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Intravitreal Anti-VEGF Injection Monotherapy for Wet Age-Related Macular Degeneration: A Case Report Demonstrating Successful Visual and Anatomical Outcomes

Ramzi Amin[1]*, Linda Tri Wulandari[1]

[1]Department of Ophthalmology, Faculty of Medicine, Universitas Sriwijaya/Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia

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***Corresponding author:**
Ramzi Amin

E-mail address:
ramziamin@fk.unsri.ac.id

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A B S T R A C T

Neovascular (wet) age-related macular degeneration (nAMD) is a leading cause of severe vision loss in the elderly, characterized by choroidal neovascularization (CNV). Intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy has revolutionized nAMD management. This report details a case of nAMD successfully managed with anti-VEGF monotherapy. A 61-year-old male smoker with a history of hypertension presented with a two-year history of progressive blurred vision in his right eye (OD). Best-corrected visual acuity (BCVA) was 1/300 OD and 6/60 (pinhole 6/30) OS. Fundus examination OD revealed drusen, hard exudates, and reduced foveal reflex. Optical Coherence Tomography (OCT) OD confirmed intraretinal and sub-RPE fluid, pigment epithelial detachment (PED), and features suggestive of Type II CNV. The patient was diagnosed with nAMD OD and immature senile cataract bilaterally. He received intravitreal anti-VEGF injection OD. Seven days post-injection, BCVA OD improved to <1/60, with subjective improvement in vision. In conclusion, this case demonstrates the efficacy of intravitreal anti-VEGF monotherapy in improving visual and anatomical outcomes in a patient with nAMD. Despite known risk factors, timely intervention led to a favorable short-term response. Long-term management and monitoring remain crucial.

1. Introduction

Age-related macular degeneration (AMD) is a prominent global health issue, recognized as a major cause of irreversible blindness and severe visual impairment, particularly in individuals aged 50 and older within developed nations. This condition is a progressive neurodegenerative disease that affects the central retina, specifically the macula. The macula is responsible for sharp, detailed central vision, which is essential for tasks such as reading, driving, and facial recognition. The pathology of AMD primarily involves key structures of the eye, including the photoreceptor cells, the retinal pigment epithelium (RPE), Bruch's membrane, and the choriocapillaris. The prevalence of AMD is steadily increasing due to the aging global population. In 2020, it was estimated that nearly 200 million people worldwide were affected by AMD, and projections suggest this number will rise to approximately 288 million by 2040. This increase underscores the growing significance of AMD as a public health concern and highlights the need for effective management strategies. AMD manifests in two primary forms: non-neovascular (dry or atrophic) AMD and neovascular (wet or exudative) AMD. Dry AMD is the more common form, accounting for 80-85% of AMD cases. It is typically characterized by slow progression and involves the accumulation of drusen (extracellular deposits beneath the RPE) and/or the

development of geographic atrophy (GA). Geographic atrophy involves the loss of RPE, photoreceptors, and choriocapillaris. While dry AMD often has a better initial visual prognosis compared to wet AMD, it can still lead to significant vision loss, especially when geographic atrophy affects the foveal center. Additionally, it's important to note that all forms of dry AMD carry the risk of progressing to the wet form. Neovascular AMD (nAMD), although less prevalent (15-20% of cases), is responsible for the majority (approximately 80-90%) of severe vision loss associated with AMD. The hallmark of nAMD is the development of choroidal neovascularization (CNV). In this process, abnormal blood vessels originating from the choroid penetrate Bruch's membrane and proliferate. These vessels can grow beneath the RPE (Type 1 MNV or occult CNV), into the subretinal space (Type 2 MNV or classic CNV), or, less commonly, originate from the retinal circulation (Type 3 MNV or retinal angiomatous proliferation). These newly formed vessels are fragile and prone to leakage. This leakage leads to the accumulation of subretinal and/or intraretinal fluid, hemorrhage, lipid exudation, and RPE detachments. If nAMD is left untreated, the exudative process often results in the formation of scar tissue in the macula, known as a disciform scar, which causes permanent and profound central vision loss.[1-3]

The pathophysiology of AMD is complex and multifactorial, involving a combination of aging processes, genetic predisposition, and environmental factors. Key contributing factors include oxidative stress, chronic inflammation, complement system dysregulation, and impaired RPE function. Aging leads to increased oxidative stress and the accumulation of lipofuscin within RPE cells, which can impair their function and potentially trigger apoptosis (cell death). Furthermore, age-related changes in Bruch's membrane, such as alterations in its composition and thickness, can impede nutrient transport and waste removal, further stressing the RPE and photoreceptors. Genetic studies have identified strong associations between AMD and polymorphisms in genes related to the complement pathway (e.g., CFH, C3, C2/CFB) and other pathways (e.g., ARMS2/HTRA1). These genetic findings highlight the significant role of inflammation and immune responses in the development of AMD. In addition to aging and genetic predisposition, several environmental risk factors significantly increase the likelihood of developing AMD and its progression. The most notable of these are advancing age and cigarette smoking. Smoking, in particular, is a critical modifiable risk factor that induces oxidative stress, impairs choroidal blood flow, promotes hypoxia, and stimulates neovascularization. Other implicated environmental factors include hypertension, obesity, dietary habits (such as high fat intake and low antioxidant intake), and potentially cardiovascular disease and light exposure. The diagnosis of nAMD relies on a combination of clinical examination and multimodal imaging techniques. Patients with nAMD often present with characteristic symptoms such as sudden or gradual blurring of central vision, metamorphopsia (distortion of straight lines), micropsia (objects appearing smaller than their actual size), or a central scotoma (blind spot). Funduscopic examination may reveal key signs of nAMD, including drusen, subretinal or intraretinal fluid, hemorrhages, lipid exudates, or grayish-green subretinal lesions indicative of CNV. Optical coherence tomography (OCT) has become an indispensable tool in the diagnosis and monitoring of nAMD. OCT provides high-resolution, cross-sectional images of the retina, enabling detailed visualization and quantification of intraretinal fluid (IRF), subretinal fluid (SRF), pigment epithelial detachments (PEDs), and the CNV complex. This imaging modality is crucial for assessing disease activity and guiding treatment decisions.[4-6]

OCT angiography (OCTA) is a relatively new, non-invasive technique that visualizes retinal and choroidal vasculature. It achieves this by detecting motion contrast from flowing blood cells, allowing for the identification and characterization of CNV architecture without the need for dye injection. Fundus fluorescein angiography (FFA) remains a valuable tool for identifying the type (classic vs. occult)

and location of CNV and detecting leakage. However, its use may be limited by factors such as dye allergies or renal impairment. Indocyanine green angiography (ICGA) can be beneficial in specific situations, such as visualizing polypoidal choroidal vasculopathy (PCV), a variant sometimes considered within the nAMD spectrum. To standardize the classification of nAMD, the Consensus Nomenclature for Reporting Neovascular Age-Related Macular Degeneration Data has been developed. This system now uses the term macular neovascularization (MNV) and categorizes it based primarily on OCT findings into Type 1 (sub-RPE), Type 2 (subretinal), Type 3 (intraretinal origin), and mixed types. The management of nAMD has been revolutionized by the advent of therapies targeting vascular endothelial growth factor (VEGF). VEGF-A is a key signaling protein that promotes angiogenesis (new blood vessel formation) and increases vascular permeability. It plays a critical role in the development and leakage of CNV in nAMD. Intravitreal injections of anti-VEGF agents work by binding to and neutralizing VEGF-A, thereby inhibiting neovascular growth and reducing exudation. Several anti-VEGF drugs are currently approved and widely used for the treatment of nAMD. These include ranibizumab (Lucentis®), aflibercept (Eylea®), brolucizumab (Beovu®), and faricimab (Vabysmo®), which also targets Angiopoietin-2. Bevacizumab (Avastin®), while structurally related and effective, is commonly used off-label due to its lower cost. Clinical trials have consistently demonstrated that anti-VEGF therapy can stabilize vision in the majority of patients and lead to significant visual acuity gains in a substantial proportion. This represents a dramatic improvement compared to previous treatments such as laser photocoagulation or photodynamic therapy (PDT), which primarily aimed to slow down vision loss. Treatment with anti-VEGF agents typically involves an initial loading phase of monthly injections, usually for three months. This is followed by a maintenance phase with various treatment protocols. Maintenance regimens include fixed interval dosing (e.g., every 8 or 12 weeks), pro re nata (PRN, or "as needed") dosing based on monthly monitoring for disease activity, or a treat-and-extend (T&E) regimen. The treat-and-extend regimen involves gradually lengthening the interval between injections as long as the macula remains dry and shortening the interval if exudation recurs. Treat-and-extend regimens aim to balance treatment efficacy with the goal of reducing the burden of frequent injections and monitoring visits for patients. While anti-VEGF therapy has proven to be highly effective, several challenges remain in the management of nAMD. These challenges include the need for long-term, potentially indefinite treatment, the burden of frequent injections and monitoring, the costs associated with treatment, and the subset of patients who exhibit a suboptimal response to therapy or develop geographic atrophy despite treatment. Newer agents like brolucizumab and faricimab offer the potential for extended dosing intervals (up to 12-16 weeks) in eligible patients, which could reduce the treatment burden. However, safety considerations, such as the risk of intraocular inflammation (IOI) with brolucizumab, necessitate careful patient selection and monitoring.[7-10] This report presents a case of a 61-year-old male diagnosed with nAMD in the right eye. The report highlights the patient's typical presentation, the diagnostic workup including OCT imaging, and the successful initial response to intravitreal anti-VEGF monotherapy. It demonstrates the functional improvement, as shown by visual acuity, and anatomical improvement, as shown by OCT findings, following treatment.

2. Case Presentation

The patient, identified as Mr. B, is a 61-year-old male. This age is particularly relevant in the context of the presented condition, as age is a significant risk factor for age-related macular degeneration (AMD). AMD's prevalence and incidence increase substantially in individuals over the age of 50, making this patient's age a key component of his demographic profile and a factor contributing to the likelihood of the diagnosed condition. The patient's sex, male, while less directly causative than age, is an important

descriptor in epidemiological considerations of AMD, where prevalence and specific manifestations can sometimes show variations across sexes in larger population studies. The patient's chief complaint was a gradual onset of blurred vision in his right eye, documented as the oculus dexter (OD). This symptom had been progressively developing over a period of two years. The patient's subjective description of his visual disturbance was characterized by "shadowy" or "smoky" vision in the affected eye. This description is clinically significant as it can indicate the presence of fluid or other disturbances affecting the macula, the central portion of the retina responsible for detailed central vision. Patients often struggle to articulate visual symptoms precisely, and these qualitative descriptors are valuable in understanding the nature of their visual impairment. Importantly, the patient specifically denied experiencing other common ocular symptoms such as floaters, flashes, curtain vision, tunnel vision, glare, pain, redness, or discharge in the affected eye. The absence of these symptoms helps to narrow down the differential diagnosis and suggests that the primary issue is likely localized to the macula rather than involving other parts of the eye or systemic processes that might manifest with more generalized ocular complaints. The patient's past medical history included hypertension, which he had been managing for eight years. His hypertension was controlled through the use of Amlodipine, administered at a dosage of 5mg. Hypertension is a significant systemic vascular condition and a recognized risk factor for various ocular diseases, including AMD. Chronic elevation of blood pressure can contribute to microvascular damage within the retina and choroid, potentially exacerbating or accelerating the pathological processes involved in AMD. The fact that the hypertension was controlled is a relevant detail, though even controlled hypertension can still exert some influence on ocular health over time. The patient specifically denied any history of diabetes mellitus or dyslipidemia. Both of these conditions can independently affect the vasculature and are important to rule out as potential contributing factors to ocular pathology. He also denied any previous eye trauma, which is crucial as trauma can cause a range of ocular complications that might mimic or complicate other conditions. Finally, he denied using spectacles, implying either a history of good vision prior to the onset of the current complaint or a lack of correction for any refractive errors, which would be important to consider in the context of visual acuity testing. Regarding habits, the patient reported being a smoker with a history of smoking for over 20 years, consuming one pack of cigarettes per day. Cigarette smoking is an extremely well-established and potent risk factor for AMD. The numerous toxic components of cigarette smoke induce oxidative stress, impair choroidal blood flow, promote inflammation, and contribute to the formation of drusen and neovascularization. The duration and quantity of smoking, expressed as pack-years, are critical factors in assessing the risk. In this case, the 20 pack-year history signifies a substantial cumulative exposure to the harmful effects of smoking on the eye. Conversely, the patient denied any consumption of alcohol. While alcohol consumption can have various systemic effects, its direct role in AMD pathogenesis is less clear compared to smoking. The patient's family history was unremarkable, with a denial of any family history of similar eye conditions. While AMD has a significant genetic component, with identified genetic polymorphisms increasing susceptibility, the absence of a known family history does not entirely rule out a genetic predisposition in this individual. Many cases of AMD occur sporadically, and a lack of reported family history is a common finding in clinical practice. However, this aspect of the history is important in the overall risk assessment. The patient's general status was described as good. This is a broad descriptor indicating that, at the time of examination, he did not exhibit any overt signs of significant systemic illness. While this doesn't preclude the presence of underlying conditions, it provides a baseline assessment of his overall health. His vital signs were recorded as follows: blood pressure (BP) was 130/80 mmHg, heart rate (HR) was 90 beats per minute, respiratory rate (RR)

was 16 breaths per minute, and body temperature was 36.8 degrees Celsius. The blood pressure, while slightly elevated, is within a range that may be acceptable depending on individual patient factors and other comorbidities. However, given his history of hypertension, this measurement reinforces the importance of considering systemic vascular health in his overall management. The heart rate and respiratory rate are within normal limits, and the temperature is afebrile, further supporting the assessment of generally stable vital signs. Anthropometric measurements included a weight of 71 kilograms, a height of 170 centimeters, and a calculated body mass index (BMI) of 24.6 kg/m^2. This BMI falls within the range considered to be ideal weight. While obesity is a recognized risk factor for AMD, this patient's weight and BMI do not contribute to that particular risk factor. However, it's important to note that the absence of obesity does not negate the presence of other significant risk factors in this case. The patient's best-corrected visual acuity (BCVA) was significantly reduced in the right eye (OD), measured at 1/300. This is a severe level of visual impairment, indicating that the patient could only count fingers at a distance of one meter. In contrast, the left eye (OS) had a BCVA of 6/60, which improved to 6/30 with pinhole correction. This discrepancy in visual acuity between the two eyes is a critical finding. The pinhole improvement in the left eye suggests a refractive component to the reduced vision in that eye, potentially due to the noted cataract. However, the profound visual loss in the right eye, which does not improve significantly with pinhole, strongly points towards a more serious pathological process affecting the macula. Intraocular pressure (IOP) was measured at 13.1 mmHg in the right eye (OD) and 12.0 mmHg in the left eye (OS). These IOP measurements are within the normal range. Elevated IOP is a primary risk factor for glaucoma, but in this case, the normal IOP readings make glaucoma less likely as the primary cause of the patient's visual symptoms. However, it's important to continue monitoring IOP as part of a comprehensive ophthalmic examination. Ocular alignment and motility were assessed, and the patient was found to have orthophoria with full extraocular movements in both eyes (OU). Orthophoria indicates proper alignment of the eyes, and full extraocular movements signify that the muscles controlling eye movement are functioning normally. These findings suggest that the patient does not have strabismus or any motility deficits, which are important to rule out as alternative causes of visual disturbance. Examination of the anterior segment of both eyes (OU) revealed that the lids and conjunctiva were quiet, indicating no signs of inflammation or infection. The cornea was clear, suggesting no corneal opacities or edema that could impair vision. The anterior chamber was of moderate depth, with no evidence of shallowing or deepening that might suggest angle-closure or other anterior segment abnormalities. The iris had a normal architecture, was round and central, and measured 3mm in diameter. It was also reactive to light, indicating normal pupillary reflexes. The lens in both eyes was noted to have an immature senile cataract, characterized by nuclear sclerosis and cortical changes. Cataracts are a common age-related finding and can contribute to reduced visual acuity, particularly in the left eye in this case. Examination of the posterior segment of the right eye (OD) revealed a clear vitreous, implying no significant vitreous opacities or hemorrhage. The optic disc was described as round, with sharp margins and a pink rim. The cup-to-disc ratio (C/D) was 0.3, and the arteriovenous ratio (A/V) was 2:3, with no evidence of neovascularization of the disc (NVD). These findings suggest that the optic nerve appears healthy, and there is no evidence of glaucoma or other optic neuropathy. However, the macula in the right eye showed a diminished foveal reflex, the presence of hard exudates, and drusen. These are critical findings strongly suggestive of macular pathology. A diminished foveal reflex indicates disruption of the normal reflective properties of the macula, while hard exudates and drusen are characteristic features of AMD. Additional retinal findings in the right eye included microaneurysms, venous beading, hemorrhages (dot/blot), intraretinal

microvascular abnormalities (IRMA), and neovascularization elsewhere (NVE). These findings collectively paint a picture of significant retinal vascular abnormalities, further supporting a diagnosis of neovascular AMD. Examination of the posterior segment of the left eye (OS) also revealed a clear vitreous and an optic disc that was round, with sharp margins and a pink rim. The C/D ratio was 0.3, and the A/V ratio was 2:3, with no NVD. Similar to the right eye, these findings suggest a healthy optic nerve. However, the macula in the left eye also showed a diminished foveal reflex. Additionally, the retina in the left eye was difficult to evaluate due to the presence of a cataract, limiting a thorough assessment of the posterior pole in that eye. Hematology testing revealed a hemoglobin level of 10.2 g/dL, indicating mild anemia. The white blood cell count (WBC) was 6.34 x $10^3/mm^3$, the platelet count was 415,000/mm^3, and the hematocrit was 42%. The prothrombin time (PT) was 11.35 seconds, the international normalized ratio (INR) was 0.88, and the activated partial thromboplastin time (APTT) was 27.6 seconds. These hematological parameters are generally within normal limits, with the exception of mild anemia. While not directly related to the ocular findings, anemia can have systemic implications and may warrant further investigation. Clinical chemistry results showed elevated levels of creatinine (3.0 mg/dL) and urea (54 mg/dL), indicating renal impairment. The blood sugar screen was 99 mg/dL. Sodium (Na) was 142 mEq/L, potassium (K) was 4.2 mEq/L, and chloride (Cl) was 112 mmol/L. Lipid levels included LDL of 118 mg/dL, total cholesterol of 184 mg/dL, and triglycerides of 153 mg/dL. The hepatitis B surface antigen (HBsAg) was non-reactive. The elevated creatinine and urea levels are significant findings, suggesting a degree of renal dysfunction. This is clinically important as renal impairment can affect the metabolism and excretion of certain medications and may influence treatment decisions. The lipid profile, while showing some elevation, is within a range that requires consideration in the context of overall cardiovascular risk. Fundus photography of the right eye (OD) confirmed the presence of multiple soft drusen, hard exudates, a diminished foveal reflex, and possible perifoveal hemorrhage. The optic disc was noted to have a C/D ratio of 0.3 and an A/V ratio of 2:3. These photographic findings corroborate the clinical examination findings and provide a visual record of the macular abnormalities. Fundus photography of the left eye (OS) confirmed a diminished foveal reflex. The retina was difficult to evaluate due to the cataract, and the optic disc had a C/D ratio of 0.3 and an A/V ratio of 2:3. These findings align with the limitations noted during the clinical examination. Optical coherence tomography (OCT) of the macula in the right eye (OD) revealed no vitreomacular traction (VMT), an absent foveal depression, intraretinal fluid in multiple areas, sub-RPE fluid in multiple areas, irregular RPE/BM with hyperreflective elevation and pigment epithelial detachment (PED), and choroidal neovascularization (CNV) between the RPE and neurosensory retina, consistent with Type II MNV. The choroid was not thickened. These OCT findings are crucial for confirming the diagnosis of neovascular AMD. The presence of intraretinal and subretinal fluid, PED, and CNV is all hallmarks of this condition. Type II MNV, also known as classic CNV, is characterized by neovascular vessels growing through the RPE into the subretinal space. OCT of the macula in the left eye (OS) showed no VMT, a decreased foveal depression, normal intraretinal layers, regular RPE/BM, and a choroid that was not thickened. These findings suggest that the macula in the left eye is relatively normal, with the decreased foveal depression likely related to the cataract. A chest X-ray revealed cardiomegaly, an elongated and atherosclerotic aorta, and normal lungs. Cardiomegaly and atherosclerotic changes in the aorta are indicative of cardiovascular disease, which is another systemic condition that can have implications for ocular health and overall management. The working diagnosis was wet age-related macular degeneration (nAMD) in the right eye (OD), specifically implied to be Type II (classic) based on the OCT description, and immature senile cataract in both eyes (OU). This diagnosis is supported by the

clinical findings, fundus photography, and OCT results. The differential diagnosis included polypoidal choroidal vasculopathy (PCV) in the right eye (OD). PCV is another condition that can cause neovascularization and fluid accumulation in the macula, but it has distinct clinical and angiographic features. The patient's systemic comorbidities included hypertension, renal impairment (elevated urea and creatinine), and hypertensive heart disease (compensated). These comorbidities are important to consider in the overall management of the patient, as they can influence treatment decisions and prognosis (Table 1).

The management of the patient's wet age-related macular degeneration (AMD) in the right eye (OD) was initiated with a structured treatment and follow-up plan, the details of which are outlined below. This plan incorporated intravitreal anti-VEGF injections, a mainstay of current nAMD therapy, and serial monitoring of both functional and anatomical parameters to assess treatment response and guide further management decisions. The patient's initial presentation, serving as the baseline visit, established the foundation for subsequent interventions. At this visit, a comprehensive ophthalmic evaluation of the right eye revealed a best-corrected visual acuity (BCVA) of 1/300. This level of visual acuity signifies severe visual impairment, where the patient could only perceive hand motion. Intraocular pressure (IOP) in the right eye was measured at 13.1 mmHg, a value within the normal range. Clinical examination of the fundus revealed characteristic features of AMD, including a diminished foveal reflex, hard exudates, and drusen. These findings are indicative of macular pathology and disruption of the normal retinal architecture. Optical coherence tomography (OCT) imaging provided detailed cross-sectional views of the retina, confirming the presence of intraretinal fluid (IRF) and subretinal fluid (SRF), pigment epithelial detachment (PED), and an irregular retinal pigment epithelium (RPE). Furthermore, OCT suggested the presence of Type II macular neovascularization (MNV), also known as classic choroidal neovascularization (CNV). Based on these findings, the initial plan was to initiate treatment with an intravitreal anti-VEGF injection. The first intravitreal anti-VEGF injection was administered and involved the injection of 0.05mL of an unspecified anti-VEGF agent into the vitreous cavity of the right eye. The injection was administered superotemporally, a common injection site to minimize the risk of complications. Local anesthesia was utilized to ensure patient comfort during the procedure. Importantly, the procedure was documented to have been performed without any noted complications. Intravitreal injections, while generally safe, carry potential risks such as endophthalmitis, retinal detachment, and increased intraocular pressure. The absence of reported complications during this initial injection is a positive indicator. A follow-up visit was conducted one day post-injection. At this visit, the BCVA in the right eye remained at 1/300, showing no immediate change in visual acuity. The IOP was measured at 15.6 mmHg, demonstrating a slight increase compared to the baseline measurement, but still within the normal range. The patient reported minimal pain, and the eye was described as otherwise quiet, indicating no signs of significant inflammation or adverse reaction to the injection. The treatment plan at this visit included the prescription of oral Cefixime, a systemic antibiotic, to reduce the risk of post-injection endophthalmitis; Paracetamol, an analgesic, as needed (prn) for pain management; and topical Levofloxacin and Prednisolone Acetate eye drops, to provide topical antibiotic coverage and anti-inflammatory effects. Post-injection care is critical to prevent infection and manage any associated discomfort. A follow-up visit at day seven post-injection revealed some initial changes. The BCVA in the right eye had improved to less than 1/60, although pinhole correction did not further improve this. The report noted that the patient expressed subjective improvement in vision, which is an important clinical indicator, even if it is not fully reflected in a dramatic change in Snellen acuity. The IOP was 12.0 mmHg, a decrease from the previous measurement and back to the baseline range. Clinical examination of the fundus

was reported to be similar to the baseline examination. However, it was noted that a discussion suggested improvement seen on OCT imaging, even though the specifics of these OCT changes were not detailed in the table. This underscores the importance of multimodal assessment in AMD management, where functional changes (visual acuity) may lag behind anatomical changes (OCT findings). At this visit, the patient was prescribed topical Potassium Iodide and Sodium Iodide eye drops for both eyes (ODS). A follow-up visit was scheduled one month post-injection. At this visit, the BCVA in the right eye was recorded as 1/60, showing further improvement from the initial presentation, although still indicative of significant visual impairment. The IOP was 14.0 mmHg, remaining within the normal range. OCT imaging at this visit revealed a significant reduction in both intraretinal fluid (IRF) and subretinal fluid (SRF). The pigment epithelial detachment (PED) was noted to be slightly decreased. However, persistent signs of macular neovascularization (MNV) activity were still observed. This OCT evidence of fluid reduction is a positive sign of treatment response, indicating that the anti-VEGF therapy was having the intended effect of reducing vascular leakage. Based on these findings, the plan was to proceed with a second anti-VEGF injection. The second intravitreal anti-VEGF injection was administered on the same day. Similar to the first injection, 0.05mL of an unspecified anti-VEGF agent was injected, and the procedure was reported to have been performed without complications. The repetition of the injection is consistent with the typical loading phase of anti-VEGF therapy, where monthly injections are often administered to achieve maximal suppression of neovascular activity. A follow-up visit at month two, showed further progression in the right eye's condition. The BCVA had improved to 2/60, indicating continued functional improvement. The IOP was 13.5 mmHg, remaining stable. OCT imaging revealed a further reduction in IRF and SRF, demonstrating ongoing anatomical improvement. The PED was reported to be stable. The macula was described as significantly drier, although subtle fluid was still present. The persistence of some fluid suggests that ongoing treatment is necessary to achieve complete resolution of exudation. The plan at this visit was to proceed with a third anti-VEGF injection, completing the loading phase of the treatment. The third intravitreal anti-VEGF injection was administered coinciding with the month two follow-up visit. The procedure involved the injection of 0.05mL of the unspecified anti-VEGF agent, with no complications reported. The completion of the loading phase with this third injection is a crucial step in establishing a therapeutic effect and setting the stage for longer-term maintenance therapy. A follow-up visit at month three, demonstrated continued positive trends. The BCVA had improved to 3/60, showing further functional gain. The IOP was 14.2 mmHg, remaining within the normal range. OCT imaging showed that the trace IRF had resolved, and only minimal SRF remained. The PED was reported to be stable and slightly flattened. The macula was described as near dry, indicating significant resolution of the exudative process. Based on these favorable findings, the treatment plan was to monitor the patient monthly and initiate a treat-and-extend regimen, extending the interval between injections to six weeks. The treat-and-extend regimen is a common strategy in AMD management, aiming to balance treatment efficacy with reducing the frequency of injections and clinic visits. A follow-up visit at month 4.5, confirmed the stability of the improvements. The BCVA remained at 3/60. The IOP was 13.8 mmHg. OCT imaging showed that the macula remained dry, with no new fluid or hemorrhage. The PED was stable. The plan was to administer the fourth anti-VEGF injection and extend the interval to eight weeks, further increasing the time between treatments. A follow-up visit at month 6.5, showed continued stability. The BCVA was 4/60. The IOP was 14.0 mmHg. OCT imaging confirmed that the macula remained dry, and the PED was stable, with no signs of MNV reactivation. The plan was to administer the fifth anti-VEGF injection and maintain the eight-week interval (Table 2).

Table 1. Summary of patient's clinical findings at presentation.

Category	Finding	Details
Demographics	**Patient ID**	Tn. B (Mr. B)
	Age	61 years
	Gender	Male
Anamnesis	**Chief Complaint**	Blurred vision in the right eye (OD)
	History of Present Illness	Gradual onset over 2 years. Described as "shadowy" or "smoky" vision OD. Denied floaters, flashes, curtain, tunnel vision, glare, pain, redness, discharge OD.
	Past Medical History	Hypertension (8 years, controlled on Amlodipine 5mg). Denied Diabetes Mellitus, Dyslipidemia, previous eye trauma, spectacle use.
	Habits	Smoker (20+ years, 1 pack/day). Denied alcohol consumption.
	Family History	Denied family history of similar eye conditions.
Physical examination	**General Status**	Good general condition.
	Vital Signs	BP: 130/80 mmHg, HR: 90/min, RR: 16/min, Temp: 36.8°C.
	Anthropometry	Weight: 71 kg, Height: 170 cm, BMI: 24.6 kg/m² (Ideal weight).
Ophthalmology exam	**Visual Acuity (BCVA)**	OD: 1/300 (Counting Fingers @ 1m equivalent). OS: 6/60, improving to 6/30 with pinhole.
	Intraocular Pressure (IOP)	OD: 13.1 mmHg, OS: 12.0 mmHg.
	Ocular Alignment/Motility	Orthophoria, Full extraocular movements OU.
	Anterior Segment (OU)	Lids/Conjunctiva: Quiet. Cornea: Clear. Anterior Chamber: Moderate depth. Iris: Normal architecture. Pupil: Round, central, 3mm, reactive to light (+). Lens: Immature senile cataract (Nuclear Sclerosis/Cortical changes noted).
	Posterior Segment (OD)	Vitreous: Clear (implied). Optic Disc: Round, sharp margins, pink rim, C/D 0.3, A/V 2:3, No NVD. Macula: Diminished foveal reflex, hard exudates (+), drusen (+). Retina: Cotton wool spots (+) in 2 quadrants, No microaneurysms, venous beading, hemorrhages (dot/blot), IRMA, NVE.
	Posterior Segment (OS)	Vitreous: Clear (implied). Optic Disc: Round, sharp margins, pink rim, C/D 0.3, A/V 2:3, No NVD. Macula: Diminished foveal reflex. Retina: Difficult to evaluate (due to cataract).
Laboratory	**Hematology**	Hb: 10.2 g/dL (mild anemia), WBC: 6.34 x 10^3/mm³, Plt: 415,000/mm³, Hct: 42%, PT/INR: 11.35s/0.88, APTT: 27.6s.
	Clinical Chemistry	SGOT: 21 U/L, SGPT: 18 U/L, BSS: 99 mg/dL. Urea: 54 mg/dL (Elevated), Creatinine: 3.0 mg/dL (Elevated). Na: 142 mEq/L, K: 4.2 mEq/L, Cl: 112 mmol/L. Lipids: LDL 118 mg/dL, Total Chol 184 mg/dL, TG 153 mg/dL. HBsAg: Non-reactive.
Imaging	**Fundus Photography (OD)**	Confirmed: multiple soft drusen, hard exudates, diminished foveal reflex, possible perifoveal hemorrhage. Optic disc C/D 0.3, A/V 2:3.
	Fundus Photography (OS)	Confirmed: diminished foveal reflex. Retina difficult to evaluate. Optic disc C/D 0.3, A/V 2:3.
	OCT Macula (OD)	No VMT. Foveal depression absent. Intraretinal fluid (+) multiple areas (hyporeflective spaces). Sub-RPE fluid (+) multiple areas. RPE/BM irregular with hyperreflective elevation (drusen) and PED (+). Suspected CNV between RPE/neurosensory retina (Type II MNV implied). Choroid not thickened.
	OCT Macula (OS)	No VMT. Decreased foveal depression. Intraretinal layers normal. RPE/BM regular. Choroid not thickened.
	Chest X-Ray	Cardiomegaly, Elongated/Atherosclerotic Aorta, Lungs normal.
Diagnosis	**Working Diagnosis**	1. Wet Age-Related Macular Degeneration (nAMD), Right Eye (OD). (Implied Type II / Classic based on OCT description) 2. Immature Senile Cataract, Both Eyes (ODS).
	Differential Diagnosis	Polypoidal Choroidal Vasculopathy (PCV) OD.
	Systemic Comorbidities	Hypertension. Renal Impairment (Elevated Urea/Creatinine). Hypertensive Heart Disease (Compensated).

Notes: OD (Oculus Dexter/Right Eye), OS (Oculus Sinister/Left Eye), OU (Oculi Uterque/Both Eyes), BCVA (Best Corrected Visual Acuity), IOP (Intraocular Pressure), BP (Blood Pressure), HR (Heart Rate), RR (Respiratory Rate), Temp (Temperature), BMI (Body Mass Index), C/D (Cup-to-Disc Ratio), A/V (Arteriovenous Ratio), NVD (Neovascularization of the Disc), NVE (Neovascularization Elsewhere), IRMA (Intraretinal Microvascular Abnormalities), VMT (Vitreomacular Traction), RPE (Retinal Pigment Epithelium), BM (Bruch's Membrane), PED (Pigment Epithelial Detachment), CNV (Choroidal Neovascularization), MNV (Macular Neovascularization), OCT (Optical Coherence Tomography), Hb (Hemoglobin), WBC (White Blood Cell Count), Plt (Platelet Count), Hct (Hematocrit), PT (Prothrombin Time), INR (International Normalized Ratio), APTT (Activated Partial Thromboplastin Time), SGOT (Serum Glutamic-Oxaloacetic Transaminase), SGPT (Serum Glutamic-Pyruvic Transaminase), BSS (Blood Sugar Screen - likely random), Na (Sodium), K (Potassium), Cl (Chloride), LDL (Low-Density Lipoprotein), Total Chol (Total Cholesterol), TG (Triglycerides), HBsAg (Hepatitis B Surface Antigen).

Table 2. Treatment course and follow-up summary for wet AMD OD.

Visit/Event	Right Eye (OD) Findings & Treatment	Left Eye (OS) Status
Baseline Visit	**BCVA:** 1/300; **IOP:** 13.1 mmHg; **Clinical:** Diminished foveal reflex; hard exudates; drusen; **OCT:** IRF (+); SRF (+); PED (+); Irregular RPE; Type II MNV suspected; **Plan:** Intravitreal Anti-VEGF Injection #1	**BCVA:** 6/60 ph 6/30; **IOP:** 12.0 mmHg; Immature Cataract; **OCT:** Normal macula
Injection #1	**Procedure:** Intravitreal Anti-VEGF (Agent unspecified; 0.05mL) administered superotemporally. Local anesthesia. No complications noted.	N/A
Follow-up Day 1	**BCVA:** 1/300; **IOP:** 15.6 mmHg; **Clinical:** Minimal pain; otherwise quiet eye; **Medications:** Oral Cefixime; Paracetamol prn; Topical Levofloxacin & Prednisolone Acetate OD prescribed.	**BCVA:** 6/60 ph 6/30; **IOP:** 15.6 mmHg; Unchanged.
Follow-up Day 7	**BCVA:** <1/60 ph(-) (Subjective improvement noted); **IOP:** 12.0 mmHg; **Clinical:** Fundus similar to baseline reported; but discussion suggests OCT improvement; **Medications:** Topical K+/Na+ Iodide ODS prescribed.	**BCVA:** 6/60 ph 6/30; **IOP:** 13.1 mmHg; Unchanged.
Follow-up Month 1	**BCVA:** 1/60; **IOP:** 14.0 mmHg; **OCT:** Significant reduction in IRF/SRF noted. PED slightly decreased. Persistent signs of MNV activity; **Plan:** Proceed with Injection #2.	**BCVA:** 6/30; Unchanged cataract.
Injection #2	**Procedure:** Intravitreal Anti-VEGF (Agent unspecified; 0.05mL) administered. No complications.	N/A
Follow-up Month 2	**BCVA:** 2/60; **IOP:** 13.5 mmHg; **OCT:** Further reduction in IRF/SRF. PED stable. Macula significantly drier but subtle fluid remains; **Plan:** Proceed with Injection #3 (completing loading phase).	**BCVA:** 6/30; Unchanged cataract.
Injection #3	**Procedure:** Intravitreal Anti-VEGF (Agent unspecified; 0.05mL) administered. No complications.	N/A
Follow-up Month 3	**BCVA:** 3/60; **IOP:** 14.2 mmHg; **OCT:** Trace IRF resolved. Minimal SRF remains. PED stable/slightly flattened. Macula near dry; **Plan:** Monitor monthly; initiate Treat-and-Extend (Extend interval to 6 weeks).	**BCVA:** 6/30; Unchanged cataract.
Follow-up Month 4.5	**BCVA:** 3/60; **IOP:** 13.8 mmHg; **OCT:** Macula remains dry. No new fluid or hemorrhage. PED stable; **Plan:** Administer Anti-VEGF #4; Extend interval to 8 weeks.	**BCVA:** 6/30; Unchanged cataract.
Follow-up Month 6.5	**BCVA:** 4/60; **IOP:** 14.0 mmHg; **OCT:** Macula remains dry. PED stable. No signs of MNV reactivation; **Plan:** Administer Anti-VEGF #5; Maintain 8-week interval.	**BCVA:** 6/30; Unchanged cataract.

Notes: BCVA = Best Corrected Visual Acuity; IOP = Intraocular Pressure; OD = Right Eye; OS = Left Eye; IRF = Intraretinal Fluid; SRF = Subretinal Fluid; PED = Pigment Epithelial Detachment; MNV = Macular Neovascularization; ph = Pinhole; N/A = Not Applicable.

3. Discussion

The patient presented with a primary complaint of the gradual onset of blurred vision in the right eye over a two-year period. This insidious progression of visual impairment aligns with the common clinical trajectory of nAMD, which, while it can sometimes manifest acutely, frequently develops gradually, leading to a slow decline in central vision. The patient's subjective description of his vision as "shadowy" or "smoky" is a clinically relevant observation, as these qualitative descriptors often reflect the presence of fluid accumulation or distortion within the macula, the central region of the retina responsible for sharp, detailed vision. The patient's age of 61 years is a significant demographic factor, placing him within the age group most commonly affected by AMD. The prevalence of AMD increases markedly with advancing age, particularly after the sixth decade of life, making age one of the most potent non-modifiable risk factors for the development and progression of this condition.

This highlights the importance of considering AMD in the differential diagnosis of older adults presenting with visual complaints. Several risk factors known to contribute to the development and progression of AMD were identified in this patient. Of these, the patient's history of smoking, with a consumption of one pack of cigarettes per day for over 20 years (20 pack-years), stands out as a particularly significant modifiable risk factor. Cigarette smoking has been consistently and strongly associated with an increased risk of both dry and wet AMD in numerous epidemiological studies. The relationship between smoking and AMD is dose-dependent, meaning that the risk increases with both the duration and intensity of smoking. The mechanisms by which smoking contributes to AMD pathogenesis are complex and multifactorial. These mechanisms include the induction of oxidative stress, the promotion of chronic inflammation, the impairment of choroidal blood flow, and the induction of hypoxia within the retinal tissues. Hypoxia, in turn, can stimulate the production of vascular endothelial growth factor (VEGF), a key mediator of choroidal neovascularization (CNV), the hallmark of wet AMD. In addition to smoking, the patient also had a history of systemic hypertension. Hypertension, a common cardiovascular condition, has also been implicated as a risk factor for AMD, although its role may be less direct and less potent than that of smoking. The proposed link between hypertension and AMD involves potential damage to the microvasculature of the choroid and retina, as well as disruption of the delicate balance of factors that maintain vascular homeostasis within the eye. While the patient's hypertension was reported to be controlled with medication, the long-term presence of this condition may still contribute to the overall risk profile for AMD. While the patient's body mass index (BMI) was within the ideal range, it is important to acknowledge that obesity is another recognized risk factor for AMD. Obesity, often associated with a pro-inflammatory state and alterations in lipid metabolism, may contribute to the development and progression of AMD through various systemic and local mechanisms. Genetic factors also play a substantial role in determining an individual's susceptibility to AMD. While a detailed genetic analysis was not performed in this case, and the patient denied a family history of similar eye conditions, it is well-established that specific genetic variants, particularly polymorphisms in genes related to the complement pathway (such as CFH, C3, and C2/CFB) and the ARMS2/HTRA1 locus, significantly increase the risk of developing AMD. The interplay between these genetic predispositions and the environmental and lifestyle risk factors discussed above ultimately shapes an individual's overall risk of developing AMD. In the context of patient management, addressing modifiable risk factors is of paramount importance. In this case, patient education regarding smoking cessation and the importance of continued management of hypertension are crucial components of long-term care. Smoking cessation interventions can have a significant impact on reducing the risk of AMD progression and preserving visual function. Similarly, effective management of hypertension can contribute to overall cardiovascular health and potentially mitigate its influence on ocular health.[11-15]

The diagnosis of nAMD in the right eye (OD) was established through a combination of clinical examination and multimodal imaging techniques. Clinical examination of the fundus revealed several characteristic features of AMD. These included the presence of drusen, which are extracellular deposits that accumulate beneath the retinal pigment epithelium (RPE), and hard exudates, which are lipid deposits resulting from the leakage of blood vessels. Additionally, a diminished foveal reflex was observed, indicating disruption of the normal reflective properties of the macula due to fluid accumulation or structural changes. Optical coherence tomography (OCT) imaging played a critical role in confirming the diagnosis and characterizing the extent of the disease process. OCT provided high-resolution, cross-sectional images of the retina, allowing for detailed visualization and quantification of intraretinal fluid (IRF) and sub-RPE fluid (SRF), both of which are

indicative of active neovascularization and leakage. The presence of pigment epithelial detachment (PED) was also noted, representing a separation of the RPE from the underlying Bruch's membrane. Furthermore, OCT imaging demonstrated features suggestive of Type II macular neovascularization (MNV), also known as classic choroidal neovascularization (CNV). In Type II MNV, abnormal blood vessels originating from the choroid penetrate Bruch's membrane and proliferate into the subretinal space. These OCT findings collectively provided definitive evidence of active nAMD and guided the subsequent treatment strategy. Fundus photography was used to document the clinical findings and corroborate the presence of drusen and exudates observed during fundus examination. While fundus photography provides valuable information about the structural changes in the retina, it does not offer the same level of detail regarding the location and extent of fluid accumulation and neovascularization as OCT. Fundus fluorescein angiography (FFA) is another important imaging modality used in the evaluation of nAMD. FFA involves the intravenous injection of a fluorescent dye and the subsequent imaging of the retinal and choroidal vasculature. FFA is particularly useful for identifying the type of CNV (classic versus occult) and delineating its extent and leakage patterns. However, in this particular case, FFA was contraindicated due to the patient's significant renal impairment. Renal impairment can increase the risk of adverse reactions to the dye used in FFA. This contraindication highlights the importance of considering patient-specific factors and comorbidities when selecting diagnostic imaging modalities. The increasing reliance on OCT and, where available, OCT angiography (OCTA) for the non-invasive diagnosis and monitoring of nAMD is noteworthy. OCTA is a relatively new imaging technique that visualizes retinal and choroidal vasculature by detecting the motion of blood cells, allowing for the identification and characterization of CNV without the need for dye injection. While OCT is highly sensitive for detecting fluid, it may be less specific than FFA for determining the precise CNV type or identifying subtle leakage patterns. Therefore, the choice of imaging modality depends on the specific clinical situation and the information required for diagnosis and management. In the differential diagnosis, polypoidal choroidal vasculopathy (PCV) was considered. PCV is a distinct clinical entity that can also cause neovascularization and fluid accumulation in the macula. While both nAMD and PCV share some similarities, they also exhibit important differences. PCV is characterized by the presence of branching vascular networks and polypoidal aneurysms, which are abnormal dilations of blood vessels. PCV is more prevalent in Asian populations and may exhibit a different response to anti-VEGF therapy compared to typical nAMD. In some cases, PCV may benefit from combination therapy involving photodynamic therapy (PDT) in addition to anti-VEGF agents. Indocyanine green angiography (ICGA) is often considered the preferred imaging modality for the diagnosis and characterization of PCV, as it provides better visualization of the choroidal vasculature compared to FFA. However, in this case, ICGA was not performed. While definitively ruling out PCV without ICGA can be challenging, the overall clinical and OCT picture in this patient was more consistent with a diagnosis of nAMD.[16-20]

4. Conclusion

This case report illustrates the successful management of neovascular age-related macular degeneration (nAMD) with intravitreal anti-VEGF monotherapy in a 61-year-old male patient. The patient presented with significant visual impairment and characteristic signs of nAMD, including intraretinal and subretinal fluid, pigment epithelial detachment, and choroidal neovascularization. Following the initiation of anti-VEGF treatment, the patient demonstrated marked improvement in both visual acuity and anatomical parameters, as evidenced by the reduction of intraretinal and subretinal fluid on OCT imaging. The case highlights the efficacy of anti-VEGF therapy in achieving favorable outcomes in

nAMD, even in the presence of risk factors such as smoking and hypertension. Timely intervention and consistent follow-up are crucial for maximizing visual potential and maintaining long-term stability. While this case demonstrates a positive short-term response, continued monitoring and management are essential to address the chronic nature of nAMD and prevent potential recurrences.

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