Oral leukoplakia is the most common potentially malignant lesion of the oral cavity. The malignant conversion of leukoplakia has been reported in up to 20% of lesions. Oral leukoplakia (OL) is defined as a white patch or plaque that will not rub off and cannot be characterized clinically or histologically as any other disease. The prevalence of OL in the general population ranges from 0.2% to 11.7%.

A 62-year-old man, nonsmoker, with type 2 diabetes treated with oral antidiabetic medications for 5 years, was admitted to the Department of Maxillofacial and Oral Surgery, Medical University of Gdansk, due to a white oral lesion. A histological examination confirmed leukoplakia. Blood test results were as follows: glucose, 7.14 mmol/l (reference range, 3.8–6.5 mmol/l); hemoglobin A1c, 6.5% (4.3–6.1%); cholesterol, 3.42 mmol/l (3.37–5.18 mmol/l); high-density lipoprotein cholesterol, 0.93 mmol/l (1.04–2.07 mmol/l); low-density lipoprotein cholesterol, 1.79 mmol/l (0.00–3.37); and triglycerides, 1.51 mmol/l (0.51–2.83 mmol/l). The patient was treated with a cream containing 0.05% tretinoin (Retin-A, Pierre Fabre, France). Topical treatment had been applied to the affected area twice a day for 8 weeks. Control visits were scheduled at 2, 4, 6, 8 weeks, and 6 months after treatment completion. There were no substantial side effects. Partial remission was observed after 1 week and complete clinical remission after 8 weeks of treatment (FIGURE 1).

To our knowledge, this is the first report in Poland on OL successfully treated using topical tretinoin. We observed the correlation between OL and diabetes. Our findings are in line with other studies indicating that the development of diabetes predisposes to OL.1,2 The pathomechanism of OL in diabetics is probably associated with progressive atrophy of the oral mucosa owing to a decreased rate of salivary secretion and low salivary pH, which leads to the loss of the normal protective barrier. The elevated blood glucose level increases the amount of free radicals and reduces the antioxidant potential of tissues.3 In addition, there can be microangiopathy in the gingival tissues causing tissue hypoxia and reduced blood supply, which together with the impaired cellular immune response may play a role in the development of OL.4 The higher incidence of OL in diabetics should alert us to the condition of oral mucosa on standard physical examination. Patients
affected by OL can be offered an effective therapy, both noninvasive (topical use of corticosteroids, retinoids, bleomycin, systemic use of vitamins, photodynamic therapy) and invasive (excision surgery, electrocoagulation, cryosurgery, laser surgery). The treatment with topical tretinoin allows to reduce the number and size of OL lesions, thereby lowering the number of ablative procedures.

We believe that OL in patients with diabetes deserves to be adequately recognized and investigated.

REFERENCES