Deep Dive into ILM in Nontractional DME: Why We Should or Should Not Remove It

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ABSTRACT

Diabetic macular edema (DME) is one of the common features and leading causes of visual disturbance in diabetic retinopathy (DR). Although anti-vascular endothelial growth factor (anti-VEGF) injections remain the gold standard for therapy, vitrectomy has its own significant role with various indications. With growing advancement of internal limiting membrane (ILM) visualization and surgical instrumentation, some surgeons might routinely perform ILM peeling during vitrectomy. ILM peeling during vitrectomy has shown benefits in tractional DME; however, its role in nontractional DME remains controversial. To our knowledge, there are few studies focusing on ILM peeling and its role specifically in nontractional DME. This review aims to comprehensively examine and synthesize the existing literature. Published clinical studies show conflicting results, with some showing anatomical improvements while others suggesting some level of functional improvement. In the absence of definitive evidence favoring one approach, both the decision to perform or forego ILM peeling in nontractional DME can be justified. Further comparative clinical trials focusing on ILM peeling in nontractional DME are warranted to guide future decision making.

Keywords: ILM peeling; vitrectomy; diabetic macular edema; nontractional diabetic macular edema; diabetic retinopathy

INTRODUCTION

Diabetic Macular Edema (DME) is a leading cause of visual disturbance in patients with diabetic retinopathy (DR).(1,2) While intravitreal anti-vascular endothelial growth factor (anti-VEGF) injections are the established gold standard therapy, a substantial portion of patients show an incomplete response, resulting in persistent or recalcitrant edema.(3) For these clinically challenging cases, surgical intervention with pars plana vitrectomy (PPV) is an important therapeutic option.(4)

A key decision during vitrectomy for DME is whether to perform an additional internal limiting membrane (ILM) peeling. While the benefits are accepted for DME with clear vitreomacular traction, its role in nontractional DME remains a significant clinical controversy. This decision is made more complex as advancements in ILM visualization and surgical instrumentations have made peeling more accessible, yet the clinical evidence remains conflicting, as anatomical improvements reported in some studies do not always translate to definitive functional gains, making the risk-benefit analysis for surgeons particularly challenging.(4)

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To our knowledge, there is a lack of literature reviews focusing specifically on this distinct clinical scenario. This review aims to comprehensively examine the arguments for and against ILM peeling in nontractional DME. By synthesizing the existing evidence, we seek to clarify the potential benefits versus the risks, providing a deeper understanding to guide this complex clinical decision.(4)

METHODS OF LITERATURE SEARCH

A comprehensive search of PubMed, Scopus, ScienceDirect and Embase databases was conducted from November 2024 to August of 2025 using the following keywords: DME, ILM peeling, visual acuity, visual outcome, with further hand searching via search engines. Inclusion criteria were published articles, clinical trials or systematic reviews, while articles written in any language other than English is excluded. All abstracts were screened, and relevant articles were included in this review.

DISCUSSION

DME Definition, Classification, and Pathophysiology

Diabetic Macular Edema (DME) is characterized by retinal thickening from intraretinal fluid accumulation, primarily within the inner and outer plexiform layers. (5) With the advent of Optical Coherence Tomography (OCT), a key clinical classification for DME is based on the vitreoretinal interface: tractional or nontractional DME. Tractional DME involves evident pulling forces on the retina such as vitreofoveal traction, vitreomacular traction, epiretinal membrane proliferation, and a taut posterior hyaloid.(6–8) While, nontractional DME is defined by the absence of such forces, with macular swelling primarily driven by increased vascular permeability.(9) In this literature, we will be using the term tractional and nontractional DME, with focus on the latter.

The pathophysiology of DME is a complex interplay of vascular, inflammatory, and neural processes. A central event is the breakdown of the blood-retinal barrier (BRB), which leads to chronic fluid leakage into the macular tissue. This is driven by two key vascular events: increased vessel permeability and progressive vessel closure.(2,5,10) Vessel closure leads to retinal ischemia, which in turn stimulates the upregulation of growth factors like a vascular endothelial growth factor (VEGF), further exacerbating fluid leakage and contributing to a vicious cycle of edema.(1,2)

Furthermore, chronic hyperglycemia promotes the formation of advanced glycation end products (AGEs). These AGEs accumulate in the vitreous and at the ILM, contributing to both structural changes at the vitreomacular interface and direct osmotic effects that can worsen the edema. The resulting thickened and altered ILM may also hinder fluid outflow from the retina, perpetuating the edematous state and making it a key structure of interest in surgical management.(2,10)

DME Treatment

The standard treatments for DME have evolved throughout the years. At first, macular laser was the treatment of choice recommended by the ETDRS. Steroid injection is also a modality of choice with varying outcome. After the introduction of anti-Vascular Endothelial Growth Factor (anti-VEGF), the approach for treatment progressed from prevention of vision loss to improvement of vision. Until the time of writing, anti-VEGF injection remains the gold standard.(3,9,11)

Although effective in some, a significant portion of patients (35-65%) does not respond to anti-VEGF treatment. There is yet to be consensus on the official term for these unresponsive patients, but the term persistent DME (pDME) is often used. pDME can be described as patients with retinal thickness improvement of less than 10-25% after 6 months of treatment. In pDME, or if timely routine intervention is challenged by cost or living situations, surgical intervention specifically pars plana vitrectomy (PPV) with or without ILM peeling should be considered.(12)

Understanding ILM

ILM and its relation to Muller Cells

The internal limiting membrane (ILM) is the basement membrane of the vitreoretinal interface, physically separating the vitreous cavity from the retina. It is primarily composed of expansions from Müller cell endfeet, along with components like collagen and glycosaminoglycans, such as collagen IV, laminin 111, nidogen 1, agrin, perlecan and collagen XVIII.(13–15) Throughout life, the ILM continues to thicken, and its biomechanical properties, such as stiffness, can contribute to pathological tractional forces on

the retina. The ILM functions as a semi-permeable barrier through a meshwork of porous channels sized 10-25 nm in diameter, permitting the passive diffusion of small molecules, such as various ions, glucose, lactose and ascorbates, while hindering larger ones. (13,16) While crucial during retinal development, the ILM is considered dispensable in adults, which provides the rationale for its surgical removal in various vitreoretinal disorders.(17)

The clinical significance of ILM peeling is intrinsically linked to its intimate relationship with the underlying Müller cells, whose endfeet anchor directly to the ILM (Figure 1). Müller cells are essential glial cells responsible for maintaining retinal structure, metabolism, and overall homeostasis, with forming the ILM and the outer limiting membrane (OLM).(17,18) Consequently, any manipulation or removal of the ILM carries an inherent risk of damaging these vital cells. Such damage can lead to subsequent glial apoptosis, potentially compromising retinal integrity and function, a central consideration in the debate over ILM peeling.(19)

ILM in DR and DME

In DR, the ILM undergoes significant pathological changes. It becomes biochemically altered with an overexpression of components like collagen and fibronectin, leading to a marked increase in thickness and rigidity, with the mean increase of 1.8 μ to 4.8 μ in thickness (Figure 2).(21,22) This thickened, stiffer ILM is believed to contribute directly to the persistence DME in two ways: first, by acting as a dysfunctional diffusion barrier that hinders normal fluid outflow from the retina, and second, by providing a scaffold for tangential tractional forces.(22)

Furthermore, the ILM in diabetic eyes becomes a reservoir for pro-inflammatory and pro-fibrotic molecules, most notably AGEs.(2,23) The accumulation of AGEs strengthens the adhesion between the vitreous cortex and the ILM, which explains the increased vitreomacular traction seen in DR and why posterior vitreous detachment (PVD) can lead to DME resolution. (2) Additionally, these AGEs can

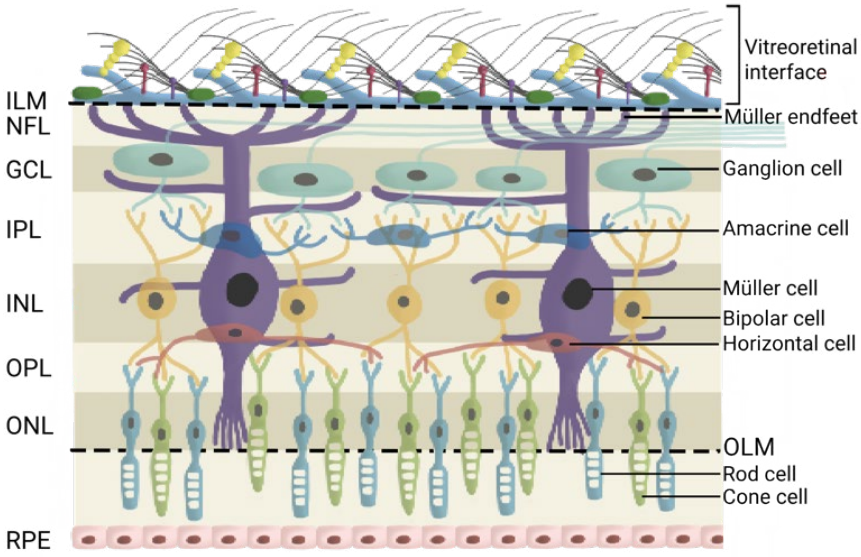

Figure 1. *Vitreoretinal interface and the nearby structures including the Muller cells endfeet. Original illustration, inspired by Agarwal et al.(20)*

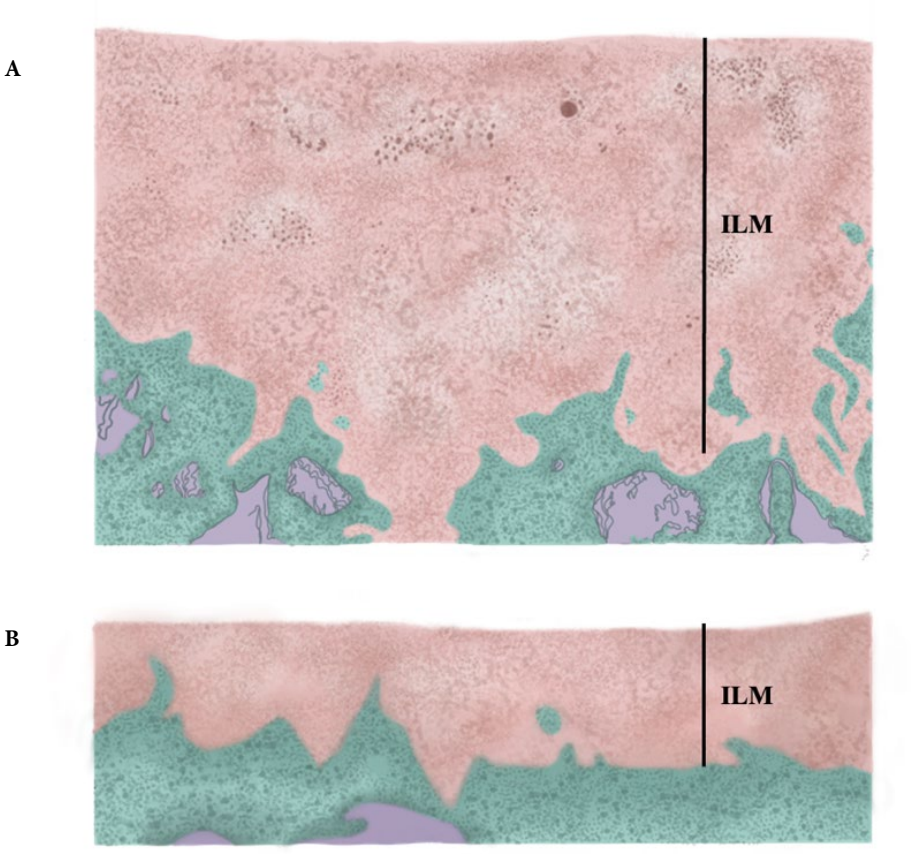

Figure 2. *ILM thickness in DME A) shows the thickness of the ILM in diabetic patients approximately double the thickness of nondiabetic patients B) shows the ILM thickness of nondiabetic patients in similar age. Original illustration, inspired by Halfter et al.(13)*

activate receptors of advanced glycation end products (RAGE) on the underlying Müller cells, promoting further inflammation and vasopermeability.(2,23) Müller cells themselves are found to upregulate VEGF in DR, adding to the cycle of leakage.(24,25) Collectively, these pathological changes provide a strong rationale for ILM peeling in DME, even in cases without obvious traction. The goal is to remove not just a physical barrier, but also a source of chronic inflammation and traction.(2,23,26)

What is ILM Peeling

ILM peeling is the removal of the ILM conducted right after vitrectomy. First, the ILM is stained for better visualisation to prevent traumatic injuries to adjacent structures. Several dyes can be used such as the trypan blue 0.15%, brilliant blue G, or indocyanine green. After staining, the ILM is removed in a circular area of two to three optical disc diameters around the fovea using a peeling forceps. (22,27,28)

ILM peeling complications are difficult to conclude because ILM peeling is usually conducted in combination with other surgical procedures. Some of the likely complications of ILM peeling are iatrogenic punctate chorioretinopathy(27,29,30), optic nerve fiber layer (which has been shown to have no effect on the visual function)(31,32), intraretinal emulsified silicone oil in ILM peeled area(33), vitreous hemorrhage (34,35), a temporary selective B-wave amplitude reduction(23) and phototoxic damage.(36)

Rationale for ILM Peeling in DME

Some rationale behind ILM peeling in nontractional DME is to prevent postoperative ERM formation. With ILM thickening in DME and its function as the scaffold for astrocyte proliferation, the fluid dynamics are potentially hindered.(22,37,38) Therefore, the removal of ILM is thought to reduce the recurrence of DME. Moreover, with the upregulation of VEGF in Muller cells and its consequent increase in the vasopermeability of retinal endothelial cells, ILM peeling might support clear removal of all vitreous cortex, including the inflammatory cells, supported by previous studies of ILM peeling in DME without taut posterior hyaloid showing significant decrease of foveal thickness and DME resolution.(2,23–26). Furthermore, improved retinal transduction in disrupted ILM also suggests anatomical retinal improvement prompted by the removal of ILM. (17)

Surgeons might also believe that ILM peeling could remove the reservoir for proinflammatory factors. The Muller cells closely located to the ILM is the location for RAGE. With the increase of RAGE axis in DM, the consequent proinflammatory features are also more likely to increase. Vitrectomy with ILM peeling is believed to remove the AGEs and prevent RAGE axis, as well as removal of the inflammatory cells.(2,23–25) Although most have attributed this to vitrectomy, a number of studies have suggested ILM peeling also play a role in improving oxygen diffusion through the vitreous cavity into the retina by removing the diffusion barrier.(8,26,28,39)

The Clinical Debate Arguments for and Against ILM Peeling

The decision to perform ILM peeling in cases of nontractional DME remains a subject of significant clinical debate. Below we have summarized a list of the points in favor and not in favor of ILM peeling featured in this paper (Table1). The procedure is underpinned by a strong pathophysiological rationale aiming to address the underlying causes of persistent edema. However, these theoretical benefits are weighed against potential risks at a cellular level and clinical evidence that shows conflicting functional outcomes.

Table 1. *Points in favor and not in favor of ILM Peeling*

In Favor of ILM Peeling	Not in Favor of ILM Peeling
It has been suggested that tractional effects might be more subtle than what OCT is able to detect. ILM peeling alongside vitrectomy is believed to remove AGE induced mechanical traction between the posterior cortex of the vitreous and the ILM, simultaneously removing the AGEs to prevent RAGE axis along its proinflammatory features.	Some studies on postmortem eyes and monkey eyes have shown damage to Muller cells after ILM peeling. The Muller cells modulate retinal ions concentration through voltage gated channels, limit excitatory signals, and partake in the metabolic functions of the inner retina. As a consequence, damage to these Muller cells can alter the retinal function.
Although it has a crucial role in the development of the eyes, the ILM is believed to be dispensable in adulthood,	The removal of ILM has been shown to expose nerve fiber layers directly to vitreous fluid in monkey and postmortem eyes.
The ILM have been shown to be thicker and more rigid in DME due to overexpression of several components, which could contribute to the persistence of edema and increased risk of tractional forces through hindering the fluid dynamics.	ILM peeling has shown delayed recovery of focal macular ERG B-waves likely caused by the Muller cells damage.
Some studies have shown significant decrease of foveal thickness and DME resolution with ILM peeling even without proof of traction. ILM peeling may help through the clear removal of all the vitreous cortex and in preventing formations of ERM, lower ERM formation after ILM peeling have been shown.	Most studies have shown no significant in visual acuity after ILM peeling, improvements shown have been limited to anatomical features.

Arguments in Favor of ILM Peeling

The primary rationale for ILM peeling is based on addressing the pathological changes the membrane undergoes in diabetic eyes. In DR, the ILM becomes thicker and more rigid, acting as a dysfunctional diffusion barrier that is thought to hinder fluid outflow and contribute to the persistence of edema.(21,22) Peeling the ILM is therefore believed to restore a more normal fluid dynamic. Furthermore, the diabetic ILM serves as a reservoir for advanced glycation end products (AGEs), which not only increase vitreomacular adhesion but also promote a pro-inflammatory state through RAGE activation on Müller cells.(23)

From a surgical standpoint, the ILM, while crucial during embryological development, is considered dispensable in adulthood.(17) This provides justification for its removal for therapeutic purposes in various vitreoretinal disorders. The clinical benefits of this removal in DME are primarily seen in anatomical outcomes. The study of Rosenblatt *et al*. and Hartley *et al*. suggested that ILM peeling in DME with no obvious traction could potentially relieve tangential traction, which allows clearance of edema and reduce retinal thickness.(23,28) Surgery was rationed by the possibility that tractional effects might be more subtle than what OCT is able to detect. Additionally, removing the ILM also eliminates the scaffold for future epiretinal membrane (ERM) proliferation, with studies showing lower rates of postoperative ERM formation in eyes that underwent peeling.(22,37,38)

Risks and Arguments Against ILM Peeling

Conversely, significant arguments against routine ILM peeling stem from evidence of iatrogenic retinal trauma. The procedure's intimate connection to the underlying Müller cell endfeet means that peeling inherently carries a risk of cellular damage. Studies on postmortem conducted by Wolf *et al.* and animal eyes coducted by Nakamura *et al.* have demonstrated tearing of Müller cell endfeet and direct exposure of the nerve fiber layer to the vitreous fluid after the ILM is removed (Figure 3).(40,41) Within the

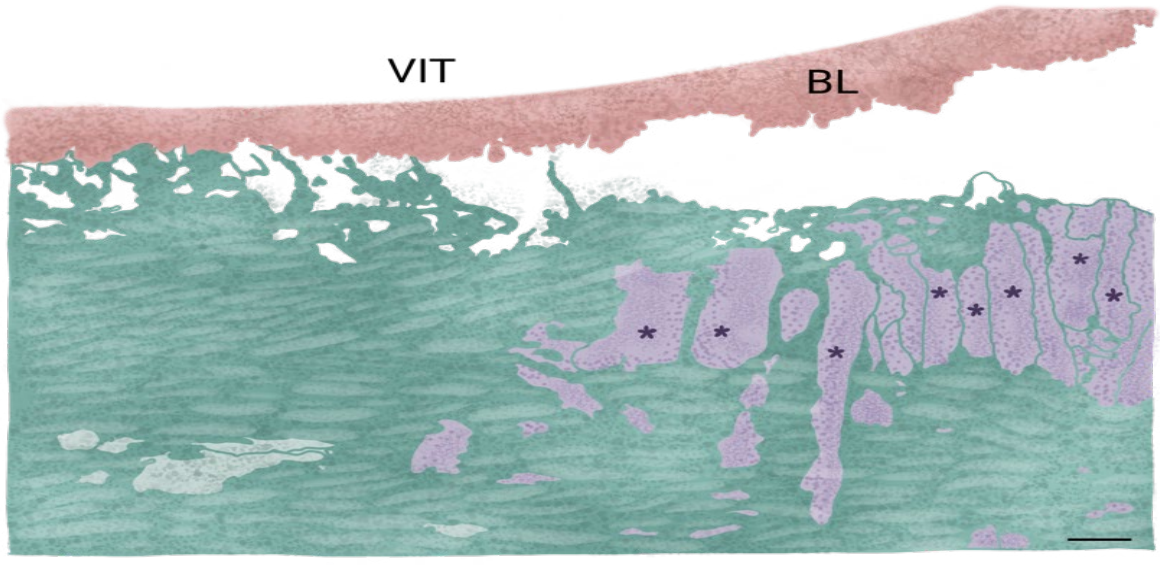

Figure 3. *Illustration of a peeled ILM showing several Muller cells endfeet damage (asterisks). VIT: vitreous, BL: basal lamina. Original illustration, inspired by Wolf et al. (50)*

peeled area, and even at its margins, a substantial number of Müller cells are damaged. This damage is not trivial, as Müller cells are vital for retinal homeostasis, and their injury can compromise overall retinal function.

This cellular damage may manifest as subclinical functional deficits. For instance, ILM peeling has been associated with a delayed recovery of focal macular ERG B-waves, an objective indicator of inner retinal function that is likely linked to Müller cell damage.(41,42) However, the most compelling argument against the procedure comes from the inconsistency in functional outcomes. Despite recent study by El-Khoury *et al.* that shows promising visual actuity improvement of ILM peeling for Refractory DME without vitreomacular traction, and consistent anatomical improvements, majority of clinical studies have failed to show a statistically significant improvement in the visual acuity. (26,28,37,43–45) A meta-analysis in 2015 also found no statistically significant difference in the visual acuity outcome nor macular edema reduction in vitrectomy alone compared to vitrectomy with ILM.(21)Another recent study by Vikas *et al.* further supported this notion when it only found anatomical improvements in both tractional and nontractional DME.(8) The study by Ranno *et al.* found neither anatomical nor functional improvement, but found that ERM formation was lower in eyes that underwent ILM peeling.(22) Most of the available clinical studies recognize that further comparative studies with larger number of participants are still needed to reach a consensus. The fact that patients undergo an additional surgical step without a guaranteed improvement in vision remains the central controversy and the strongest argument against its routine use in nontractional DME.

Conclusions

Vitrectomy with ILM peeling remains an option especially in refractory DME, or patients who cannot tolerate timely intravitreal anti-VEGFs injections. Without further comparative study, we cannot provide a clear consensus as to whether ILM peeling should or should not be conducted in nontractional DME. However, we hope the following points can help clinicians in weighing the risks and benefits on a case-to-case basis.

Conflict of Interest

The authors declare that there is no conflict of interest to disclose.

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