FullThicknessMacularHoleas aComplication
ofIdiopathicParafovealTelangiectasia:
A Spectral-Domain OCT Analysis

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Introduction

Full thickness macular hole (FTMH) is a rare complication of idiopathic parafoveal telangiectasia (IPFT), with only a few reports to date. Prior reports have analyzed the anatomical changes at the macular using time-domain optical coherence tomography (TD-OCT), which is inferior in terms of image resolution, reproducibility, and artefact minimization compared to spectral-domain optical coherence tomography (SD-OCT).

The aim of this study is to document the macular changes in a patient with FTMH secondary to IPFT using SD-OCT, and to propose possible mechanisms of FTMH formation.

Methods

Fundus fluorescein angiography (FFA) confirmed the diagnosis of IPFT. SD-OCT was performed using the Spectralis OCT system (Heidelberg Engineering, Vista, California, USA).

Case Report

A 62 year old Asian woman with a history of hypertension and hyperlipidemia presented with blurring of vision without metamorphopsia in the left eye for a few months. Visual acuity was 20/30 in the right eye and 20/60 in the left eye.

In the right eye, disruption of the retinal layers at the fovea was also noted. The external limiting membrane (ELM) and inner segment/outer segment (IS/OS) junction were disrupted at the fovea, and the retinal architecture was disorganized. Small cystoid spaces were seen in the retinal layers at the fovea.

In the left eye, a full thickness disruption of the macular layers was seen down to the retinal pigment epithelium (RPE). Intra-retinal cystoid changes were seen in the nasal margin of the macular hole with an up-sloping retinal contour and a prominent posterior hyaloid face. The inner limiting membrane (ILM) was completely discontinuous over the hole. The ELM and IS/OS junction were also disrupted with lateral separation of the photoreceptors.

Discussion

The TD-OCT features of FTMH associated with IPFT reported to date include intra-retinal cavitation with overhanging, undermined edges to the macular hole, an intact ILM bridging the hole, and an absent ELM. These features are consistent with the theory that hole formation occurs secondary to atrophic changes associated with chronic cystoid macular edema.

Our findings on SD-OCT however suggest a different mechanism. The edge of the macular hole sloped upwards, with cystoid changes in the retinal layers immediately adjacent to the hole, a taut posterior hyaloid layer, and full thickness disruption of retinal layers. These findings are similar to the findings seen in idiopathic FTMH which are believed to arise from anomalous vireo-macular traction. Olson has suggested that the abnormal foveal telangiectasis alter the adhesion between the hyaloid face and the macula, leading to macular hole formation from tractional effects.

In conclusion, we have reported on the SD-OCT findings of FTMH formation associated with IPFT. The imaging findings suggest that vitreo-macular traction may be the underlying mechanism of hole formation.

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