ABSTRACT
Herpes zoster (HZ) is a viral disease caused by the Varicella-Zoster virus. Primary infections usually occur in childhood when the virus causes a varicella infection. Virus is then detained in spinal nerve ganglia in latent form. In adults, triggers such as stress, trauma, chronic and malignant diseases, AIDS, and even sunburns, lead to reactivation of the virus and clinical manifestation of the herpes zoster infection. The incidence of herpes zoster is higher in elderly populations and in people with damaged cell-mediated immunity. The typical clinical appearance of herpes zoster comprises clusters of clear vesicles and blisters on the erythematous base; clusters are unilateral and localized in one dermatome, which is innervated by the affected spinal nerve ganglion. In immunocompromised patients, the clinical appearance of HZ can be varied, often accompanied by local and/or systemic complications that, in some cases, can lead to lethal outcomes. We present a case of herpes zoster infection in a 59-year old woman with a two-year history of rheumatoid arthritis and mixed connective tissue disease. The illness exhibited acutely beginning with fever and malaise. The ulcerative form of herpes zoster was clinically present, with slow and only slightly archived epithelialisation. As a result, an atrophic scar remained on the affected skin.

Key words: Herpes zoster, rheumatoid arthritis, mixed connective tissue disease.

INTRODUCTION
Herpes Zoster is an infectious disease caused by the Varicella-Zoster virus (family Herpetoviridae), and it usually occurs in adults. The illness is the second manifestation of the Varicella-Zoster infection. The first attack of the virus happens in most cases during childhood, when it causes varicella infection (1). After varicella infection, the virus remains in spinal nerve ganglia in a latent form (2). In some cases, when the immune system is disturbed by benign causes (stress, trauma, sunburns, etc.) or severe causes (chronic and malignant diseases, AIDS, radiotherapy, etc.), the virus starts to replicate and becomes active again after many years in latency. Clinical manifestations of herpes zoster are usually unilateral (in severe immunodeficiency, symptoms can be disseminate and clinically resemble varicella) and located in one dermatome, which is innervated by the affected spinal nerve. In the beginning, the skin is sensitive; pain is present and can be so severe as to mimic some other diseases, such as heart attack, migraine, or acute appendicitis (1). After 1-2 days, skin lesions can be seen.
On the erythematous base, varying numbers of vesicles and blisters with serous or serohemorrhagic fluid are present. They leave deep erosions or ulcers that can lead to scarring. The most common problem following herpes zoster infection is hyperpigmentations. Pain is present at the very beginning of the illness, during, and 2-3 months (sometimes longer) after the resolution of skin manifestations (postherpetic neuralgia) (1). A diagnosis is usually made on clinical grounds, but histopathology, viral culture, and serological examination can also be used to confirm the diagnosis. Antiviral drugs (Acyclovir, Famcyclovir, Valacyclovir) and antiviral ointments are the basis for herpes zoster therapy. Antibiotics are also used, as well as vitamins, analgesics, and sedatives (in difficult cases of postherpetic neuralgia) (1). In immunosuppressed patients, the clinical manifestation of herpes zoster is complicated. Ulcerative, necrotic or disseminate forms of herpes zoster are seen in these patients; after resolution, they leave varicelliform, deep scars on the affected skin. In cases like this, relapses of the disease are possible, but they are very rare.

**CASE REPORT**

A 59-year old female patient with a two-year history of rheumatoid arthritis (RA) and mixed connective tissue disease (MCTD) was admitted to the Dermatology Unit with clinical manifestations of herpes zoster (HZ) infection localized on the right half of the trunk in the dermatome that innervates TH 12. Subjective, severe pain was present, as well as fever and malaise. Clinical examination showed numerous vesicles and bullas with hemorrhagic fluid on the erythematous base (figure 1). After 48 hours, these efflorescences became confluent and the affected epidermis became eroded, leaving large, deep, erosive surfaces in some parts, covered with eschara (figure 2). A diagnosis was made on clinical grounds. Laboratory studies showed a normal sedimentation rate; a complete blood count and urine analysis were within normal limits. Rheumatoid factor was elevated. Abdominal ultrasound and chest radiography showed no abnormalities. Antiviral therapy (Acyclovir tbl 400 mg, administered 5 times a day for 10 days) was used to treat herpes zoster; antibiotic therapy (Cephraxon amp 2 g/day for 7 days) was used as well. Topical therapy comprised an erosive surface antibiotic gel and local antiseptic. To treat RA and MCTD, the patient was given Methotrexate (15 mg per week) and Prednisolone (15 mg per day). The reaction to antiviral and antibiotic therapy was not very good; the clinical course of a viral disease that usually lasts for 2 to 3 weeks was delayed. Epithelialisation of the deeply eroded surface was very slow (figure 3). It took two months until a large, atrophic scar appeared on the skin affected by infection. Severe pain was present throughout the entire course of the illness, and postherpetic neuralgia lasted for 3 months. Local and oral analgesics were used to suppress the pain.

**Figure 1.** Numerous vesicles and bullas with hemorrhagic fluid covered with anesthetic talc, localized on the erythematous base on the right side of the trunk.

**Figure 2.** Ulceration covered with eschara and pieces of eroded epidermis.
DISCUSSION

HZ occurs more frequently in patients with RA and MCTD than in the general population (3). Impaired cellular immunity is probably the reason for this high incidence (4). Clinical manifestation of HZ in patients with RA and MCTD varies. Some authors suggest that the course of HZ in these patients is benign and self-limiting, while other authors have observed patients challenged with life-threatening herpes zoster infection, in which case skin manifestations were accompanied by severe systemic involvement (3,5,6). HZ causes significant morbidity in immunosuppressed patients, while the reported mortality rate is below 5%. Complications are much more frequent in these patients compared to the general population, including meningoencephalitis, hepatitis, retinopathy, vasculopathy, coetaneous dissemination, and particularly delayed healing (7). Our experience supports findings by Antonelli et al (3). In our case, the disease had an acute onset with subjective features such as fever, malaise and severe pain. The ulcerative type of HZ was presented on the skin without any systemic manifestation. Despite a good recovery, the course of the disease was prolonged and self-limiting because it took two months until epithelialisation was complete.

ABBREVIATIONS:

HZ- herpes zoster
MCTD- mixed connective tissue disease
RA- rheumatoid arthritis

REFERENCES