

# Incidence of Cystoid Macular Edema After Descemet Membrane Endothelial Keratoplasty

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**Purpose:** The incidence of and risk factors for cystoid macular edema (CME) after Descemet membrane endothelial keratoplasty (DMEK) remain uncertain. This study examines the incidence of and risk factors for CME after DMEK.

**Methods:** This retrospective, single-center study included patients with no history of CME who had undergone DMEK. Patients were examined weekly for 1 month after surgery and at 3 and 6 months after surgery. Follow-up examinations included visual acuity (VA) assessment, pachymetry, anterior segment optical coherence tomography, biomicroscopy, intraocular pressure measurement, and funduscopy. Eyes suspected of having CME (reduced VA and/or abnormal fundoscopic findings) underwent macular optical coherence tomography. Potential risk factors for CME examined included age, axial length, anterior chamber rebubbling, not using a topical nonsteroidal antiinflammatory after surgery, and concurrent DMEK and cataract surgery (triple-DMEK).

**Results:** Eighty eyes (74 subjects) were included. Eleven eyes (13.8%) developed CME within 6 months after undergoing DMEK. Univariate analyses did not identify any significant CME risk factors. Interestingly, the triple-DMEK procedure did not put subjects at risk for developing CME ( $P = 0.184$ ). Visual prognosis after medical treatment for CME was excellent, and subjects with and without CME had comparable VA at 6 months [CME: logarithm of the minimum angle of resolution (logMAR) VA = 0.3 (first–third quartile: 0.1–1.0), 20/40; no CME: logMAR VA = 0.3 (0.1–0.5), 20/40;  $P = 0.391$ ].

**Conclusions:** Although CME frequently occurred after DMEK, no CME risk factors were identified. In addition, CME did not significantly affect long-term visual outcomes when it was appropriately treated.

**Key Words:** cornea, cystoid macular edema, Descemet membrane endothelial keratoplasty, surgical complication

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Descemet membrane endothelial keratoplasty (DMEK) is a relatively new partial thickness corneal grafting technique that is used to treat corneal endothelial disorders, most commonly Fuchs corneal endothelial dystrophy. The DMEK procedure was first proposed by Melles et al in 2006.<sup>1,2</sup> Since that time, advancements in the DMEK technique have been made, and the procedure has become popular among corneal specialists, largely because functional and anatomical outcomes are superior to those of Descemet stripping automated endothelial keratoplasty and penetrating keratoplasty.<sup>3–5</sup> A number of research teams are currently studying DMEK outcomes and DMEK-associated complications. The largest of these studies to date ( $n = 500$  eyes) showed that common complications of DMEK include graft detachment (15.8%), rebubbling (3.0%), graft failure (0.4%), cataract (0.4%), graft rejection (0.2%), and retinal detachment (0.2%).<sup>6</sup> However, that study did not assess the incidence of cystoid macular edema (CME) after DMEK. Postoperative CME frequently occurs after ocular surgery through inflammatory pathophysiological mechanisms. More specifically, phospholipase A2 activation occurs, which leads to inner blood–retinal barrier rupture and a subsequent increase in perifoveal vessel permeability.<sup>7,8</sup> The risk factors for CME development after cataract surgery (Irvine–Gass syndrome) include age, history of uveitis, diabetes, retinal vein occlusion, prostaglandin eye drop use, and intraoperative complications (eg, posterior capsular rupture).<sup>9,10</sup>

Five research groups have previously investigated CME after DMEK. Heinzelmann et al<sup>11</sup> and Hoerster et al<sup>12</sup> reported similar CME incidences of 13% (20 of 155 eyes) and 12% (9 of 75 eyes), respectively. By contrast, Flanary et al,<sup>13</sup> Dapena et al,<sup>14</sup> and Chaurasia et al<sup>15</sup> reported an incidence of 7.5% (13 of 173 eyes), 0.7% (1 of 135 eyes), and 1% (3 of 292 eyes), respectively, in eyes that underwent DMEK. In addition, Chaurasia et al<sup>15</sup> observed a CME incidence of 1.5% (3 of 200 eyes) in eyes that underwent concurrent DMEK/ataract surgery. The current study further examined CME incidence after DMEK and investigated risk factors for developing CME.

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## METHODS

This study was reviewed and approved by the Edouard Herriot University Hospital Institutional Review Board (Lyon, France). Because this was a retrospective study, the requirement for obtaining study informed consent was waived. However, all subjects provided written informed consent to undergo standard-of-care DMEK or DMEK/cataract surgery according to our hospital's standard procedures. All study conduct adhered to the tenets of the Declaration of Helsinki.

### Study Subjects

This retrospective study included all patients who underwent DMEK alone or DMEK with concurrent cataract surgery (triple-DMEK) at the Edouard Herriot University Hospital (Lyon, France) between March 2014 and December 2015. Patients were excluded from analyses if they had a history of macular edema (regardless of the origin) before DMEK surgery.

### Surgical Techniques

Donor corneal tissue was obtained from the Lyon Cornea Bank at Edouard Herriot Hospital in all cases. All DMEK procedures were performed by 2 corneal specialists (V.K. and A.S.M.) using the same surgical techniques. Procedures were performed under general or local anesthesia, as per the surgeons' discretion.

### Graft Preparation

All DMEK grafts were harvested at the cornea bank or in the operating room using the "no touch" technique.<sup>16</sup> First, the endothelium was stained with trypan blue 0.06% (BCC; Croma-Pharma GmbH, Leobendorf, Austria) for approximately 4 minutes. Next, the Descemet membrane and endothelium were incised along the trabecular meshwork, elevated along the periphery (for approximately 2 mm) using a corneal epithelium spatula (MMSU 1113; Malosa Medical, Halifax, United Kingdom), and detached from the stroma by pulling upward with Kocaba DMEK forceps (MMSU1499S; Malosa Medical). A corneal trephine (Coronet, NM-51-903-8.00, single use; Network Medical Products Ltd, Ripon, United Kingdom) was used to cut an 8-mm central segment. Last, the trephine (still containing the donor button) was placed in a 0.09% sodium chloride solution to enable endothelium-out graft scrolling.

### Graft Injection and Positioning

After standard preparation for ocular surgical procedures, 4 paracenteses were made using a 15-degree cataract knife (BVI 374891; Beaver Visitec, Milton, United Kingdom). Iridotomy was then performed using the double-needle technique. Next, air was injected into the anterior chamber to enable performing descemetorhexis, which was done using a Price hook (17,302; Moria SA, Antony, France). A clear corneal incision was enlarged using a 2.2-mm slit knife (8065982265; Alcon, Fort Worth, TX). If not performed at the cornea bank, the graft was prepared for transplantation by

trypan blue 0.06% staining (2 baths, 4 minutes each), scrolling, and drawing it into a glass injector (G-38635, single use; Geuder AG, Heidelberg, Germany). The graft was then injected into the anterior chamber and deployed by gently tapping on the corneal surface. After placement verification, the clear corneal incision was hydrated to ensure self-sealing, and air was injected into the anterior chamber to seat the graft.

Patients undergoing both DMEK and cataract surgery had cataract surgery performed first using phacoemulsification through a 2.2-mm corneal incision. After cataract extraction, an intraocular lens was implanted into the posterior chamber, and Miochol-E (1:100, acetylcholine chloride intraocular solution; Bausch & Lomb, Rochester, NY) was injected to induce miosis for peripheral iridotomy before the DMEK procedure. All patients received a subconjunctival corticosteroid injection at the end of surgery.

Postoperative medication use included artificial tears (Celluvisc; Allergan France, Courbevoie, France), a corticosteroid antiinflammatory agent (dexamethasone/neomycin suspension, Alcon; 4 times a day for month 1, 3 times a day for months 2 and 3, twice a day for months 4–8, and once a day for months 9–12), and a miotic agent [Europtal (pilocarpine 1%), Vidal France, Issy-les-Moulineaux, France]. Patients who underwent a triple procedure also used a topical nonsteroidal antiinflammatory drop (NSAID) [indomethacin (indocollyre) 0.1%, Chauvin, France, 4 times a day for 5 weeks]. Patients were required to remain in a supine position until the anterior chamber air bubble disappeared. All patients remained hospitalized for 3 days after surgery. The anterior chamber was rebubbled if graft detachment occurred, regardless of the detachment size.

### Study Examinations

All subjects attended follow-up visits as per standard protocols. Postoperative examinations occurred at weeks 1, 2, 3, and 4 and at months 3 and 6. Examinations at each visit included corrected and uncorrected visual acuity (VA) assessment [logarithm of the minimum angle of resolution (logMAR) scale], intraocular pressure measurement, funduscopy, pachymetry (Pachy-map module, Visante-OCT 1000 system; Carl Zeiss Meditec, Inc, Dublin, CA), and anterior segment optical coherence tomography (OCT; Visante-OCT 1000). Patients suspected of having CME (reduced VA despite a clear cornea or abnormal fundoscopic findings) also underwent macular OCT imaging (Spectralis-OCT; Sanotek, Heidelberg, Germany). Macular thickness was automatically measured by the device. Subjects were diagnosed with CME if central macular thickness (central 1000  $\mu\text{m}$ ) was  $\geq 300 \mu\text{m}$  on Spectralis HRA imaging<sup>17</sup> or intraretinal (intraretinal edema) or subretinal (serous retinal detachment) fluid was detected on OCT.<sup>18</sup>

Subjects diagnosed with CME were initially treated with a topical NSAID [indomethacin (indocollyre) 0.1%, Chauvin, France; 3 times a day for 2 mo] or topical corticosteroids (dexamethasone/neomycin ophthalmic suspension, Alcon; 3 times a day for 2 mo) and oral acetazolamide (Diamox, Sanofi, Paris, France; 3 tablets a day). If

CME did not resolve with treatment, a 0.7-mg dexamethasone intravitreal implant (DEX implant, Ozurdex; Allergan plc, Dublin, Ireland) was placed in the affected eye.

## Data Collection

The following data were retrospectively collected from subject medical records: age, sex, laterality, surgical indication, ophthalmic history (including uveitis, retinal vein occlusion, and diabetic macular edema), axial length (IOLMaster 500; Carl Zeiss Meditec, Jena, Germany), preoperative prostaglandin use, intraoperative complications, and proposed postoperative NSAID use. The number of rebubbings was also noted.

## Statistical Analyses

Qualitative variables are presented as numbers and percentages. Continuous variables are presented as median (first and third quartiles). The 2 surgical groups (DMEK and DMEK/cataract) were compared using nonparametric tests (the Fisher test and Wilcoxon test for qualitative and continuous variables). Considering the discrete nature of follow-up visits, which were identical for all patients, an ordinal multinomial logistic regression model was used to estimate the effect of potential CME risk factors. Statistical significance was defined as  $P < 0.05$ . All data analyses were performed using R statistical software (version 3.0.2; R Development Core Team, Vienna, Austria).

## RESULTS

A total of 78 patients met all study inclusion criteria. Four patients were excluded from analyses because of follow-up loss (3 patients) and retinal detachment development 1 month after surgery (1 patient). Therefore, 80 eyes of 74 patients were ultimately included in analyses. Median subject age was 72.0 years (63.7–79.0 years), and 34 subjects (42.5%) were men. Tissue for the DMEK procedure was prepared at the cornea bank in 27 cases and in the operating room in 53 cases.

Table 1 summarizes the characteristics of subjects who underwent DMEK and triple-DMEK. Indications for surgery were Fuchs endothelial dystrophy [40 eyes (50.0%)], pseudophakic endothelial decompensation [24 eyes (30.0%)], primary graft failure [11 eyes (13.8%): perforating keratoplasty (PK) in 1 eye, Descemet stripping automated endothelial keratoplasty (DSAEK) in 2 eyes, and DMEK in 8 eyes], and endothelial decompensation after phakic intraocular lens implantation [5 eyes (6.3%)]. Axial length, diabetes history, rebubbling need, graft failure, and postoperative topical NSAID use/nonuse were not significantly different between surgical groups. All patients who underwent DMEK alone were pseudophakic and had undergone cataract surgery at least 6 months before DMEK.

The 6-month incidence of CME was 13.8% (11 of 80 eyes) in all included subjects. Most CME appeared between M1 and M3, as shown in Figure 1. Subjects who underwent DMEK alone had an 8.0% (3 of 37 eyes) CME incidence, and

**TABLE 1.** Characteristics of Patients Who Underwent DMEK Alone and DMEK in Combination With Cataract Surgery

Characteristic	DMEK in Combination With Cataract Surgery			P*
	DMEK Alone (N = 37 Eyes)	DMEK in Combination With Cataract Surgery (N = 43 Eyes)	All Patients (N = 80 Eyes)	
Age (yr)	73.0 (63.0–80.0)	69.0 (62.0–78.5)	72.0 (63.7–79.0)	0.42
CME	3 (8.1%)	8 (18.6%)	11 (13.8%)	0.301
Sex				
Male	21 (56.8%)	13 (30.2%)	34 (42.5%)	
Female	16 (43.0%)	30 (69.8%)	46 (57.5%)	0.023
Axial length (mm)	23.7 (22.9–24.3)	23.8 (22.9–24.8)	23.9 (22.8–24.7)	0.748
Rebubbling	22 (59.5%)	26 (60.5%)	48 (60.0%)	>0.999
Graft failure	11 (29.7%)	8 (18.6%)	19 (23.8%)	0.297
Subjects with CME	0 (0%)	0 (0%)	0 (0%)	
History of diabetes	4 (10.8%)	3 (7.0%)	7 (8.8%)	0.698
Subjects with CME	0 (0%)	0 (0%)	0 (0%)	
Postoperative NSAIDs	6 (16.2%)	32 (74.4%)	38 (47.5%)	<0.001
Subjects with CME	2 (5.4%)	5 (11.6%)	7 (8.8%)	
Indications				
Fuch dystrophy	0 (0%)	40 (93.0%)	40 (50.0%)	
Pseudophakic ED	24 (64.9%)	0 (0%)	24 (30.0%)	
Primary graft failure	10 (27.0%)	1 (2.3%)	11 (13.8%)	
ED after phakic IOL	3 (8.1%)	2 (4.7%)	5 (6.3%)	

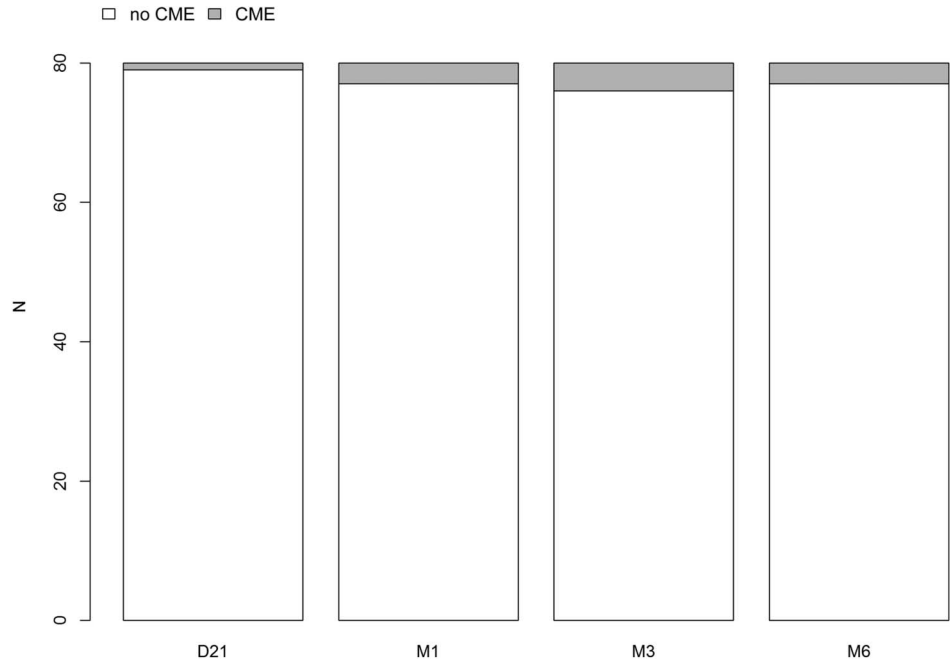
Data are presented as median (first–third quartile) or n (%).

\*P calculated using a Fisher or Wilcoxon test.

ED, endothelial decompensation; IOL, intraocular lens implantation.

subjects who underwent triple-DMEK had an 18.0% (8 of 43 eyes) CME incidence ( $P = 0.184$ ). Subjects who developed CME had a median central macular thickness of 467.0  $\mu\text{m}$  (356.0–568.5  $\mu\text{m}$ ). The logMAR VA was not significantly different between subjects with and without CME at month 6 [CME: 0.3 (0.1–1.0); no CME: 0.3 (0.1–0.5);  $P = 0.93$ ]. Median logMAR VA at month 6 was also not significantly different between the DMEK [0.3 (0.1–1.0)] and triple-DMEK [0.3 (0.1–0.5)] groups ( $P = 0.391$ ).

Average CME thickness was 467.2  $\mu\text{m}$  (345–800  $\mu\text{m}$ ), but CME did not occur in enough patients to compare thickness differences between groups. First-line medical therapy sufficiently treated CME, with the exception of 1 subject in the triple-DMEK group who had a remote history of ocular trauma. This subject needed second-line treatment with intravitreal dexamethasone. Three included subjects with primary open-angle glaucoma were using prostaglandins before surgery (2 patients in the triple-DMEK group and 1 patient in the DMEK group). None of these subjects had CME recurrence. Furthermore, univariate analyses revealed



**FIGURE 1.** Plot showing the incidence of CME in the first 6 months after Descemet membrane automated endothelial keratoplasty (n = 80 eyes). CME, cystoid macular edema.

that rebubbling, axial length, postoperative topical NSAID use/nonuse, combined surgery, and age were not statistically significant risk factors for developing CME (Table 2).

Surgical complications included rebubbling [48 eyes (60.0%)], graft failure [18 eyes (22.5%)], and postoperative ocular hypertension [1 eye (1.3%)]. Graft failure was not influenced by the type of surgery performed. All eyes with graft detachment were rebubbled, regardless of the detachment size. The median number of rebubbles for all included subjects was 1 (0.0–1.2, maximum = 4). Posterior capsule rupture occurred in 2 eyes in the triple-DMEK group (2.5% of all eyes, 4.7% of triple-DMEK eyes), none of which developed postoperative CME.

## DISCUSSION

This study found a relatively high CME incidence of 13.8% after DMEK. The CME incidence was not significantly different between patients who underwent only DMEK (8.0%) and those who underwent a combined DMEK/cataract

procedure (18.0%). Our results are consistent with previous reports in the literature. In a large prospective study involving 155 eyes, Heinzlmann et al<sup>11</sup> reported a post-DMEK CME incidence of 13%, with no significant difference in the CME incidence after DMEK and triple-DMEK. More recently, Hoerster et al<sup>12</sup> reported a CME incidence of 12% in a study that included 75 eyes, and Flanary et al<sup>13</sup> reported an incidence of 7.5% in a study that included 173 eyes.

Interestingly, none of the potential risk factors examined (age, axial length, surgery type, postoperative topical NSAID use/not use, and rebubbling) significantly increased the risk of developing CME. However, the incidence of CME observed after DMEK and triple-DMEK was higher than the incidence after cataract surgery alone (1%–2%).<sup>15</sup> This suggests that the DMEK procedure itself might be a risk factor for postoperative CME. The incidence of CME after cataract surgery has also been shown to be lower than that after Descemet stripping automated endothelial keratoplasty (5%)<sup>19</sup> and penetrating keratoplasty (9%).<sup>20</sup>

Evidence indicates that inflammation plays a large role in postoperative CME development. Heinzlmann et al<sup>11</sup> examined the CME incidence after DMEK and systematically performed macular OCT during the follow-up period. Therefore, it is not surprising that they found a higher CME incidence than other studies that did not examine all subjects using OCT. Indeed, in eyes with Irvine–Gass syndrome, OCT can be used to detect asymptomatic CME.<sup>21</sup> Interestingly, eyes with a short axial length are at greater risk for developing CME.<sup>11</sup> Theoretically, a shorter eyeball would lead to a shorter diffusion distance between the cornea and the retina. This would theoretically facilitate inflammatory cascade activation and, ultimately, edema development. As already shown after cataract surgery, intraoperative iridotomy and the presence of an anterior chamber bubble can stimulate the iris,

**TABLE 2.** Univariate Analyses of Suspected Risk Factors for Developing CME After DMEK

Risk Factor	Odds Ratio (95% CI)	P*
Rebubbling	1.90 (0.50–9.20)	0.373
Axial length	1.17 (0.75–1.77)	0.458
Postoperative NSAIDs	1.87 (0.52–7.68)	0.349
Combined surgery	2.59 (0.69–12.58)	0.184
Age	0.98 (0.93–1.03)	0.419

\*P calculated using Fisher and Wilcoxon tests.  
CI, confidence interval.



which contributes to increased postoperative inflammation.<sup>22</sup> Dapena et al<sup>14</sup> performed iridotomy before DMEK and found a much lower CME incidence of 0.7% after surgery. Their rebubbling rate was also very low (5.9%). In our study, intraoperative iridotomy was associated with a high rate of rebubbling, which may explain the 13.8% CME incidence observed here. In agreement with our findings, Heinzlmann et al<sup>11</sup> found that CME development did not significantly affect long-term visual outcomes and that the majority of CME cases could be successfully treated using topical medications. As in our study, they had only 1 patient who required intravitreal dexamethasone therapy.

Steroid therapy studies further support the large role of inflammation in postoperative CME development. Hoerster et al<sup>12</sup> directly compared intensive topical steroid therapy (hourly administration) with standard steroid therapy (administered 5 times a day) for preventing CME after triple-DMEK. None of the subjects who received intensive therapy for 1 week developed CME. By contrast, 9 of 75 standard therapy subjects (12%) developed CME. It should be noted that these findings may not be applicable to all clinical situations because of iatrogenic complication risks and the fact that only triple-DMEK cases were included in their study. All 9 patients with CME responded well to topical medical treatment and did not need intravitreal dexamethasone therapy. As in the current study, final VA was not significantly different between subjects with and without CME after CME treatment.

Our study found that the incidence of CME after DMEK is generally high and that there is not a “typical” patient who develops it. Flanary et al<sup>13</sup> examined post-DMEK CME incidence in patients who had recently (between 2 wk and 6 mo) and remotely (>6 mo) undergone cataract surgery. In agreement with our findings, these pseudophakic eyes had a CME incidence of 7.5%, with no significant difference between groups. Their data were also retrospectively collected and not all subjects underwent OCT macular imaging during follow-up. It should be noted that CME generally occurred in our study within the first 3 months after surgery [8 of 11 eyes (72.7%)], as is characteristic of Irvine–Gass syndrome.<sup>23</sup>

Examination of the graft failure rate was not a specific goal of this study. However, our study population had a high graft failure incidence (13.8%). This likely reflects the high level of preoperative severity of included cases. In addition, DMEK was performed on patients with various types of severe conditions, including Fuchs corneal dystrophy, pseudophakic endothelial decompensation, primary graft failure, and endothelial decompensation after phakic intraocular lens implantation.

Our study had several limitations, including its retrospective design, which introduced inherent biases, and a small sample size, which limited our ability to assess risk factors for post-DMEK CME. In addition, our study subjects did not undergo macular OCT imaging preoperatively or systematically at each postoperative visit. Therefore, our rates of CME detection may have been artificially low. Furthermore, fluorescein angiography was not performed, but can be helpful in detecting CME (represented as papillary and

macular leakage in the intermediate phase). Therefore, future studies are needed on a larger population to confirm our findings. These studies should include scheduled preoperative and postoperative macular imaging (OCT and fluorescein angiography) to rule out CME unrelated to surgery and to improve CME detection. Last, our study population was too small to assess the influence of diabetes, intraocular inflammation, and retinal vein occlusion on CME development.

In conclusion, our study of 80 eyes (the largest French DMEK case series to date) identified a postoperative CME incidence of 13.8%. This number is consistent with previously reported rates. Moreover, our findings suggest that, when appropriately treated, postoperative CME does not adversely affect final DMEK visual outcomes.

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