Initial and unique treatment of macular edema due to branch retinal vein occlusion with antiangiogenic agents. A comparative pilot study
Tratamiento inicial y único del edema macular asociado a oclusiones de rama venosa retiniana con fármacos antiangiogénicos. Estudio piloto comparativo

Dear Sir,

Macular edema (ME) is the main cause of loss of vision associated to non-ischemic occlusion of the retinal venous branch (ORVR-NI) (1). A number of treatments have been tried out, including macular grid photocoagulation, intravitreous triamcinolone, vitrectomy associated to peeling of the internal limiting membrane or dissection of the arterial-venous adventitia, with variable results involving complications and/or side effects. VEGF is essential in ME associated to ORVR-NI (2), and accordingly its inhibition by intra-vitreous injection of anti-VEGF drugs constitutes an important therapeutic target (3). However, to this date it has not been established whether a drug which inhibits all the isoforms of VEGF-A could be superior to another one that selectively inhibits isoform 165.

The objective of this study was to make a comparative assessment of the efficacy of two anti-angiogenic drugs (Pegaptanib and Ranibizumab) as the initial and single treatment for ME associated to ORVR-NI.

Twelve patients with ME secondary to ORVR-NI and macular thickness above 400 µ were randomly and consecutively assigned to the treatment group with Pegaptanib and Ranibizumab. The treatment protocol consisted in injecting the first dose of each drug and treat again every six weeks the cases in which the macular thickness remained above 300 µ. The patients were assessed every six weeks up to completion of a 30 week follow-up period.

With Pegaptanib (n=6) the mean best corrected visual acuity (BCVA) improved from logMAR 0.617 ± 0.17 to 0.367 ± 0.10 (p=0.026); the mean macular thickness went down from 498.83 ± 86.37 microns to 266.33 ± 20.70 (p=0.028). After the 30 week follow up an average of 3.3 injections per patient were needed (fig. 1). With Ranibizumab

Fig. 1: ME secondary to ORVR-NI (A) treated with three injections of Pegaptanib (Macugen®) (B). Improvement from initial 712 microns to 275 microns. The vision improved from 0.7 logMAR (decimal equivalent of the Snellen scale: 0.20) to 0.5 logMAR (decimal equivalent of the Snellen scale: 0.32).

Fig. 2: ME associated to ORVR-NI (A) treated with two injections of Ranibizumab (Lucentis®) (B). Improvement from 881 microns to 283 microns. Vision went from 0.80 logMAR (decimal equivalent of the Snellen scale: 0.02) to 0.2 logMAR (decimal equivalent of the Snellen scale: 0.63).
the mean BCVA improved from logMAR 0.717 ± 0.19 to 0.351 ± 0.21 (p=0.026); the mean macular thickness went down from 545.67 ± 87.66 microns to 260.67 ± 31.76 (p=0.028). After the 30 week follow up an average of 2.3 injections were needed for each patient (fig. 2). No statistically significant difference was detected between both groups in what concerns improvement of BCVA (p=0.715), reduction of macular thickness (p=0.173) or number of treatments (p=0.141). All the patients in whom the entire macular thickness map was reduced below 300 microns (Retinal Map Thickness analysis protocol) remained stable without ME increase throughout the follow-up and did not require subsequent treatments up to the 30 weeks follow-up.

Our preliminary results suggest that treatment of ME associated to ORVR-NI implemented initially and uniquely with antiangiogenic drugs produces similar efficiency when utilizing Pegaptanib (Macugen®) or Ranibizumab (Lucentis®). Randomized multicentre studies with a higher number of cases and follow-up time are necessary to confirm the conclusions of this pilot study.

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REFERENCES