (BMT), graft-versus-host disease (GVHD) is induced by donor lymphocytes, which recognize the antigenic differences on recipient tissues. Two distinct diseases are recognisable: acute and chronic GVHD. Acute GVHD is observed at 1–2 months after BMT and its clinical manifestations are erythematous skin, diarrhea and liver impairment. Chronic GVHD is observed at 3 months after BMT and continues for years if the disease becomes refractory to immunosuppressive treatment. The manifestations of chronic GVHD are skin rash, sicca syndromes (dry eye and dry mouth), bronchiolitis obliterans in the lungs, abnormal motility and stricture in the gastrointestinal tract, cholestasis in the liver, and variable cytopenia in the hematopoietic system, etc. Virtually all autoimmune disease manifestations have already been described in association with chronic GVHD. In the musculoskeletal system, fascitis is common in sclerodermatous GVHD, although underrecognized (1). It manifests as a limitation of the capacity of the skin to slide over the muscle and there is a decreased range of motion. Myasthenia gravis can also be occasionally seen. However, myositis with tender muscles and elevated muscle enzymes is a rare complication. In this issue, Takahashi et al (2) reported a patient who suffered from myositis in the course of chronic GVHD.

Although the involvement of skin and fascia is commonly observed in chronic GVHD, myositis is a rare event. Sullivan et al (3) reported that 4 of 52 patients (7.6%) had myositis as a complication of chronic GVHD. Parker et al (4) reported that myositis occurred in 11 of 318 patients (3.5%) with chronic GVHD; nine patients had other organ involvement and two patients had only myositis as a manifestation of chronic GVHD. Tse et al (5) reported a case in which myasthenia gravis and myositis occurred in chronic GVHD. In several case reports, patients developed myositis with or without other manifestations in the course of chronic GVHD (6–9). In Takahashi’s report, the immunohistochemical examination was performed on a biopsy sample of atrophied muscle. The histological findings indicated that most of the infiltrating cells were T cells, and the numbers of CD4+ and CD8+ cells were equal in perimysium, whereas the infiltrating CD8+ cells were more predominant than the CD4+ cells in endomysium. Such a precise analysis of infiltrating cells in the sample of muscle is informative for understanding the pathogenesis of myositis. Muscles of the truncus and the proximal muscle of extremities are involved in polymyositis of autoimmune diseases, and myositis associated GVHD usually involves the proximal muscles of extremities predominantly (4, 7). However, their case showed weakness and atrophy of the upper extremities, and to a lesser extent in the proximal muscles of lower extremities. It is interesting that the distribution of the involved muscles in their report is different from other reports.

It has been reported that prompt initiation of corticosteroid therapy results in a rapid improvement of myositis (10). Polymyositis can be the only manifestation of chronic GVHD. Awareness of this complication is important because it can be confused with other causes of muscle weakness after BMT. Therefore, special attention should be paid to muscle weakness in the course of chronic GVHD.

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References