Coagulation cascade and therapeutic interventions of diabetic macular degeneration.

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Abstract

Diabetic macular oedema is a leading cause of vision loss. Macular oedema aetiology appears to be multifaceted. The gold standard of therapy for macular oedema is laser photocoagulation. However, there are some circumstances where laser therapy is ineffective. For the therapy of this illness, several therapeutic strategies have been presented. Several variables and mechanisms implicated in the aetiology of macular oedema are discussed in this article (vasoactive factors, biochemical pathways, anatomical abnormalities). Combining pharmaceutical and surgical therapy appears to be the best technique for managing diabetic macular oedema.

Keywords: Diabetic macular oedema, Macular degeneration, Laser photocoagulation, Edema.

Introduction

Hyperglycemia is a major risk factor for diabetic retinopathy development. It causes elevated intracellular glucose levels, the production of free radicals (oxidative stress), and the activation of protein kinase C. Chronic hyperglycemia also promotes the production of Advanced Glycation End products (AGEs), which may be the trigger for diabetic retinopathy and maculopathy. The accumulation of AGEs in the vitreous and vitreoretinal interface is linked to the neurovascular damage seen in diabetic retinopathy. Although damaged BRB is important in the pathophysiology of diabetic macular oedema, altered vitreomacular interface may contribute considerably to macular edoema progression. Other factors that contribute to diabetic macular oedema progression include hypoxia, abnormal blood flow, retinal ischemia, and inflammation. Within the diabetic retinal vasculature, inflammatory mechanisms such as increased VEGF levels, endothelial dysfunction, leukocyte adhesion, decreased Pigment Epithelium Derived Factor (PEDF) levels, and increased protein kinase C production induce BRB breakdown and increased vascular permeability. AGEs have been implicated in the activation of all of these mechanisms in animal studies. Using fundus contact lens bio microscopy, diabetic macular oedema is diagnosed stereoscopically as retinal thickness in the macula. Diabetic macular oedema was characterised by the Early Treatment Diabetic Retinopathy Study as retinal thickness or the presence of hard exudates within 1 disc diameter of the macula's centre. This criterion has been utilised consistently in the majority of diabetes research papers. Older onset diabetic patients have a tendency to develop macular edema earlier in the course of their disease (prevalence: 3–8% with up to 3 years of disease duration) compared to younger onset diabetic patients (prevalence: 0.5% with up to 10 years of disease duration) [1]. In the presence of macular edema, 50% of older onset diabetic patients have visual acuity worse than 20/40 compared to 20% of younger onset diabetic patients. However, it is recommended that in all diabetic macular oedema patients, efforts be made to correct elevated blood glucose, lipids, lower raised blood pressure, and enhance cardiac or renal status.

In contrast to diffuse DME, which is characterised by widespread areas of leakage in the area centralis, focal DME is distinguished by well-defined, localised areas of leaking from the microaneurysms on the FA. Furthermore, focused DME responds to focal laser photocoagulation, whereas diffuse DME presents a more difficult clinical scenario and is often resistive to laser photocoagulation. In some circumstances, a grid design of laser treatment may be beneficial [2]. Patients with very severe non-proliferative diabetic retinopathy have a times larger risk of developing diffuse macular edoema than those with less severe non-proliferative diabetic retinopathy, while patients with proliferative diabetic retinopathy have a 7.7 times greater risk.

The blood-retina barrier

Disruption of the BRB is the most common cause of diabetic macular oedema. The BRB separates the neurosensory retina from the eye's vascular component. The upkeep and effective operation of the BRB is a complex process involving the interaction of various components [3]. The BRB is made up of two basic parts: the outside barrier and the inside barrier. The inner BRB is a biological unit composed mostly of tight junctional complexes between RVE cells in the retina [4].

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Improved Screening Therapy techniques for Diagnosis

Traditional macular disease evaluation methods, like as slit-lamp biomicroscopy and stereo fundus photography, are relatively insensitive to minor changes in retinal thickness. There are several additional diagnostic ocular imaging modalities available, including ultrasound, high-frequency ultrasound, and scanning laser ophthalmoscopy. However, the resolution of these approaches is insufficient to generate therapeutically meaningful images of retinal anatomy [5].

Conclusion

Proteome examination of vitreous samples from AMD patients who received intravitreal combination therapy that included a core vitrectomy, steroids, and bevacizumab revealed AMD-specific proteome alterations. The discovered AMD-related proteins shed light on the pathophysiological alterations associated with AMD.

References

1. Feener EP. Plasma kallikrein and diabetic macular edema. Curr Diabetes Rep. 2010;10(4):270-5.
2. Teufel DP, Bennett G, Harrison H, et al. Stable and long-lasting, novel bicyclic peptide plasma kallikrein inhibitors for the treatment of diabetic macular edema. J Med Chem. 2018;61(7):2823-36.
3. Panagiotoglou TD, Ganotakis ES, Kymionis GD, et al. Atorvastatin for diabetic macular edema in patients with diabetes mellitus and elevated serum cholesterol. Ophthalmic Surg Lasers Imaging. 2010;41(3):316-22.
4. Koss MJ, Hoffmann J, Nguyen N, et al. Proteomics of vitreous humor of patients with exudative age-related macular degeneration. PloS One. 2014;9(5):e96895.
5. Emerson MV, Lauer AK. Emerging therapies for the treatment of neovascular age-related macular degeneration and diabetic macular edema. Bio Drugs. 2007;21(4):245-57.

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