North Clin Istanb 2020;7(2):185–186
doi: 10.14744/nci.2019.06881

Tacrolimus-induced gingival hyperplasia and recovery from tacrolimus to everolimus switching

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Cite this article as: *Oral A, Bakan A. Tacrolimus-induced gingival hyperplasia and recovery from tacrolimus to everolimus switching. North Clin Istanb 2020;7(2):185–186.*

A 28-year-old female patient presented with a complaint of gingival hyperplasia (GH). The patient has diagnosed with chronic renal failure due to focal segmental glomerulosclerosis. She had a renal transplant eight months before admission to our hospital. The patient was using tacrolimus (TcR) 9 g/day, mycophenolate mofetil 720 mg/day, prednisolone 5 mg/day, lansoprazol 40 mg/day. She did not use nifedipine or phenytoin. Physical examination revealed GH (Fig. 1), but there were no other pathologies. General systemic examination and laboratory tests were normal. The blood TcR level was 11 ng/ml (normal range: 5–20 ng/ml). TcR was interrupted and switched to everolimus. It was observed that GH improved after one month in the outpatient clinic (Fig. 2).

GH occurs as unwanted side effects of the drugs. These drugs are usually immunosuppressants (such as cyclosporin A (CsA) and TcR), calcium channel blockers (nifedipine) and anticonvulsants (phenytoin) [1]. CsA-induced GH was found more frequently than TcR. There are even studies showing that TcR does not cause GH alone [2, 3]. When CsA-induced GH is developed, it is shown that GH is reduced if CsA is replaced by TcR [4]. In our case, TcR-induced GH was switched to everolimus, and GH regressed.

Figure 1. Tacrolimus-induced gingival hyperplasia.

Figure 2. Improved gingival hyperplasia.

Received: May 15, 2019 *Accepted:* September 13, 2019 *Online:* November 20, 2019

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Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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