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

A 28-year-old female patient presented with a complaint of gingival hyperplasia (GH). The patient had been diagnosed with chronic renal failure due to focal segmental glomerulosclerosis. She had a renal transplant 8 months ago. The patient was using tacrolimus (TcR) 9 g/day, mycophenolate mofetil 720 mg/day, prednisolone 5 mg/day, lansoprazol 40 mg/day. She didn’t 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 drugs. These drugs are usually immunosuppressants (cyclosporin A (CsA), TcR, etc.), calcium channel blockers (nifedipine) and anticonvulsants (phenytoin). CsA-induced GH was found more frequently than TcR. There are even studies showing that TcR doesn’t perform GH alone. When CsA-induced GH is developed, it is shown that GH is reduced if CsA is replaced by TcR. In our case, TcR-induced GH was switched to everolimus and GH regressed.