A decade ago, the treatment of choroidal neovascularization (CNV) secondary to various macular diseases like age-related macular degeneration, pathologic myopia, and angioid streaks was a crucial challenge. Laser photocoagulation could be performed for juxtapfoveal and extrafoveal CNVs and different surgical treatment options like macular translocation surgery and subretinal CNV excision were available. The published studies were usually about neovascular age-related macular degeneration (nAMD), and we tried to adapt them to the other causes of CNV. However, the outcomes of these treatment options were variable and far away from patient and physician satisfaction [1].

In early 2000s, photodynamic therapy (PDT) was introduced and for the first time we could treat the subfoveal lesions with a non-surgical method; however, again the treatment outcomes were mostly about the patients who lost less than 3 LogMAR lines [1]. After the intravenous administration of Verteporfin, a photoactive agent; non-thermal diode laser was applied to the CNV. PDT was satisfactory enough for preserving visual acuity; however, visual acuity increases could have been achieved only in a very low portion of the patients. Also, lately many of the patients developed chorioretinal atrophy areas corresponding to the previous PDT spots. Then the intravitreal anti-vascular endothelial growth factor agents came with better visual results [1]. The first anti-VEGF drug used and licensed to treat CNV secondary to nAMD was pegaptanib Pegaptanib [1]. It was an aptamer of VEGF A-165 receptor isoform, and selectively targeted the mentioned receptor of VEGF [1]. First results were promising, but pegaptanib could not achieve the visual results of the following drugs which were antibodies against all isoforms of VEGF-A: the off label one bevacizumab, and on label one ranibizumab [1]. The first results of single center, non-randomized studies concerning about intravitreal bevacizumab treatment in nAMD were very satisfactory and we began to talk about visual acuity gains. Then in 2005 ranibizumab was introduced which was the rhuFab fragment of the monoclonal antibody against all isoforms of VEGF-A. Many studies were published about the efficacy of ranibizumab in the treatment of CNV secondary to various diseases. The monthly, quarterly, as-needed treatment regimens were evaluated in the studies such as MARINA, ANCHOR, EXCITE, PRONTO [1].

After these studies, monthly and especially as-needed treatment regimens have become very popular. Lately, new flexible treatment
sustained-releases, refillable reservoirs, and suprachoroidal delivery are being investigated [1,3].

As a conclusion, two decades ago, we were desperately inevitable in the treatment of CNV secondary to macular diseases; a decade ago, we became visual protectors; finally thank to anti-VEGF agents that we now may save the visual acuity in most of the CNV patients and may obtain significant visual acuity gains in nearly half of the CNV patients.

References