May Calcitonin Spray Cause Nasal Deformation?

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Calcitonin is a powerful inhibitor of bone resorption and as such has been used in osteoporosis, Paget's disease of the bone, skeletal bone pain and in hypercalcemia of malignancy. Recently, a new device, a nasal spray, has been introduced for the administration of salmon calcitonin. The results of many studies have demonstrated that intranasal calcitonin preparations are well tolerated, increase patient compliance and reduce the incidence of side effects [1, 2].

We report the case of a 58-year-old woman affected by Sjögren's syndrome who developed a nasal deformation during the treatment with calcitonin nasal spray for her osteoporosis.

Fig. 1. Nasal deformation possibly caused by calcitonin spray.

Case Report

A 58-year-old woman with Sjögren's syndrome had two 60-day therapeutic cycles with salmon calcitonin spray, 100 IU per nostril, once a day, for her osteoporosis. During the therapy, she had complained of severe nasal pain. During the second cycle, she had noted a progressive bilateral deformation of the alar nasal cartilages, which appeared more pronounced on one side. Two depressed, symmetric, rectangular areas finally appeared (fig. 1), so therapy was stopped after 4 months. An otolaryngological evaluation showed no nasal lesions; radiography demonstrated nasal septum deviation. A year after the interruption of the calcitonin spray therapy, the nasal cartilage deformation persisted, and subjective symptoms were limited to dryness of the nasal passages.

The exact temporal connection between the appearance of nasal deformation and calcitonin therapy leads us to assume a pathogenic correlation. Possible causes of such deformation may be looked for, in our opinion, in various elements: first of all in preexisting pathology, that is Sjögren's syndrome which is characterized by mucosal atrophy, sometimes also by atrophic rhinitis with connective tissue sclerosis and a reduction in the number of submucosal glands; secondly, in the nasal septum deviation which might have modified the angle of insufflation concentrating the flux onto very limited areas, thus increasing the possibility of traumatic tissue lesions; finally, in the treatment length, once a day for many months, which might have made irreversible the small damages caused, day after day, by the calcitonin spray on an anatomically injured mucosa. The absence of propellents in a calcitonin spray should prevent the sudden thermal lowering on the inner surface of the nasal passages which happens at the moment of delivery with common sprays and which is responsible for vasoconstriction and serious damage of mucosa and perichondrium. Nevertheless it is possible that each delivery may provoke moderate but repeated vasoconstriction phenomena; these, together with preexisting poor mucosal conditions, nasal septal deviation and naturally scanty

vascularization of cartilages could have provoked an irreversible ischemia and consequent alar cartilage collapse. So, when prescribing a drug for the intranasal route of administration, it is important to envisage possible pathological preexisting conditions which may be responsible for mucosal and nasal passage alterations, for nasal cycle variations and mucociliar clearance. The excellent compliance in patients and the positive clinical effects of calcitonin spray justify its future use on a vaster scale, and new local side effects will probably appear. We wish to draw the dermatologists' attention to these possible, still unknown effects.

References

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