# Pattern of Cystoid Macular Edema

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## ABSTRACT

**Background:** Cystoid Macular Edema (CME) in its various forms can be considered one of the leading causes of central vision loss in the developed world. It is not a disease itself, It represents a common pathologic sequel of the retina and occurs in a variety of pathological conditions such as, diabetic retinopathy, central or branch retinal vein occlusion(CRVO,BRVO), intraocular inflammation and following cataract extraction.

**Objective**: This study was done to investigate the pattern of CME in patient attending Erbil Teaching Hospitals.

**Type of the study:** Cross- sectional study.

**Methods and Materials:** This is a hospital base cross-sectional study that included 61 patients (75 eyes) conducted at Erbil Teaching Hospitals for six months. All patients underwent a comprehensive assessment including medical and ophthalmic history, detailed ophthalmic examination and Optical Coherence Tomography (OCT) examination.

**Results:** of the 61 patients 32 (52.5%) were females and 29 (47.5%) were males. The mean age was (56.4±10.8) years. Out of the 75 eyes included in the study, 41 eyes (54.66%) had diabetic retinopathy, 10 eyes (13.34%) had CME following cataract operation (Irvine-Gass syndrome), 8 eyes (10.67%) had BRVO, 6 eyes (8%) were had CRVO, 5 eyes (6.66%) had age related macular degeneration, 3 eyes (4%) with uveitis, and 2eyes (2.67%) had Retinitis Pigmentosa. The average macular thickness was (415.6± 107).

**Conclusions:** Diabetic retinopathy is the most common predictive factor of CME, followed by cataract surgery. CME is more severe in diabetic retinopathy, CRVO and after cataract surgery.

**Key words**: Cystoid, macula, edema.

*Al-Kindy College Medical Journal Vol.12 No.1.Page: 122-128.*

* *Received at 27th April 2015, accepted in final 24th Feb 2016.*

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Cystoid Macular Edema (CME) in its various forms can be considered as one of the leading causes of central vision loss in the developed world.[1], It is not a disease itself, rather than the endpoint of a variety of processes that lead to the accumulation of fluid in the central retina.[3], it represents a common pathologic sequel of the retina and occurs in a variety of pathological conditions such as; diabetic retinopathy, central or branch retinal vein occlusion, intraocular inflammation like uveitis and following cataract extraction (Irvine-Gass syndrome)[4], approximately 20% of the patients who underwent uncomplicated phacoemulsification or extracapsular cataract extraction develop angiographically proven CME.[5]

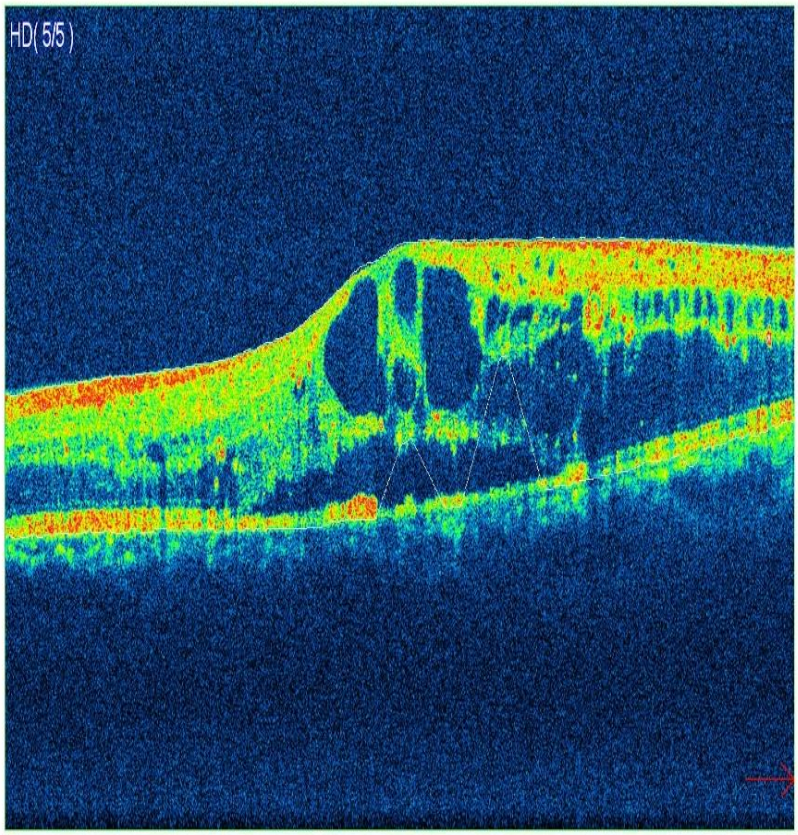
Other causes of CME are retinitis pigmentosa, gyrate atrophy, age related macular degenerations, viteromaculare traction syndrome, macular epiretinal membranes, tumor's such as heamangioblastoma and choroidal heamangioma and may caused by medications such as topical Adrenaline 2%, topical latanoprost and systemic nicotinic acid.[11]

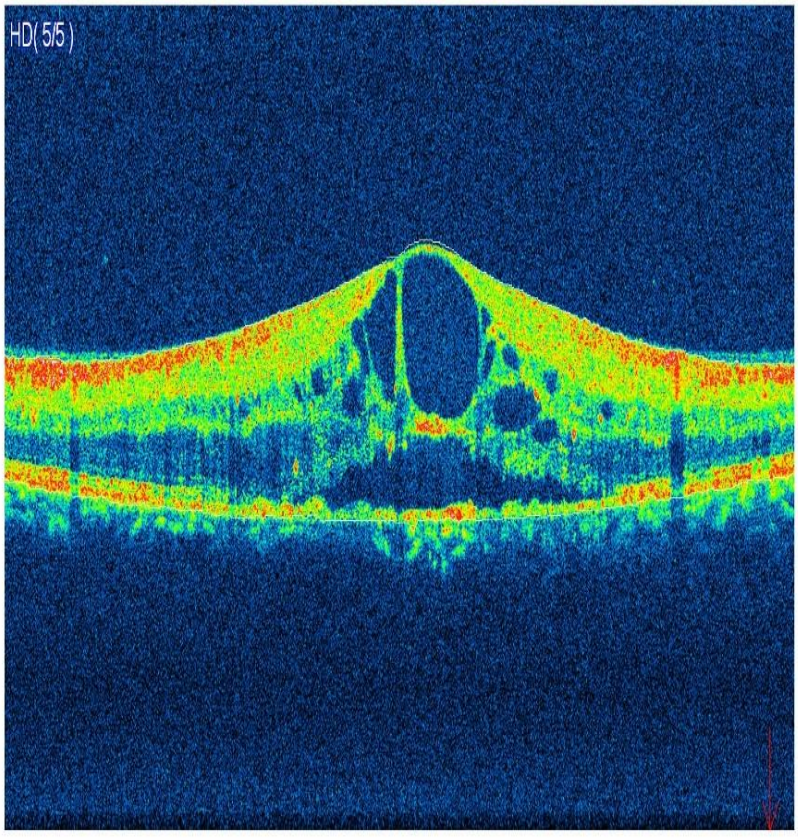
**Cystoid Macular Edema is** the result of accumulation of fluid in the outer plexiform and inner nuclear layers of the retina with the formation of fluid filled cyst like changes.[11]. The pathologic process varied from transudation, exudation to liquefaction necrosis, disruption of the blood-retinal barrier at the retinal vasculature and retinal pigment epithelium were noted in CME. Vascular leakage occurs after a breakdown of the blood-retinal barrier during traumatic, vascular, and inflammatory ocular diseases, and allows the serum to get into the retinal interstitium. Since intraretinal fluid distribution is restricted by two diffusion barriers, the inner and outer plexiform layers, serum leakage from intraretinal vessels causes cysts mainly in the inner nuclear layer while leakage from choroid/pigment epithelium generates (in addition to subretinal fluid accumulation) cyst formation in the Henle fiber layer.[13] . Clinical CME has historically been defined as a reduction in vision to 0.5 on logMar or less that is attributable to ophthalmoscopically or angiographically visible CME. It can present with symptoms of blurred or decreased central vision, and painless retinal swelling.[15]. CME is often asymptomatic and may only be detected with fluorescein angiography or Optical Coherence Tomography (OCT).[16]

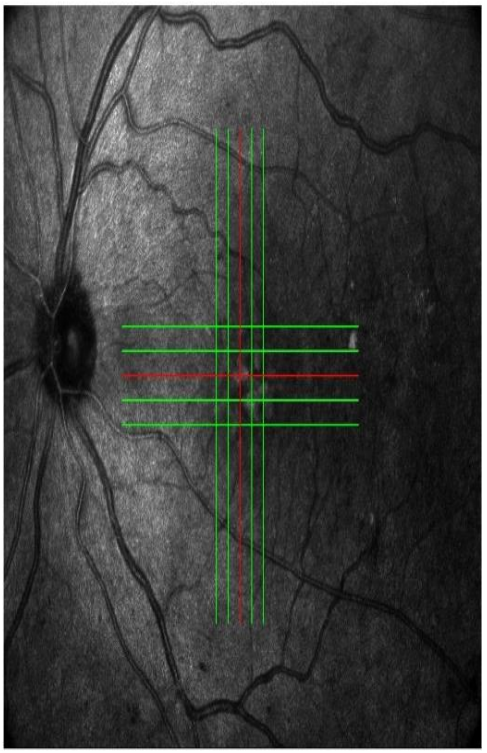
**Fundus fluorescein angiography (FFA)** is the gold standard for the diagnosis of CME, and give a flower-petal appearance as a result of the radial arrangement of both mullerian glia and Henle layer at fovea. However, as FFA is an invasive and qualitative method to detect CME, there is a tendency to use noninvasive and quantitative method like Optical Coherence Tomography (OCT).[17]

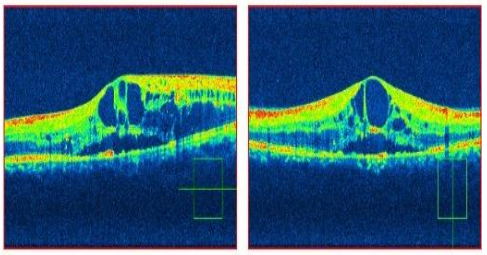
**Optical Coherence Tomography (OCT)** provides cross-sectional images of the retina, with the help of ~800 nm diode laser light.[18-21], and provides excellent visualization of cystoid fluid, which appears as cystic spaces of low reflectivity more prominent in the inner nuclear and outer plexiform layer as in Figure (1), and can assist clinicians in monitoring the progression of CME. [22] .

Statistical analyses were performed using a commercially available statistical software package (SPSS for Windows, Version 16.0, SPSS, and Chicago, IL, USA). Univariate categorical analyses were performed using Student's *t*-tests and Pearson's Chi-square tests, and a *p*-value of <0.05 was considered statistically significant.





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**Materials and Methods:**

This hospital based prospective, nonrandomized clinical study was carried out at Hawler and Rizgary Teaching Hospitals in Erbil between March 2013 and March 2014. Seventy five eyes of 61 patients with CME were recruited into the study

The patients were recruited into the study if they had significant CME (>320 µm) as measured by OCT (NIDEK, Model RS 3000 NAVIS-EX, Japan), decrease of visual acuity to 0.5 or less on logMar chart, and one of predictive factors of CME including patients that already diagnosed by medical department to have systemic problems like diabetes mellitus , systemic hypertension or hyperlipidemia and visual problem . The diagnosis of each patient was confirmed by OCT showing significant CME.

The exclusion criteria were the absence of significant CME and any opaque media that prevent visualization of the retina by OCT (dense corneal scar or cataract and vitreous hemorrhage).

All the patients underwent detailed ophthalmologic examinations, examination by OCT to prove the diagnosis of CME and to measure macular thickness.

Statistical analyses were performed using a commercially available statistical software package (SPSS for Windows, Version 16.0, SPSS, and Chicago, IL, USA). Univariate categorical analyses were performed using Student's *t*-tests and Pearson's Chi-square tests, and a *p*-value of <0.05 was considered statistically significan

**RESULTES**: Out of sixty one patients (75 eyes) who were included in our study, 29 were males (47.5%) and 32 were females (52.5%).Mean age of our sample was (56.43±10.8) years ranging from (17-72) years. Of the 61 patients, 14 patients (23%) had bilateral CME, 12 (85.76%) of them had diabetic retinopathy (regardless of stage). Figure (2)

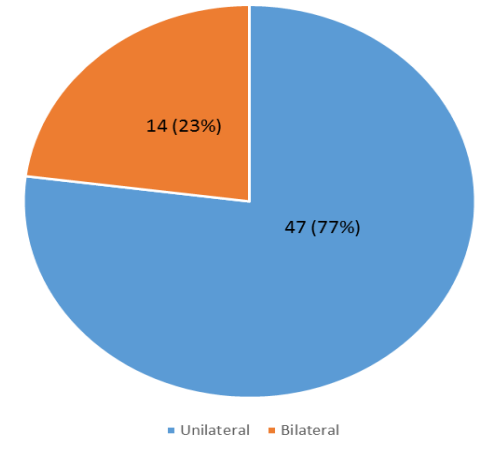



**Figure (2) No. and percentage of unilateral and bilateral cases**

**Figure (3) No. and percentage of patients with DM.**

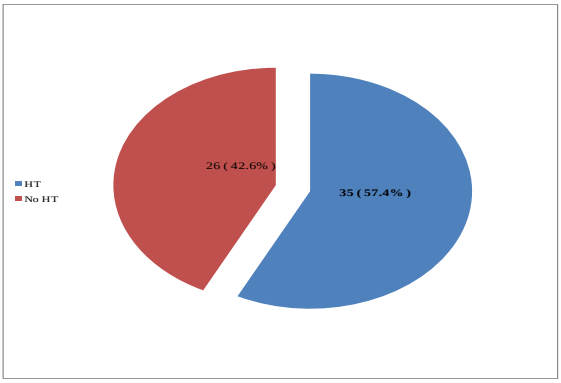



**Figure (4) No. and percentage of patients with systemic Hypertension**

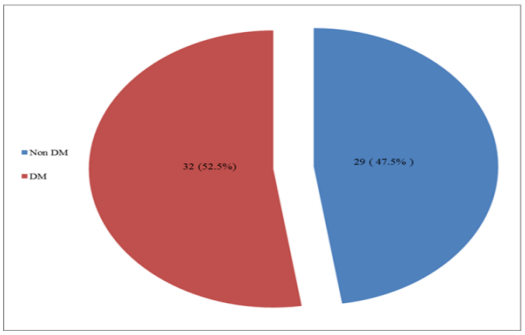
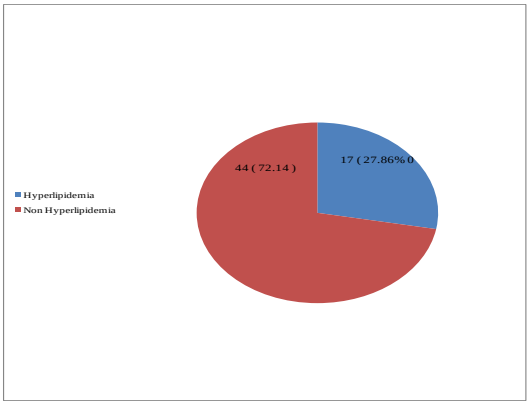



**Figure (5) No. and percentage of patients with Hypertension**




By studying the predictive factors of the 75 eyes that included in the study, 41 eyes
(54.66%) were diagnosed to have diabetic retinopathy, which is the most common predictive factor for the development of CME, followed by 10 eyes (13.34%) with CME after cataract operation ( Irvin Gass syndrome ), then 8 eyes (10.67%) were diagnosed to have BRVO, 6 eyes (8%) were diagnosed to have CRVO, 5 eyes (6.66%) had age related macular degeneration, 3 eyes

(4%) with uveitis, and lastly 2 (2.67%) with Retinitis Pigmentosa. Figure (6)

**Discussion:** The present study shows that the mean age of patients with CME was 56 years, and around this age, DM, and systemic hypertension are common, and both are risk factors for the development of CME, this was also reported by a study done in Iran [23]. 47.5% of patients were males and 52.5% of patients were females, the prevalence of CME was higher in females than males. The possible explanation could be that female gender is a significant risk factor for the development of diabetic maculopathy as reported by English town study [24], also hypertensive retinopathy seen more in females than in males and this agrees with a study done in Iran [23] and in Jordan Diabetes mellitus was present in 32 (52.5%) of the study patients, and 12 (37.5%) of those diabetic patients have bilateral CME. In 41eyes (54.66%) diabetic retinopathy affect the retina . This match a study done in Erbil city by Mustafa [26] in 2011.

In this study 35 (57.37%) patients had systemic hypertension. This could be explained by high prevalence of hypertensive retinopathy (48.5%) in Erbil city based on a study done in 2012 by Said [27], also arterial hypertension is a risk factor for the development of central and branch retinal vein occlusion , that are predictive factors of CME. Hyperlipidemia was found only in 17 (27.86%) of the patients with CME, this was consistent with United Kingdom study [24] that showed no significant association between serum cholesterol and maculopathy, but this finding didn`t match a studay in Germany [28] , that showed significant association between maculopathy and high serum cholesterol. The frequency distribution of eyes with CME was high in eyes with diabetic retinopathy, 41 eyes (54.66%). It is known that diabetes mellitus is a common disease, and the prevalence of diabetic retinopathy was high , and (20%) of patients with diabetic retinopathy had developed CME as shown in a previous study done in Erbil city by Mustafa [26] in 2011.

Colin J. [29] (2007) and Cable M. [30] (2012) postulate that CME is one of the most common causes of vision loss after cataract surgery. Its pathogenesis is likely multifactorial, but inflammation caused by surgical manipulations appears to be a major cause. In this study 10 eyes (13.34%) had CME that occurred after cataract extraction, and this is consistent with previous studies. [31] [32] [33] .

Measurement of macular thickness in the study patients revealed that, the average macular thickness was higher in diabetic retinopathy, it was statistically non-significant, but the values of retinal thickness in diabetic retinopathy were consistent with the values of previous study done in Erbil city [34].

The macular thickness and the severity of macular edema was more centrally, and lessen gradually as it goes peripherally in the macula, and this could be explained by understanding the anatomy and histology of macula, and the pathophysiology of CME, the macular region is predisposed to the collection of transudated fluids by virtue of its anatomic structure, the horizontal course of the outer plexiform layer extend transversally from cone nuclei to bipolar cells, and the resultant laxity of this layer predisposes to the formation of reservoir for the accumulation of transudate (Yamada [35]). Furthermore, the a vascularity of the foveolar area restricts absorption of fluid (Jaffe [36]). As a result of this predilection for the accumulation of fluid, the macula has been said by some investigators to "act as a sponge" (Cogan et al [37]). In addition to these anatomic considerations, the foveal region has large concentrations of cells with a high metabolic activity, inflammatory, metabolic, or vascular disturbance that can lead to increased concentrations of tissue metabolites with loss of biochemical activity (Ffyche and Blach [38]).

**Conclusions:** Diabetic retinopathy is the most common causative factor of CME, (54.66%) of cases, followed by cataract surgery (13.34%) and the macular edema is more severe in diabetic retinopathy, CRVO and after cataract surgery. Macular edema is more at the fovea centralis and reduced gradually as it goes peripherally in the macula, therefore OCT is recommended in any case with blurred vision and has risk factor for CME.

**Figure (6) the frequency of CME by predictive factors.**

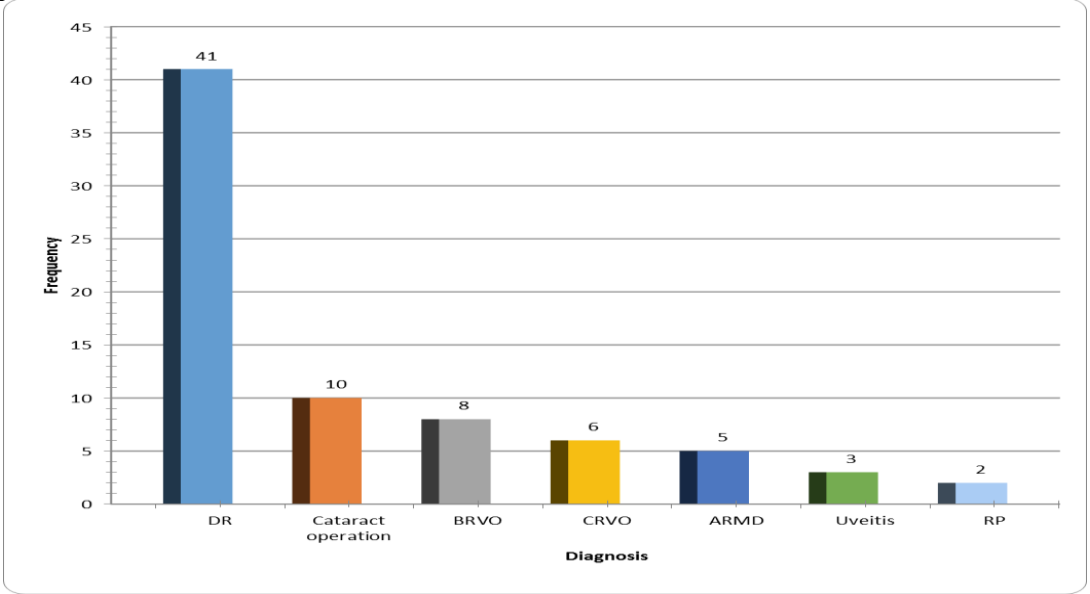


**Table (1). The mean average macular thickness at 6mm area among predictive factors.**

| Diagnosis | Mean | Std. Deviation |
|---|---|---|
| DR | 442.1165 | 123.66744 |
| CRVO | 419.1481 | 121.47642 |
| Cataract operation | 412.7444 | 57.33812 |
| RP | 380.8333 | 62.93250 |
| BRVO | 360.7778 | 42.43484 |
| Uveitis | 353.5556 | 14.65825 |
| AMD | 338.7111 | 68.56576 |
| Total | 415.6163 | 107.01552 |

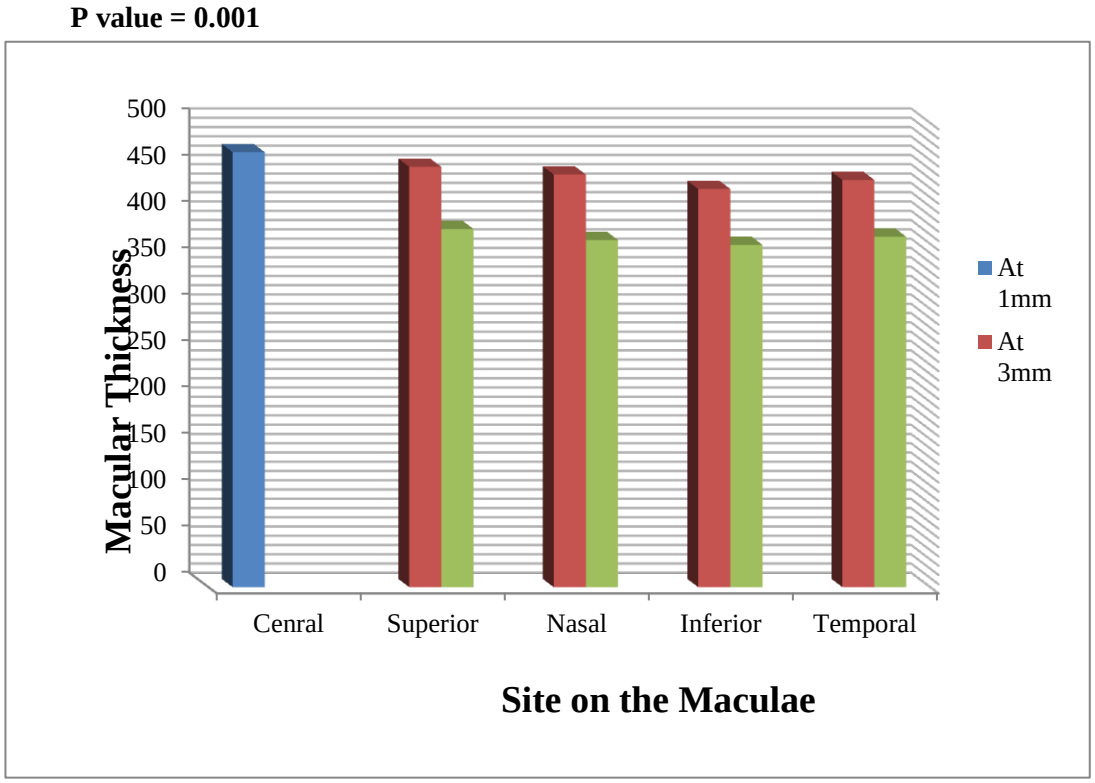
P value=0.206

**Table (2).The mean central macular thickness at 1mm among predictive factors.**

| Diagnosis | Mean | Std. Deviation |
|---|---|---|
| CRVO | 568.0 | 170.788 |
| Cataract operation | 499.0 | 112.823 |
| DR | 493.6 | 176.776 |
| RP | 478.5 | 166.170 |
| BRVO | 395.0 | 92.796 |
| Uveitis | 348.3 | 34.034 |
| AMD | 269.2 | 125.314 |

**Pvalue = 0.026**

**Figure (7) Mean macular thickness at different sites of macula.**

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