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# A MULTIDIMENSIONAL APPROACH TO CLINICALLY SIGNIFICANT MACULAR OEDEMA- AN OBSERVATIONAL SINGLE CASE STUDY

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## ABSTRACT

**Introduction**: Type 2 diabetes mellitus is a condition that is extremely serious from both clinical and public health stand point. its management needs appropriate guidelines for glycemic control and for handling complications to reduce mortality and morbidity, several aspects like diet, counseling, rehabilitation for complications and psychological management need specialized care, in this case report a patient centered muti-modality approach was designed considering the type and stage of the disease and empirical evaluation of the Ayurvedic management is been documented. **Methods:** Source of the data:36years old male patient who reported to OPD-GAMC Bengaluru with c/o uncontrolled blood sugar levels and defective vision – **Assessment criteria** – **Subjective parameters**-fatigue, diminished vision, distorted vision, floaters, flashes; **objective parameters**: FBS/PPBS/HbA1C,visual acuity 6/12 or less, amsler's grid aberration, fundus examination, OCT was analyzed before and after treatment; **Study design**- an open label single case report considering the stage of the disease was targeted with treatment protocols which was multi-focused. **Results:** There was reduction in subjective symptoms by 80%, objective assessment – showed good glycemic control with no future progression in the condition.
**Discussion:** The fact that high percentage of diabetic complications can be prevented by early detection of the symptoms and adopting Ayurvedic protocols which not only helps to arrest the disease progression but also helps to restore quality of life with no potential risks of side effects.

**Keywords**: Diabetes mellitus, Diabetic retinopathy, clinically significant macular edema.

## INTRODUCTION

Macular edema is one of the major causes of vision loss in individuals with diabetes[1, 2] and its development depends, in part, on the breakdown of the blood retinal barrier. Thus, we hypothesized that macular edema may be a marker of similar microvascular changes that may result in cardiovascular morbidity and mortality. The purpose of this study was to evaluate the association of clinically significant macular edema (CSME) and long-term survival in a population-based study of persons with type 1 and type 2 diabetes. Type 2 diabetes which accounts for 85-95 per cent of all diabetes has a latent, asymptomatic period of sub-clinical stages which often remains undiagnosed for several years[3].

As a result, in many patients the vascular complications are already present at the time of diagnosis of diabetes, which is often detected by an opportunistic testing. Asian populations in general, particularly Asian Indians have a high risk of developing diabetes at a younger age when compared with the western populations[4]. Therefore, it is essential that efforts are made to diagnose diabetes early so that the long term sufferings by the patients and the societal burden can be considerably mitigated. CSME was defined according to the Early Treatment Diabetic Retinopathy Study classification protocol[5] as the presence of retinal thickening at or within 500µm of the center of the macula or hard exudates at or within 500µm of the center of the macular if associated with thickening of the adjacent retina and/or zones of retinal thickening 1 disc area in size, at least part of which being within 1 disc diameter of the center. Epidemiological data from India have shown the presence of a number of risk factors which can be easily identified by simple non-invasive risk scores[6, 7]. In ayurveda it can be understood as timira due to *madhumeha* upadrva[8].

**Material and Methods:**

A Patient aged about 36 years reported approached to Shalakya dept., GAMC Bengaluru with C/O progressive blurriness of vision in both the eyes for both distant and near vision since 15days and developed floaters in LE occasionally and flashes of light in RE which caused a rapid drop of vision in BE within 1week.

**PAST HISTORY**: Nothing Contributory

**FAMILY HISTORY:** Patient's father and mother are known case of DM with DR; this could be a potential contributing factor.

**HISTORY OF CHIEF COMPLAINTS**: A 36-year-old Indian male, who is normosthenic, well nourished was coincidentally diagnosed with type 2 diabetes (hyperglycemia) around SIX months ago with a random sugar level of 449 mg/ dl, Surprisingly, he did not have any symptom of diabetes except FATIGUE, BOV for both distant and near objects, frontal headache (dragging) SINCE 15DAYS for which he randomly checked his blood sugar levels and found it to be high.

In the next day observation, the Fasting blood glucose level was found to 263 mg/dl and Post Prandial glucose level was measured to 402 mg/dl and HbA1c-14%.

Immediate after this shocking incidence, he consulted a OPTHALMOLOGIST as the blurrness of vision worsened within a week and was diagnosed with diabetic macular oedema BE, he was immediately treated with laser (LE) and intravitreal Intravitreal ranibizumab (0.5 mg) was advised to (RE) and was referred to a endocrinologist for management of his diabetes he was started on insulin (H.Mixtard 6-0-4units) and tab. voglibose 50/100 1-0-1, and after 15days he was treated with intravitreal injection to (RE) but his BOV remained the same and there was no improvement in his symptoms of fatigue hence he consulted at GAMC BENGALURU- EYE CLINIC ON 16/04/2018.

**TREATMENT HISTORY:** On consulting an ophthalmologist he was diagnosed as BE CSME and was treated with laser for LE- 1 SITTING, Intravitreal injection RE- 1 SITTING

And was advised for vitrectomy- RE Due to vitreous haemorrhage which the patient refused to undergo as there was no improvement in his vision found post laser or intravitreal injection.

On consulting an endocrinologist he was started on inj. human actrapid 6-0-4 units s/c, Tab voglibose 50/100 1-0-1 to control his blood sugars.

**INVESTIGATIONS**:

The systemic investigations revealed an uncontrolled hypoglycaemic status with fasting blood glucose of 263mg% and PPBS 402mg%, RBS 449mg %, HbA1C- 14%. Fundus photography confirmed the clinical diagnosis of CSME in BE, with foveal thickness of 339 (RE), and 316(LE)

**OCULAR EXAMINATION:**

**Visual Acuity for Distant Vision and near vision**

| V/A | Without glass | PH | Without glass | PH |
|---|---|---|---|---|
| **RE** | **1/60** | **1/60** | **N18** | **N18** |
| **LE** | **1/60** | **1/60** | **N18** | **N18** |

**AMSLER GRID TEST:**

RIGHT EYE: Patient visualized distorted lines in right upper and lower quadrants

LEFT EYE: Normal

**TABLE 1:** Confrontation Test

| OCULAR EXAMINATION | RIGHT EYE | LEFT EYE | NORMAL VALUES IN DEGREES |
|---|---|---|---|
| **ABOVE** | Normal | Normal | 50 degree |
| **BELOW** | Normal | Normal | 70 degree |
| **Medial (Nasal side)** | Normal | Normal | 60 degree |
| **Lateral (temporal side)** | Normal | Normal | 90 degree |

**TABLE 2:** External Ocular Examination

| STRUCTURE | RIGHT EYE | LEFT EYE |
|---|---|---|
| **CONJUNCTIVA** | No abnormalities | No abnormalities |
| **CORNEA** | Normal | Normal |
| **LENS** | clear | clear |
| **PUPIL** | Normal | Normal |

**FUNDOSCOPIC EXAMINATION:**

**TABLE 3:** Direct Ophthalmoscopy

| | RIGHT EYE | LEFT EYE |
|---|---|---|
| **MEDIA** | Hazy | Hazy |
| **FUNDUS** | Hard exudates++<br>Microaneurysms++<br>Haemorrhages++ | Hard exudates++<br>Microaneurysms++<br>Haemorrhages++<br>Laser spots + |
| **VESSELS** | Not appreciated | Not appreciated |
| **MACULA** | Not appreciated | Not appreciated |
| **OPTIC DISC** | CDR- 0.3 | CDR- 0.3 |

**TABLE 4:** Indirect Ophthalmoscopy

| RIGHT EYE | LEFT EYE |
|---|---|
| **Vitreous haemorrhage +** | Vitreous haemorrhage+ , LASER spots + |

**TABLE 5:** Slit-Lamp Biomicroscopy

| RIGHT EYE | LEFT EYE |
|---|---|
| Normal | Normal |

## 2. METHODS:

**CLINICAL STUDY:** An observational clinical study was carried out after assessing the *vyadhi avasta* and accordingly *chikitsa siddhanta* was planned.

Study design:- it was a observational study

Source of data:- Patient who reported to eye OPD GAMC, Bengaluru

Assessment criteria:

- Subjective – V/A Distant and near vision
- Distorted vision
- Floaters
- Flashes
- Objective – FBS
- PPBS
- HbA1c
- Fundus photograph
- **OCT**

After examination of the patient his consent was obtained for the treatment and prognosis was explained in detail.

Patient is been treated on OPD basis and the observational study was conducted for about 48days.

Treatment Plan:- *Samprapti Vigatana Chikitsa*

Frame of *samprapti- Dosha –sannipataja* with *kapha, pitta* predominance- due to leakage, exudates and oedema with haemorrhage

| | | | |
|---|---|---|---|
| Agni deepana, Ama pachana – 3 days | Vaishwanara churna- ¾ tsp tds | Kriya kalpa-<br>Mrudveekadi kashaya seka | Justification |
| Virechana – 7days | Snehapana – durva ghritha<br>D1- 30ml<br>D2-70ml<br>D3-120ml<br>D4-moorchitha taila abhyanga and bashpa sweda<br>D5-abhyanga and sweda<br>D6-abhyanga and sweda<br>Virechana – Trivruth avalehya 60gm with draksha kashaya 100ml | Mrudveekadi kashaya seka, Triphala guduchi bidalaka | Kleda Hara,<br>vatanulomana-To arrest neovascularisation<br>srotorodha hara,<br>rakta pitta, shotha hara. -to reduce the macular odema |
| Moordha lepa- 7days | Vasa, musta, amalaki, punarnava,manjista brishta with patoladi gritha | Mrudveekadi kashaya seka | Sandhana and skandana of Urdhwaga Rakta Pitta - To Promote The Resorption Of The Bleed |
| Durva ghritha pratimarsha nasya | Haemostatic,<br>anti hypoglycemic effect | | Rakta Prasadana,<br>Pramehahara Chikitsa |
| Shamana - | Maha vasakadi ksshaya- thikta rasa | Prevents the pitta dosha from being affected | Pramehahara Chikitsa |
| Madhumeha chikitsa | Chandraprabha vati 2-0-2 | Rasayana | Sophahara,Rasayana Chikitsa |
| After vishrama kala of 7days<br><br>After basti during parihara kala of 20days | Madhutailika basti[10]- kala basti was given -8days<br><br>Mahavasakadikashaya 100ml BD<br>Tab. Chandraprabha vati 2-0-2<br>Shatavari amalaka[12] granules 6gm bd | Mrudveekadi kashaya seka continued<br><br>Ushiradi anjana[11] 1-0-1<br>30mg was administered. | • Rasayana<br>• To arrest the progression,<br>• To maintain the integrity of neuro retinal rim<br>• To promote blood supply and reduces oxidative stress at cellular level. |

*Dooshya- rakta dhatu, rakta vaha srotas*

- *Srotas- sanga in sookshma srothuses*
- *Vatapratilomata in sukshma srotas*
- *Sopha* ***or*** *rakta srava* develops in nethra – Timira chikitsa[9]

Samprapti – due to agninasa and kleda vriddhi leads to sanga (occlusion of retinal vessels), siragranthi (aneurysms) athipravruthi (neo vascularisation) vimargagamana (retinal haemorrhage) leading to shopha and srava in netra.

Vyadhi – INDRIYAPRADHOSHAJA VYADHI

**DRUSTI VIKARA - MADHUMEHA JANYA TIMIRA**

(Parathantra vyadhi)

48 Days- Study Period

Follow Up- 1month

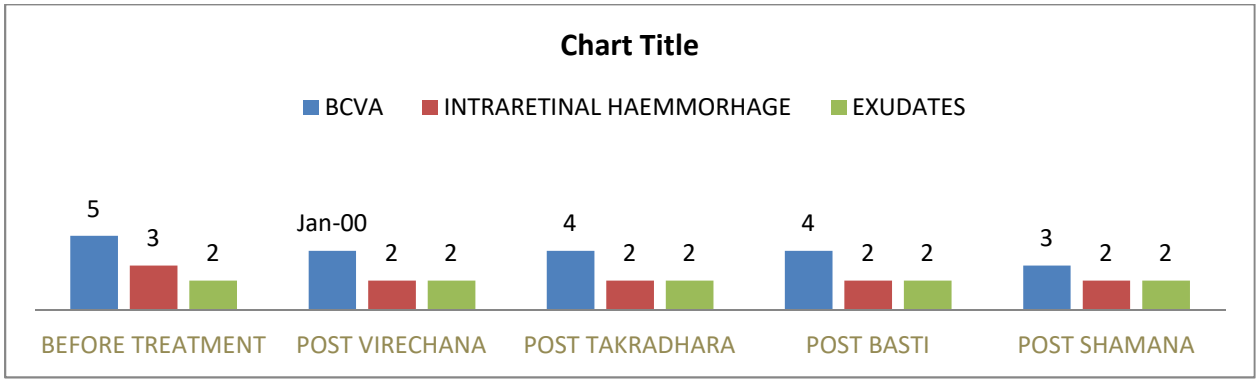
**OUT COME AND FOLLOW-UP:**

During the subsequent follow-ups clarity of vision had improved in the both eyes and blood sugar was well under control.

Results were assessed based on general functional capability grades both for subjective and objective parameters.

80% relief was found in subjective parameters

60% - control in objective parameters




## DISCUSSION

As per ayurveda, eye is tejomaya. This means that eye is highly vascular in nature. The pitta-rakta hara composition of the eye is responsible for its karma, and it has threats always from kapha dosha, and it is also evident that *prameha* is a disease having kapha domination in the beginning of samprapti. The kaphadosha and resultant kleda leads to vascular changes in the eye which are later manifested as diabetic retinopathy, the major samprapti in DR is the khavaigunya in the sookshma raktavahasrothuses of eye. The events related with micro angiopathy in retinal vessels are initially caused by kapha and kleda in thejomaya netra. Later the events are haemorrhage leading to severe visual loss, understanding the same pathology this clinical case was subjected to chikitsa siddanta to reverse the samprapti and the documentation was done with respect to its subjective and objective parameters which showed marked improvement in vision and the same was well maintained even in the follow up period.

| | POST VIRECHANA DT | POST TAKRADHARA | POST BASTI | POST SHAMANA- AT |
|---|---|---|---|---|
| BCVA- DISTANT VISION -BE | 4/60 , 4/60 | 6/60 , 6/60 | 6/36P, 6/24P | 6/36P, 6/24P |
| NEAR VISION | N24 , N24 | N24 , N24 | N24 , N24 | N24 , N24 |
| FLOATERS AND FLASHES | OCCASSIONALLY PRESENT | ABSENT | ABSENT | ABSENT |
| COLOUR PERCEP- | ABLE TO APPRECIATE | ABLE TO APPRECI- | ABLE TO APPRECIATE | ABLE TO APPRECIATE |

| TION | TILL PLATE 2 | ATE TILL PLATE 2 | TILL PLATE 2 | TILL PLATE 2 |
|---|---|---|---|---|
| Hba1c,<br>RBS | 14%<br>485 | 166 | 132 | 5.9%<br>116 |
| FUNDUS | VESSELS DETECTED | PARA FOVEAL COT-TON SPOTS SEEN | HAEMMORHAGE <9/12 TH QUANTILE | HAEMMORHAGE <9/12 TH QUANTILE |

## CONCLUSION

DR passes through different dosha dominant stages, though the overall pathogenesis in DR is sannipathika in nature with specific dosha profile the treatment requires to be effectively adopted to arrest the progression of the disease at the earliest and to bestow best visual rehabilitation possible, restoration of functional and structural integrity was the objective in this case and the treatment principle adopted in this clinical case proved efficacious in controlling haemorrhages, oedema and in subsequent follow-ups improved vision was documented, hence a right approach which is multi-dimensional will definitely retard the progression of the disease and helps maintains the retinal function.

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