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An Overview of Best Disease

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Abstract:- The German ophthalmologist Friedrich Best named Best vitelliform macular dystrophy (BVD), sometimes referred to as Best disease, in 1905 when he discovered a family with a history of early-onset macular degeneration. A Swedish study assessed the frequency of BVMD to be 2 in 10,000, whereas a Danish examination found 1.5 in 100,000 cases. If a parent with the condition has a kid with an unaffected spouse, there is a 50% chance that the child will inherit the condition from the parent. The syndrome is caused by a mutation in the VMD2 or BEST1 gene found at chromosome 11q12-q13. It is yet unclear how bestrophinopathies pathophysiologically operate. An ionic imbalance in the RPE milieu, which BEST1 gene mutations can bring on, can lead to impaired RPE functions. The best sickness is in five phases. There is currently no recognized treatment for the best sickness. One treatment option for CNV is VEFG injections, either used alone or in conjunction with photodynamic therapy (PDT). Anti-vascular endothelial growth factor (Anti-VEGF) treatments can prevent or reduce the creation of new blood vessels. This can postpone the onset of blindness and slow down the rate at which they leak.

Keywords:- *Best Vitelliform Macular Dystrophy, Electro-Oculography, Bestrophin, Choroidal Neovascularization, Photodynamic Treatment*

I. INTRODUCTION

The German ophthalmologist Friedrich Best named Best vitelliform macular dystrophy (BVD), sometimes referred to as Best disease, in 1905 when he discovered a family with a history of early-onset macular degeneration. Alternative names for BVD include vitelliform macular dystrophy 2 (Best disease, bestrophin), BEST, BEST1_HUMAN, BMD, and VMD. [1] The best disease is the second most common form of juvenile macular degeneration, usually presenting before the age of fifteen. It accounts for about 1% of all cases of macular degeneration. Even within the same family, there may be significant differences in the age at which symptoms manifest and the severity of central vision loss. According to Booij et al., the median age at which visual symptoms initially manifested was 33 years (range: 2-78). For individuals with BVD, a cumulative decline below 0.3 was linked to a 50% cumulative risk at age 66 and a 75% cumulative risk at age 74. Conversely, a visual acuity (VA) below 0.5 was linked to a 50% cumulative risk at age 55 and a 75% cumulative risk at age 66. Doctors refer to this disorder's yellowish macular lesions as vitelliform macular dystrophy, named for the shape of an egg yolk.[2] Age-related macular degeneration is the most common cause of blindness in Western-aged individuals, and the Best illness shares many characteristics. A dominantly inherited macular degeneration, BVD is characterized by a very variable expression. The gradual reabsorption of the yellow material from the atrophic area of the retinal pigment epithelium (RPE) frequently results in subretinal fibrosis. In the initial phases of the illness, the patient's vision is normal. As time passes, central visual acuity starts to deteriorate and metamorphopsia sets in. Peripheral vision and dark adaptation are within normal limits for the patients. An abnormal combination of electro-oculography (EOG) and standard full-field electroretinography (fERG) is one feature of this disorder. Macular flicker or multifocal electroretinograms (mfERGs) may, however, show reduced amplitudes in the central regions even in the early stages.[1,2,3]

II. PREVALENCE

The prevalence of BVMD in the US is unknown; just two studies have attempted to estimate it yet. A Swedish study assessed the frequency of BVMD to be 2 in 10,000, whereas a Danish examination found 1.5 in 100,000 cases. AVMD, also known as adult-onset foveomacular vitelliform dystrophy, is a kind of pattern dystrophy. The hallmarks of AVMD, which often manifests between the ages of 30 and 50, are symmetric, isolated, round, or oval vitelliform lesions of 1/3 to 1 disc diameter.[4] Some patients have metamorphopsia and impaired vision, while others have no symptoms at all. A normal electrocardiogram (EOG), smaller lesion size, slower disease progression, and age of onset are important characteristics in clinically differentiating AVMD from BVMD. Given the prevalence of late-onset cases, one could argue that age of onset should not be a requirement for BVMD. When it comes to BVMD, there is a wide range in the development of symptoms, and some individuals who have the disease-causing mutations never show any symptoms at all. In select instances, AVMD has been associated with mutations in peripherin/RDS and BEST1, and autosomal dominant inheritance of AVMD has also been demonstrated. It is arguable whether AVMD brought on by the BEST1 mutation qualifies as BVMD. As far as we are aware, no earlier studies on the frequency of AVMD have been published.[5,6]

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III. ETIOLOGY

The syndrome is caused by a mutation in the VMD2 or BEST1 gene, which is found at chromosome 11q12-q13. This gene largely codes for the transmembrane protein known as Betrofin 1, which is mostly located in the basolateral membrane of the RPE.[3] Betrofin has two distinct functions: it is both a pentameric anion channel and a regulator of intracellular Ca++ signaling. Bestrophin 1 has the potential to function as a bicarbonate channel in addition to a calcium-activated chloride channel. It is also essential for preserving the calcium homeostasis of the RPE. Consequently, bestrophin plays a crucial role in controlling the ionic environment of the subretinal area and the RPE. The ionic equilibrium of the RPE is essential for the retina and RPE to adhere.[7] The mutation causing Best Vitelliform Macular Dystrophy lowers the Arden ratio in both carriers and affected individuals. About 5% of patients with the BEST1 mutation will have macular findings that are normal or almost normal. The BEST1 mutation causes vitelliform macular dystrophy 2 (BVMD) (Phenotype MIM No. 153700), retinitis pigmentosa 50 (RP50)/concentric retinitis pigmentosa (Phenotype MIM No. 613194), autosomal dominant vitreoretinochoroidopathy (Phenotype MIM No. 193220), autosomal dominant micro cornea, rod-cone dystrophy, cataract, and posterior staphyloma 193220 (Phenotype MIM No. 193220), and adult-onset vitelliform macular dystrophy.[6,7]

IV. HOW THE DISEASE IS INHERITED

Mutations in the BEST1 (VMD2) gene are the cause of best disease. The illness is inherited in families through the autosomal dominant mode of inheritance. One copy of the normal gene and one copy of the Best disease gene are carried by an individual with this type of heredity. If a parent with the condition has a kid with an unaffected spouse, there is a 50% chance that the child will inherit the condition from the parent. The only partner who will pass on normal genes is the unaffected one. If a child lacks the matching gene, they cannot get the Best disease and infect their progeny. See a genetic counselor for additional information on inheritance, family planning, genetic testing, and related subjects.[3,8]

V. PATHOPHYSIOLOGY

It is yet unclear how bestrophinopathies pathophysiologically operate. As previously indicated, mutations in the BEST1 gene may produce an ionic imbalance in the RPE milieu, which can lead to impaired RPE activities. The lack of chloride current that characterizes the majority of BVMD mutations is typically caused by a dominant negative mechanism.[8] It has been demonstrated that patients with AVMD have either dominant negative (no chloride current) or haploinsufficiency (between 10 and 40 percent of the wild-type chloride current) mutations in the BEST1 gene. Patients with BEST1 mutations in AVMD and BVMD show variable expression and incomplete penetration. In contrast to BVMD, where there is no apparent association, BEST1 mutations appear to be associated with the symptoms of ADVIRC and MRCS syndrome. The previously discovered mutations affect splicing in both ADVIRC and MRCS, leading to in-frame duplications in ADVIRC and in-frame deletions in MRCS syndrome. Compound heterozygous for nonsense or missense mutations in the BEST1 gene might result in autosomal recessive dystrophinopathy (ARB), a null phenotype.

A. Vitelliform Material

The first hypothesis underlying BVMD and AVMD proposed that a disruption in ionic transport and fluid balance results in the accumulation of fluid and vitelliform material.[9] This would result in the development of unphagocytosed photoreceptor outer segments, toxic fluorophores, and toxic damage to the RPE and photoreceptors. It would also cause fluid to accumulate in the potential space between the RPE and photoreceptor cells. More recent studies indicate that theories other than the "classical" one might provide a more satisfactory explanation for the pathophysiology of bestrophinopathies. As the most notable clinical feature of BVMD and ARB, lipofuscin deposition has been suggested to be the basis for the pathogenesis of bestrophinopathies. Hyperspectral autofluorescence imaging (HAI) studies clearly show that these RPE fluorophores represent the premature dysfunction of the affected RPE, rather than having a role in the etiology of bestrophinopathies.[5,8,10]

B. Cholesterol Homeostasis

A disturbance in the regular regulation of cholesterol levels, which is essential for the appropriate growth and operation of the outer segments, is a characteristic of bestrophinopathies. Changes in cholesterol homeostasis in Best1 mutant retinae include:

- The RPE has higher levels of unsterified cholesterol.
- The esterified cholesterol is not well absorbed by the photoreceptor outer segments from Bruch's membrane.
- Retinal buildup of HNE adducts, a byproduct of lipid peroxidation, 4-hydroxy-2-nonenal.

Chronic inflammatory stimuli that are linked to decreased calcium signaling and fluid flow, as well as altered distribution of cholesterol esters and HNE-adducts at the photoreceptor layer, maybe the cause of the decrease in adhesive forces between RPE, NSR, and the interphotoreceptor matrix [10,11].

C. Retinal Pigment Epithelium-Photoreceptor Interface

Bentrophin deficiencies break the link between RPE and photoreceptors:

- In RPE cells with the Best1 mutation, apical microvilli retract;
- It seems that the normal bilayered extracellular sheath covering the cones is absent from Best1-mutant retinae, allowing the RPE to stick to the outer segments of the cones.[12]

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VI. ADULT ONSET VITELLIFORM MACULAR DYSTROPHY

In contrast to Best disease, the changes in adult-onset vitelliform macular dystrophy occur much later in life, do not progress in the same way, and affect less of the back of the eye. Between the ages of 30 and 50, vitelliform macular dystrophy frequently causes mild to moderate vision loss when it initially appears in adults. The visual alteration is usually unintentionally found during a routine eye checkup because it can be so mild. The best disease often has a worse prognosis for vision than adult-onset vitelliform dystrophy. Most of the time, the underlying etiology of adult-onset vitelliform macular dystrophy is still unknown. While PRPH2, IMPG1, and IMPG2 can occasionally have mistakes, BEST1 is the most often implicated gene.[13] The exact origin of adult-onset vitelliform macular dystrophy is unknown, and many affected individuals do not have any of these genes at fault. We are now unsure of the genetic transmission mechanism for vitelliform macular degeneration. Not everyone who inherits a faulty gene experiences symptoms, and not everyone with the condition has a family history of it. A foggy or distorted central vision is one of the symptoms. The condition progresses slowly, thus many patients might be able to maintain their vision long into old age.[14]

VII. THE FIVE STAGES OF THE BEST DISEASE

An optometrist or ophthalmologist can identify the five stages of Best disease by looking at the macula. There is no pain in your eyes when doing any of these actions.

- **Stage 1:** At this point, your macula seems healthy, but there hasn't been any noticeable change. Vision is usually unaltered, though a layer beyond the macula may experience slight changes.[6]
- **Step 2:** This phase is referred to as the vitrelliform phase. Right now, your macula can have a blister that looks like an egg yolk. Patients may not notice any effects on their vision at this point, or they may only notice slight changes, even if an optometrist or eye specialist can identify these changes. Typically, this developmental stage lasts from the age of three to fifteen.[8]
- **Step 3:** This phase is referred to as the pseudohypopyon phase. At this stage, you can peel off a portion of the layer behind your retina to show the yellow substance-caused blister that resembles an egg. A cyst forms beneath the retina as a result. Again, the degree of sight might not change significantly. This stage usually manifests itself during adolescence.[15]
- **Stage 4:** Known as the "vitelliruptive" stage, this stage takes place in stage four. A few retinal cells may be harmed as the blister lesion begins to burst. At this point, your impression of straight lines might start to change. You can have trouble reading small print, and straight lines might start to look wavy.
- **Stage 5:** The most advanced sickness reaches its final phase. This is the period of cell death. The yellowish-green culprit in the blister lesions begins to drain and disappear. The drawback is that it leaves scars and harms your retina. At this time, if your vision becomes more seriously affected, reading could become difficult.

It's possible that you won't experience all five phases and that your health will remain unchanged at any time. Choroidal neovascularization, or CNV, is an additional stage that may manifest in certain people. The eye is currently actively working to create new blood vessels in an attempt to repair the macula damage. Because of their acute sensitivity to leaking and bleeding, these recently formed blood vessels run the risk of developing scar tissue and further vision loss. Conversely, the great majority of people with Best illness do not have CNV. If you notice a sudden change in your vision, you should see an ophthalmologist right once to rule out CNV. Treating CNV requires early detection.[15,16]

A. Ophthalmological Evaluation

The primary evaluations concentrate on the field of vision and visual acuity. Patients may have mild center sensitivity deficiencies in their later visual fields, even while their peripheral visual fields appear normal. Jarc-Vidmar M et al. evaluated central visual function mapping in patients with Best's vitelliform dystrophy using microperimetry (MP) and autofluorescence. There was a high correlation between MP and the static perimetry indices (MS, MD, and CLV) (R=0.75, -0.76, -0.48). Microperimetry allowed for a highly sensitive topographic monitoring of retinal function.[17] The results showed a clear shift to the preferential retinal locus (PRL) in eyes with visual acuity of 0.2 or less, indicating the loss of central function, and central or pericentral fixation in the early stages. PRL aligned with the hyperfluorescent ring but was always outside of the core uniform hyperfluorescent area, according to autofluorescence imaging. Static perimetry demonstrated worse VA and more dense central scotomas, both of which were markers of the progression of BVD and were linked with MP results. Additional tests like fluorescein angiography, RPE autofluorescence, optical coherence tomography, full-field electroretinogram, and multifocal electroretinogram may be helpful for a more precise diagnosis of BVD. Even though a patient with Best disease initially exhibits no symptoms, an examination frequently reveals fundus lesions. When doctors initially see symptoms similar to those of other macular dystrophies, they may mistakenly diagnose patients with bilateral macular abnormalities. Cone or Stargardt dystrophies. Two possible problems related to vision include metamorphopsia and poor sharpness (blurring). These symptoms may worsen during the disease's atrophic stage. Due to the extended duration of normal visual acuity and the late onset of atrophy in the 50s, BVD is typically diagnosed late in life. The gold standard for diagnosing sickness is anomalies in electrooculograms, which are present even in asymptomatic patients and do not include any light-induced rise. Electromyography (EOG) measures electrical potentials across the retinal pigment epithelium (RPE) and, according to Deutmann (1971), reveals widespread retinal pigment epithelium failure.[18,19] The best disease decreases the Arden ratio, which is the ratio

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of the light peak to the dark trough. A light-to-dark ratio, or Arbour ratio, of greater than 1.8 is regarded as usual. The electrooculogram (EOG) exhibits an aberrant Arden ratio below 1, along with elevations at the outer retina-retinal pigment epithelium complex, retinal pigment epithelium, and retinal thickness during the intermediate clinical phases of Best disease.[18] Spectral-domain optical coherence tomography can now be used to identify deficiencies in the pigment epithelial layer and cone. In the later stages of the disease, when the retinal pigment epithelium is degenerating, it can be difficult to differentiate between different types of macular degeneration. In the early stages of BVD, fundus fluorescein angiography is obscured by vitelliform material; as the disease advances, hyperfluorescence with or without leakage is seen. Maruko et al. examined eight BVD patients, reporting the findings of indocyanine green angiography and contrasting them with fluorescein angiography and ophthalmoscopy results. In all eight eyes, ophthalmoscopy and fluorescein angiography showed several hyperfluorescent patches in the periphery and mid-periphery, respectively, in areas free of any obvious abnormalities. The volume and distribution of lipofuscin have been monitored by the fundus autofluorescence hyperfluorescence, which has been seen in extensive BVD lesions that have accumulated in the retinal pigment epithelium (RPE)[20,21].

B. Treatment

There is currently no recognized treatment for the best sickness. One treatment option for CNV is VEFG injections, either used alone or in conjunction with photodynamic therapy (PDT). In certain people with Best disease, there may be an increase in new blood vessels on or beneath the macula, a condition referred to medically as choroidal neovascularization (CNV). There is a treatment for CNV.[22] An injection of a medication that blocks VEFG can treat new blood vessels and prevent further visual loss. While there is no certainty that vision will return with the regeneration of new blood vessels, it does assist prevent further macula and visual impairment.[23]

Anti-vascular endothelial growth factor (Anti-VEGF) treatments can prevent or reduce the creation of new blood vessels. This can postpone the onset of blindness and slow down the rate at which they leak. Even while the NHS no longer regularly gives anti-VEFGs to patients with CNV linked to vitelliform macular dystrophy, your ophthalmologist can evaluate your unique needs and suggest the best course of action. Potential gene therapy treatments for several inherited macular dystrophies are being researched. Gene therapy aims to insert a healthy gene in place of the damaged one. The genetic material is transferred by injecting a harmless virus that contains normal genes into the retina. Ideally, the impaired retinal cells will regain their normal function, potentially mitigating or even reversing the damage. Gene therapy is yet another emerging treatment option. There is currently no gene therapy treatment available to treat Best disease.[24] There is presently just one gene therapy treatment option available for a particular type of hereditary retinal degeneration caused by mutations in the RPE65 gene. It is exciting that a gene therapy treatment for Best Disease and other inherited retinal eye illnesses is being developed that is as successful. There is no evidence that dietary modifications slow down the progression of Best disease. On the other hand, eating a diet high in fresh fruits and vegetables is necessary to keep healthy eyes. Given that quitting smoking may delay the onset of Best disease and other forms of macular degeneration, it makes sense.[25]

VIII. CONCLUSION

Best vitelliform macular dystrophy (BVD), sometimes called Best disease, is an uncommon hereditary disorder that damages the macula and may result in blindness. It is inherited autosomally dominantly and is brought on by mutations in the BEST1 gene. There are several stages of the disease, and symptoms usually start to show in early adulthood or youth. It is thought that just 1% of all cases of macular degeneration are caused by BVD, indicating a low prevalence of the disorder. The significance of primary evaluations with an emphasis on visual field and acuity in patients with Best's vitelliform dystrophy. The assessment of central visual function mapping is aided by the application of microperimetry and autofluorescence, which offers insightful information for disease diagnosis and tracking. Best disease treatment possibilities include gene therapy research, anti-VEGF medications for choroidal neovascularization, and lifestyle modifications like stopping smoking and adhering to a healthy diet to potentially reduce the illness's progression.

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