Commentary: Ocular changes after bariatric surgery - An evolving domain

Globally the prevalence of obesity is reaching epidemic proportions, both in the developed and developing countries. As per WHO estimates, more than 1.9 billion adults over 18 years are overweight, of which around 650 million are obese. Bariatric surgery has emerged as an effective treatment modality for management of obesity.[1]

The ocular changes secondary to obesity can be explained by mechanical and chemical changes. Increase in intraorbital adipose tissue leads to mechanical compression of episcleral outflow which precipitates rise in intra-ocular pressure (IOP) and subsequently glaucoma.[2] Hyperleptinemia, which is associated with obesity causes oxidative damage to the trabecular meshwork leading to glaucoma.[1] Ghrelin is another important biomolecule which acts as an antioxidant and a neuroprotective agent in the eye, especially on the retina.[1,3] Lower levels of ghrelin have been found in obese individuals as compared to non-obese individuals. Its association with glaucoma was first demonstrated by Katsanos *et al.* who demonstrated lower ghrelin levels in the anterior chamber of glaucoma patients in comparison to controls.[3] Increase in retinal nerve fiber layer (RNFL) thickness and retinal ganglionic cell layer (RGCL) thickness respectively and a reduction in IOP have been demonstrated in patients undergoing bariatric surgery for obesity. However, in the present study by Gonul el al., the authors did not observe any significant change in RNFL and visual field parameters post-bariatric surgery. It would have been interesting to note if there was any significant change in IOP following the surgery in the current study.[4]

Changes in choroidal thickness (CT) in obesity has been a matter of great contention in literature. There are contrasting

studies that have shown the CT to increase, decrease and even remain unaffected by obesity. Based on the mechanical theory, obesity can lead to an increase in intraorbital venous pressure due to excessive adipose tissue. This can affect drainage of choroidal vessels causing backpressure changes and subsequently an increase in CT. These changes have been demonstrated in the study by Gonul *et al*.[4] Conversely, the chemical theory which is based on biomolecular changes distinctly supports a reduction in CT in obesity. Nitrous oxide (NO) levels which cause vasodilatation have been found to diminish with obesity.[5] This can not only lead to choroidal constriction but also lead to an overall reduction in ocular blood flow which can trigger raised IOP and glaucoma. Obesity has also been associated with increased levels of other vasoconstrictor molecules such as endothelin-1 (ET-1) and angiotensin-II (Ang-II).[5] Hyperleptinemia which is seen in obesity also leads to an increase in sympathetic outflow and thus cases vasoconstriction. Due to these molecular changes, a thinning of choroid may be seen in obese people which has been shown to improve over time after a bariatric surgery.

Dogan *et al*. have shown a significant increase in central macular thickness (CMT) and total macular volume (TMV) at 3 months and 6 months post-bariatric surgery.[5] However, this increase was within the normal range of these parameters. A meta-analysis has shown that bariatric surgery has beneficial effects on progression of diabetic retinopathy (DR) but there was insufficient evidence regarding its association with regression of DR.[6]

Macular pigments, especially lutein and zeaxanthin, filter the harmful blue light and thus act as an important antioxidant carotenoids that protects the macula.[7] Adipose tissue actively absorbs these neuroprotective pigments thereby reducing their concentration in the macula. Hammond *et al*. have demonstrated an inverse relationship between the macular pigment optical density (MPOD) and body mass index (BMI) and the percentage of body fat.[7] Additionally, obesity is a known pro-inflammatory condition in lines with age related macular degeneration (ARMD). Schaumberg *et al*. has shown that men with normal BMI had lower incidence of ARMD.[8] This relationship between obesity and ARMD needs to be explored further by long-term studies which can also evaluate the possibility of stabilization or slowing down or even reversal of ARMD changes post-bariatric surgery.

In conclusion, obesity is a known risk factor for glaucoma, DR, and ARMD while its association with the CT is debatable. Further long-term studies with age matched controls are warranted to establish the role of bariatric surgery on ocular changes, including its role on ocular parameters such as IOP, CT, CMT, CMV, MPOD, RNFL, and RGCL. It is also imperative to perform molecular and cellular studies to gain further insight into the role of obesity and bariatric surgery on the eye.

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