

# Analysis of Autoimmune Retinopathy and age-related macular degeneration

Aaron Domej\*

Department of Ophthalmology, Duke University Medical Center, Durham, North Carolina

\*Corresponding author: Aaron Domej, Department of Ophthalmology, Duke University Medical Center, Durham, North Carolina, Email: aarodom@yahoo.com

**Received date:** December 07, 2021, Manuscript No: IPADO-22-13073; **Editor assigned date:** December 09, 2021, PreQC No. IPADO-22-13073 (PQ); **Reviewed date:** December 23, 2021, QC No. IPADO-22-13073; **Revised date:** December 27, 2021, Manuscript No. IPADO-22-13073 (R); **Published date:** January 10, 2022, DOI: 10.36648/2471-8513.8.1.010

**Citation:** Domej A (2022) Analysis of Autoimmune Retinopathy and age-related macular degeneration. J Autoimmune Disord Vol.8 No.1: 010

## Description

Retinal degeneration is one of the leading causes of visual loss in the world. In the majority of cases, the underlying cause is unknown. Some types of acquired retinopathies may be explained by an autoimmune involvement, since patients with retinopathy have circulating antibodies against retinal proteins, in particular, patients with retinopathy and extraocular tumor. These paraneoplastic retinopathies, including cancer-associated retinopathy (CAR), have been the most intensively studied group of autoimmune retinopathies, in which progressive symptoms of cone and rod dysfunction are present with systemic cancer but are in the absence of other neurological symptoms. This review focuses on the pathogenic role of anti-retinal autoantibodies associated with the CAR syndrome.

## Paraneoplastic and Autoimmune Retinopathies

The majority of patients with CAR have smallcell carcinoma of the lung although other malignant tumors have also been reported. Clinically, CAR is characterized by sudden and progressive visual loss, suggesting retinal dysfunction with ring scotoma, photopsia, attenuated retinal arterioles, and abnormalities of the a- and b-waves of the electroretinogram (ERG). These symptoms occur in the absence of eye inflammation or low inflammatory response in the eye. Antibodies against recoverin (so-called CAR antigen) were first reported in association with small cell carcinoma of the lung. The presence of these antibodies has been detected several months before diagnosis of cancer; therefore, anti-recoverin antibodies might have a diagnostic value for early detection of cancer. Pathology, when available, confirms the loss of retinal cells, which is in agreement with antibody specificity, e.g. the loss of cells in the photoreceptor cell layer and the presence of anti-recoverin antibodies. However, not all patients with CAR possess anti-recoverin antibodies. Autoantibodies to other retinal antigens detected by Western blotting have also been found, including a-enolase w3x, Heat Shock Cognate (HSC) protein 70, 40-kDa protein Da protein, PTB-like protein, neurofilament proteins w8x, and other retinal proteins. Importantly, autoantibodies against the ganglion cells of the retina, which share antigens with small cell carcinoma cells, were found in CAR patient's cerebrospinal fluid and immune

deposits in their retinas. Moreover, within the central nervous system, only the retina and optic nerve showed tissue damage with the specific loss of retinal ganglion cells and their processes. Thus, it is believed that circulating anti-retinal autoantibodies might contribute to retinal pathology.

The immune privilege mechanisms are in most cases effective in preventing inflammation, yet autoimmune retinopathies and inflammatory uveitis are observed in patients and can be induced in mouse models. In part this may be due to low levels of tolerance for antigens whose expression is restricted to the retina. Different mouse strains have varying degrees of susceptibility to autoimmune uveitis, and this susceptibility is related to levels of retinaspecific protein in the thymus. It remains to be seen if genetic variability in humans similarly affects tolerance to retinal antigens and pathogenesis of autoimmune retinopathies. In any case, it is likely that the mechanisms of ocular privilege are responsible for the impaired development of tolerance to retinal antigens. The triggers for autoimmune retinopathy are largely unknown, but antibodies to bacterial antigens have been associated with both AIR and AMD (see below). Infectious agents might provoke autoimmunity by activating antigen-specific immunity – which could be particularly dangerous if bacterial antigens mimic endogenous human antigens. The ability of infectious agents to provide a nonspecific second signal for autoimmune responses is known as the adjuvant effect, and this phenomenon has been examined in detail by Rose and colleagues. Pathogen-induced inflammatory responses may also disrupt the blood–retinal barrier or otherwise expose the normally sequestered retinal antigens to the immune system. Once retinal autoimmunity has been initiated, targeting of the retina leads to tissue damage and vision loss. Damage may occur due to immune complex deposition, complement activation, cytotoxic T cell activity and/or macrophage activity. The exact causes of damage may vary with the type of disorder. Autoimmunity has been linked to both AIR and AMD, though the pathogenic mechanisms and autoantigens may differ between the two disorders.

## Renal Oncocytoma Associated with Erythrocytosis

The eyes from the rats were enucleated, briefly immersed in 70% ethanol, and washed 2 times with cold DMEM containing

10% FBS and 2.5 mg/ml of fungizone. After the anterior chamber was removed, the retina was carefully dissected with fine forceps and cut into small explants (2x3 mm) with corneal scissors under a dissecting microscope. Two or 3 retinal explants were incubated at 37 C in a 6-well plate filled with 1.5 ml DMEM containing 2% FBS and 1.25 mg/ml fungizone. Serum was added at 1:30 dilution to the explants, which were incubated in vitro for different periods of time, depending on the test. The control retinal explants were treated with healthy subjects' sera without specificity for the retina or with rat MAb against recoverin Rec-1 (positive control), or were left untreated. The retinal explants were fixed with 4% paraformaldehyde in phosphate-saline buffer, pH 7.2, for 2 h, followed by treatment with 10%, 20%, and 30% of phosphate-buffered sucrose solution. Then, the retinal explants were embedded in an OCT medium and quickly frozen. Transverse sections (10 µm) were cut in a cryostat and placed on gelatin-coated slides. Serial sections obtained from the same piece of tissue were used to examine the localization of antibodies and apoptotic cells.

Rats were anaesthetized with a cocktail of ketamine/ xylazine/ acepromazine (1 mg/kg). The cornea was anaesthetized with 0.5% topical proparacaine hydrochloride before intravitreal injection. Five microliters of serum antibody was injected into the vitreous of one eye, and the second eye received normal serum or phosphatebuffered saline (PBS). The eyes were collected 24 h after injection. They were fixed with 4% paraformaldehyde in phosphate-saline buffer, pH 7.2, for 2 h, followed by treatment with 10%- (for 1 h), 20%- (for 2 h), and 30%-PBSesucrose solution overnight. Then, the whole eye was embedded in an OCT medium and quickly frozen. Transverse sections (10 µm) were cut in a cryostat and placed on gelatin-coated slides. Serial sections obtained from the same eye were used to examine the localization of antibodies and apoptotic cells. Our previous studies have shown that anti-α-enolase antibodies are cytotoxic to retinal E1A.NR3 cells and induce their apoptotic death.