

Introduction

Diabetes mellitus (DM) is defined as a group of metabolic diseases the common feature of which is an elevated blood glucose level (hyperglycemia). Chronic hyperglycemia is associated with the long-term consequences of diabetes that include damage and dysfunction of the cardiovascular system, eyes, kidneys, and nerves. The complications of diabetes are often divided into two groups: microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (ischaemic heart disease, stroke, peripheral vascular disease). Together, these make diabetes the seventh most common cause of death in the developed world (**Romero-Aroca et al., 2008**).

Diabetic macular edema (DME) is the most common cause of visual impairment in patients with diabetes mellitus and affects approximately 75,000 new patients in the United States every year. The pathogenesis of DME is complex and multifactorial. It occurs mainly as a result of disruption of the blood-retinal barrier (BRB), which leads to increased accumulation of fluid within the intraretinal layers of the macula (**Bhagat et al., 2009**).

Hyperglycemia is a major risk factor for development of diabetic retinopathy. It leads to high intracellular levels of glucose, formation of free radicals (oxidative stress), and protein kinase C activation. Chronic hyperglycemia also leads to formation of advanced glycation end products (AGEs), which may be the inciting event for diabetic retinopathy and maculopathy. Accumulation of AGEs in the vitreous and vitreoretinal interface is associated with neurovascular injury seen in diabetic retinopathy (**Witmer et al., 2003**).

Diabetic macular edema tends to be a chronic disease. Although spontaneous recovery is not uncommon, 24% of eyes with CSME and 33% of eyes with center involving CSME will have a moderate visual loss (15 or more letters on the ETDRS chart) within 3 years if untreated. The incidence of macular edema increases significantly with increasing severity of diabetes in both younger onset and older onset diabetic patients (**Bhagat et al., 2009**).

Although DME is found frequently in patients with retinopathy secondary to diabetes mellitus types it was noted that the morphologic changes within the different retinal layers corresponding to the leakage pattern and exudative activity are poorly identified by clinical examination (**Bolz et al., 2009**).

DME can be diagnosed using noncontact stereoscopic biomicroscopy, contact lens biomicroscopy, fluorescein angiography (FA), and optical coherence tomography (OCT) (**kozak et al., 2008**).

Fluorescein angiography is a standard method used to evaluate patients with DME that is sensitive for qualitative detection of fluid leakage (**Kang et al., 2004**).

Although FA can assess DME qualitatively, OCT provides quantitative measurement of foveal thickness. Therefore, the pathophysiologic aspect of DME can be determined by FA, and its anatomical features such as the extent of retinal thickening and the retinal layer involved can be assessed best using OCT. multiple studies have shown that both FA and OCT are highly sensitive in detection of ME of various etiologies, with OCT superior to FA according to certain parameters (**Kang et al., 2004** , **Özdek et al., 2005**).

OCT, gained significant importance throughout the last decade because it allows evaluating retinal morphologic features in detail similar to an in vivo histologic examination. However, conventional OCT imaging is based on 6 radial, cross-sectional scans, and the information therefore is limited to a few randomly selected locations and an overall low resolution of structural details. The fourth-generation OCT, high-definition OCT (HD OCT), uses a fast spectral-domain technique and performs scans in a raster pattern throughout the entire macular area, at a superior resolution of 5 μ m in axial and 20 μ m in transverse directions. As a result, the retinal morphologic features can be imaged at all locations transversally and can be located to all retinal layers axially. These advances in OCT technology offer novel insight per se, but also may be used to complement findings from conventional angiographic evaluation (**Bolz et al., 2009**).

The correlation of morphologic and pathophysiologic information maintained by 2 different examination techniques, FA and OCT, is an important step to gain further insight in the pathophysiologic features of DME. Because new treatment strategies of this disease, such as anti-vascular endothelial growth factor agents administered intravitreally, are currently under investigation, a detailed knowledge of pathologic retinal alterations is essential. So many studies were done to provide a direct correlation of FA and OCT findings in DME in each macular location and for each angiographic pattern to identify the specific impact of vascular leakage on retinal morphologic features (**Bolz et al., 2009**).