REVIEW OF LITRATURE
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Complications of Diabetes mellitus are known to occur in almost every part of the visual organ. Some of these changes are of no importance and are not characteristic. Other, however, are pathognomonic for diabetes. Statistical data about the incidence of blindness due to its ophthalmic complications is 20 times more than the incidence of blindness in general population. According to data from statistics on blindness in model reporting area, 1962-70, diabetic retinopathy was responsible for 11.1% of the new cases of legal blindness in all the age groups and 19.1% of those in the 20 to 64 year age group.

The ocular changes related to diabetics are:

Lids:

Blepharitis, recurrent Hordeolum and eczema with poor healing tendencies should always place diabetes as a cause on the list of differential diagnostic possibilities. In diabetics, simple infections may take a serious course. Gangrene of the lid following hordeolum or orbital cellulitis in dacryocystitis are examples.

Conjunctiva:

Aneurysms of the conjunctival blood vessels are found in 55% of diabetics (Mc Culloch and Pashby 1950). The significance is disputed. We agree with Velhagen 1943,
who stated that these conjunctival changes allow no conclusions as to type and stage of the diabetes.

Cornea:

This is affected in several ways. A classic sign is presence of corneal striae or folds of Descemet's membrane sometimes called Beetham's lines, secondary to severe ocular hypotony Waite and Beetham 1935. However, these changes are not specific for diabetes. Staining of the cornea was found by Janert to be more common in diabetics. Epithelial keratodystrophy was published in Italian literature by Quaranta 1954 and confirmed by Iolipada in 1864. The decreased corneal sensitivity was first demonstrated by Schullica and Proto and confirmed Schwartz in 1974, decrease in corneal sensitivity is believed to be part of a generalized polyneuropathy that develops in diabetes mellitus (Schwartz 1974). The neuropathy mechanism is known but on ischemic angiopathy of vasa nervorum or other small vessels may be involved (Woltman, Wilder 1929).

A demyelinating process may exist, perhaps related to chronic schwann cell dysfunction or alteration in the level of nerve free myoinositol content may also contribute to the neuropathy (Thomas 1966).
Awasthi et al (1974) have recently shown that the reduction in corneal sensitivity is more significant in patients with proliferative retinopathy. Recurrent corneal erosion which do not heal rapidly is seen (Joslin 1978). Deficient tear production and basement membrane abnormalities may also contribute to epithelial breakdown or epithelial healing problem in some diabetic patients (Mandelcorn 1976). In a study of 100 cases by Prakash Kannan and Ananda noted following corneal changes like decrease in corneal sensitivity. Epithelial defects, erosions, neurotrophic ulcers, stroma increase in corneal thickness, opacities, Descemets membrane wrinkles, Endothelium undergoing polymegathism, pleomorphism, pigment deposits. Deep corneal opacities were also observed by Noothoven Van Goor and Schaly (1963).

Iris:

Diabetes uniformly affects the iris. The presence of diabetes in life can be inferred by the finding of glycogen deposition in pigment epithelium of the iris in post-mortem specimens. (Becker 1883, Waite 1935). Diabetic iritis was first described by Marchal in 1863 and occurs in 0.8 - 8% of cases. Elschnig 1937 differentiates a metastatic type of iritis with a severe course from a different type that is caused by destruction of pigment and
tend to cause glaucoma. Hydropic degeneration of the pigment epithelium is the cause of the black staining of the aqueous humour during intraocular surgery (Armaly and Baloglov 1967).

Diabetic rubeosis iridis was first described in 1876 by Abadie and was named by R. Salus 1928. This is characterized by neovascularization in the pupillary portion of iris and in the chamber angle. Rubeosis develops in iris without signs of inflammation. This was also explained by Kurz 1932 and Francois 1951. Fibrovascular membrane develop causing radial contraction with the result that the pupil is distorted and the posterior iris pigment epithelium pulled through the pupil into the anterior surface of the iris Ectopic uveae, this membrane may grow further producing pupillary membrane (Schulze 1967). As these new vessels external into the angle they, along with fibrous membrane, which accompanies them, start to occlude the aqueous outflow channel leading to increase intraocular tension (Smith 1954). Occasionally the new vessels disappear spontaneously and with them the raised tension. Fixed pupils may occur on one or both sides in diabetics. Glycogen infiltration into the epithelial cells and subsequent degeneration in iris epithelium may be responsible for diabetic pupil to respond delay to mydriatics (Duke Elder 1940).
Lenses :

The first report of diabetics relation with cataract was given by John Rallo (1798). While by the time Bendt (1834), T Bendict (1842) and Himly (1843) have also reported that the patients who are diabetic they are prone to develop cataract.

The percentage of patients representing for surgical treatment of cataract has been found to be higher among diabetic than non-diabetics (Clegg 1920), Anthonisen 1936, Towners and Carey 1955, Norm 1967). All found that about 5% of patients who have had cataract extraction were diabetics. Caird et al 1964 and Pirie 1965 found 3% of all patients who came to cataract OPD were having diabetes.

Kirby 1933, found that 64% of all the diabetics has lenticular changes of same kind of which 70% were of senile cataract type. 21% nuclear, 7% posterior cortical and 2% subcapsular type. Lenticular opacities may appears at a relatively early age in people in the prediabetic state (Pautique and Michand 1965; Buckner 1965; Van Salm 1966). Caird, Pirie and Ramsell 1969 noted 4 to 6 times cataract more likely to develop at a young's age and to progress more rapidly. The recorded proportion of juvenile diabetics having opacities varies from 2 to 5% (Keim 1950, Janert 1956, Mohrike 1956, Gunther 1956, Forsium et al 1964, Knowles
et al 1965, Burdi Et and Caird 1965) found a frequency of 5% and 11% after 10 years of diabetes and 23 to 32% after 20 years.

Classically the diabetic or floccular cataract consists of showers of small granular opacities (Christmas tree pattern). These appear as "snow flake" dots directly under both the anterior and posterior lens capsules. Van Heyningen (1950) sought the pathogenesis of metabolic cataract he said increased concentration of glucose enter the lens and activate the enzyme aldose reductase which favours the conversion of glucose to sorbitol. Sorbitol is not promptly metabolized but through osmotic effect draws water into the lens bringing with it sodium, which damages the lens fibers and result cataract formation.

The risk of cataract associated with diabetes is greater in females than in males (John J harding 1993). It was seen that cataract extraction is relatively commoner in diabetic women than in men specially under the age of 70 years according to study by Pules 1956, Fitzgerald et al 1961.

Perkins ES (1983) incidence of diabetes in cataract series was 13.66%. The higher ratio of women to men over in the diabetes was particularly striking and the age at which the surgery was performed was significantly lower in both men and women than in non-diabetics.
Statistics of blindness registration for cataract thought to be diabetic in origin are few in number and are subject to all the reservations in relation to blindness registration for cataract as a whole. Data from Canada 1865 and from a small study in west of Scotland (Committee on blindness 1910) suggest that blindness from cataract in diabetics total about 25 - 33% of that due to diabetic retinopathy and represents about 2% of all blind registrations of diabetics registered blind from cataract 70% are over 70 years old and 81% are women.

Cataract extraction in the diabetic carries increased risk of rubeosis iridis, neovascular glaucoma, acceleration of proliferative diabetic retinopathy with or without vitreous haemorrhage and difficulty in corneal wound and epithelial healing.

There is good agreement that the visual results of cataract extraction are almost good in diabetics without retinopathy as in non-diabetics. About 80% of both groups will gain vision of 6/12 or better. Where if retinopathy is present only 34% of patients will achieve this acuity. Retinopathy is main reason for poor visual results after cataract extraction in diabetics, as other ocular diseases are in non-diabetics (Caird et al 1965). Retinopathy is by...
in presence of quite severe retinopathy and when the visual acuity is not greatly improved the patient may obtain visual benefit from operation.

Diabetic patients have the same range of refractive errors as non-diabetics but there is some evidence that they become presbyopic at a younger age than person in general population. Quite often the presenting sign of diabetes is myopia induced by hyperglycemia. Diabetic patients often state that vision is less clear at certain times of the day because refractive errors change along with blood glucose. So eye glasses should not be prescribed during an acute illness. Diabetics often complain of visual disturbances in association with hypoglycemic episodes. These are not refractive errors but are commonly due to CNS effects of hypoglycemia.

Colour vision:

A possible association between loss of colour vision in patients with diabetes mellitus was first reported in 1905 (Roy et al 1986). The first controlled study of colour vision in diabetic patients was reported in 1972 and 1973 by Knnear and Lahowski and Colleagues who showed in large group of scottish diabetic patients that blue, yellow and blue green colour vision losses were found significantly more among the diabetic patients with retinopathy than in normal controls.
Other more recent studies, using controls (Lombrail et al 1984) and normative data from Belgium (Condit R and Breenik et al 1982-1984) have confirmed that colour vision is significantly altered in diabetic patients with advanced diabetic retinopathy.

However, it still remains unclear whether or not colour vision losses are already present in diabetic patients who have little or no diabetic retinopathy.

Dubois poulson and Cochet and Verriest 1954 to 1964 were first to note, in case reports, and alterations of colour vision in diabetic patients without retinopathy. Thus clinical data shows no significant association between presence or absence of a colour vision defect and age, sex, age at onset or duration of diabetes. Some studies reveal insulin dependent diabetics patients with no to minimal diabetic retinopathy had significantly more colour defects than controls. In all these studies it is important to exclude diabetic patients with any type of lens changes.

**Intraocular pressure in Diabetes:**

It was Heine (1903) and Krause (1904), who first observed a striking hypotony of the eyeball during diabetic coma. Crafe (1924) and Poos (1930) drew attention to the extreme variation in blood sugar levels, considering that these reacted on the intraocular pressure when conditions
for a glaucoma were present. Igersheimer (1944) Philips (1946), Curtiz (1947, 1948), Weinstein (1948) and Larsen (1960) have all stressed how a changing intraocular pressure could as a mechanical factor, initiate or favour the development of diabetic retinopathy.

Armstrong, Daily, Dobsen and Girorod (1960) on the basis of a relatively permanent intraocular pressure of 23.4 mmHg or higher (Schiotz Tonometer) have recently found an incidence of glaucoma in diabetes of at least 6.6% while previously Waite and Beetham in 1935 and reported only 0.5% of clinical glaucoma in 2002 diabetic patients.

The mean intraocular pressure in diabetics is 19.25 mmHg (Arora and Prasad 1983) which is higher than the normal mean intraocular pressure reported in general population i.e. 16.1 mmHg (Becker and Schaffer). While in Juvenile diabetics the mean intraocular pressure through lower 17.93 mmHg than the mean intraocular pressure in maturity onset diabetes mellitus, was higher than the average normal mean intraocular pressure. However, Palmar (1935) and Armaly and Bologlous (1967) observed low intraocular pressure in diabetics than non-diabetics.

Intraocular pressure in reaction to different grades of retinopathies. Christiansons (1960) studied 172 diabetic
presence between 12 - 50 years of age, the aim was to observe
the reaction of diabetic disease on the pressure of eye,
while ignoring as far as possible the influence of age. He
found that the diabetic retinopathy was set up only in
45.6% cases, a similar co-relation was found by Kornurup
(1955) i.e. 48.6%. He also reported that the diabetic eye
have a higher intraocular pressure than a corresponding
normal eye. Insulin treatment seems to have but little
relevance as regards this difference in tension, with increas-
ing retinopathy, difference in tension is accentuated.

Christiansen found that in grade I retinopathy
IOP was 16.1 mmHg on an average mean, in grade II, the
tension decreased to 12.3 mmHg and in grade III still
decreased to 9.3 mmHg. He also reported eye with diabetic
retinopathy of grade IV have lower intraocular pressure than
other retinopathic group and the co-efficient of facility
of outflow rises.

Igershemier (1944) suggested that ocular hypotony
in diabetes might play a role in development of diabetic
retinopathy. It has also been demonstrated that diabetic
retinopathy often progress rapidly in such ocular hypotonic
states as pregnancy and after eye operations (Lieb WA 1967,
Radan et al 1968).
A number of studies have pointed out a greater occurrence of proliferative retinopathy has been appreciated in diabetics with consistently low intraocular pressure than those with elevated intraocular pressure (Igersheimer 1944) or conversely a greater occurrence of proliferative retinopathy is extremely rare in diabetic individual with primary open angle glaucoma.

Topical corticosteriods have been demonstrated to induce increase intraocular pressure in human eyes. The degree of pressure response was genitically transmitted in Mandelian fashion of particular interest was close relationship between this genitically determined response and primary open angle glaucoma.

Becker and Khan (1964), suggestions have been made that elevated intraocular pressure might be used to prevent the patients with diabetes from developing proliferative retinopathy.

**Diabetic Retinopathy**

Diabetic retinopathy first described by Von Jaegar in 1855 is one of the major tragedies of ophthalmology, in our present generation it is predictable but not preventable chronic and progressive in its course and leading to blindness in distreting percentage of cases. Desmarres (1856) AV Graefe (1859), T Leber (1875), S McKenzie and E. Nettleship (1877) J. Mirschberg 1890 and many others described the clinical
In the insulin era after 1923, the relation between the clinical picture and the morphologic findings in diabetic retinopathy were studied by Ballantyne and Lowenstein (1943), Friedenwald (1943), Ashton (1949), Thiel (1956), Cogan, Kuwabara, Toussaint (1961), Wolter, Bloodworth (1961) Fatz and Maumence (1962).

Diabetic retinopathy has been divided into three stages according to fundus changes seen after dilation of pupils (Lloyd, Aiello et al 1978).

Stage I:
Background retinopathy which includes presence of microaneurysms with or without small dot & blotch hemorrhages, hard exudates and fewer than five soft exudates, minor venous abnormalities characterized by irregularities in the width of veins, sheathing of veins, tortuosity of veins, arteriolar narrowing, arteriovenous nicking, retinal edema.

Stage II:
Preproliferative diabetic retinopathy which includes presence of cotton wool spots more than five, venous beading and duplication, intra retinal microvascular abnormalities. Areas of nonperfusion or capillary closure, macular edema in young patient.
Stage III:

Proliferative diabetic retinopathy which includes -

i) Vasoproliferation new vessels on the disc or elsewhere fibrous tissue membrane.

ii) Fibrous growth stage with contractile leading to retinal haemorrhage, vitreous haemorrhage, tractional retinal detachment.

Makensie and Nettleship (1877) were the first to discover capillary aneurysms in a case of Glycosuria. Ballantyne and Loewenstein (1943) succeeded in proving that the earliest sign in diabetic retinopathy is the microaneurysm. These seen ophthalmoscopically are small size round in shape, sharply defined and sometime show a light reflex which iridicate spherical form, these may be confused with punctate haemorrhage which are not globular but petechial in shape. The microaneurysms can be found in any part of fundus but when few usually near the macula. They often seem to be attached to five perimacular vessels with line. These undergo sclerosis or complete hyalinisation with or without thrombosis leading to formation of opaque scar, which appears as surrounded by a fank narrow halo or white spot.

Haemorrhages, most characteristically occuring in the deeper layers of retina and hence round and regular in
shape and are also a relatively early feature of diabetic retinopathy (Ballantyne and Lowenstein 1943). These may occur due to rupture of microaneurysms which are at level of ganglion cell layer and punctate shape. These are grouped under dots and blots.

The exudates usually situated in intranuclear layer but later breaking down barriers between these space and forming compact masses waxy patches. These differ from that of hypertensive exudates which is fibrinous, large patch granular appearance and of a silvery grey colour while of diabetics the exudates are of fatty or lipid substance, smooth hamogenous texture of hyaline or fatty deposits.

Hard exudates are more common. As regards the retinal vessels their contest clinical change is probably a general fullness of the larger veins (Ballantyne and Cowanster 1943, Michaelson 1949).

Phlebosclerosis formation of loops and cells are new built preretinal vessels O' Brien and Allen 1940, Philips 1946 are pathogenomic of diabetics. Network of capillaries Retemirabile which occur in all conditions characterized by circulatory stasis in the retina are common in diabetics.
The pre retinal vascular proliferation may remain there or regress leaving a vitreo-retinal scar, frequently, however, it invades the vitreous space either through gaps in the cortical vitreous (Tolentino et al 1966) or else, because it is pulled forward by a shrinking vitreous (Davies 1966). The proliferative phase of diabetic retinopathy is a true neovascularisation with the accompaniment of connective tissue (Dobree 1968). These new vitreal vessels arising from the intraretinal vessels do not have intramural pericyte cells (Hogan 1967). The contraction of the connective tissue may give rise to retinal detachment (Dobree 1968). Proliferative retinopathy is found in about 2 to 16 percent of diabetics (Wilson et al 1957; Postmann 1954; Engelson 1954; Scott 1951-53; Kornerup 1958).

Fluorescein photography helps to understand the pathological process, by which we can demonstrate capillary dilatation disturbance of normal capillary pattern which are among the earliest sign of diabetic retinopathy (Kohner 1967).

The development of disturbance of microaneurysm may take place in less than a year (Keen and Smith 1959). Exudates and haemorrhage may also come and go.
Incidence of Diabetic retinopathy:

The general increase in diabetes mellitus is high for its affects between 1-4 to 1.7% of population. It occurs particularly in people in fifth and sixth decade of life. There has be continous increase in the incidence of diabetic retinopathy in past few decades.

Wagenar figure of this point are; in 1921 immediately prior to the introduction of insulin, found an incidence of 8.5% of diabetic retinopathy among diabetics (Wagener and Wilder 1921). In 1934, the incidence has risen to 17.7% (Wagener et al 1944) and in 1945 - 29.6% (Wagener 1945). Typical figures based on large number of patients are those of Kornuruf (1957) who found 601 cases of diabetic retinopathy in 1285 unselected diabetics (47%) and Dollfus (1945) 681 cases in 1,303 patients (52.4%). It follows that, at present time diabetic retinopathy may be expected to develop atleast in 50% of all the cases of diabetes. In evaluating the statistics one has to consider the facts that until the introduction of insulin in 1921 many patients died before the occurrence of retinopathy, while thereafter, they continued to life to develop retinopathy and also that examining techniques are much more precise today than they were years ago. This is due to the improvement of ophthalmoscope and slit lamp and fluorescein angiography.
Diabetic retinopathy in relation to the age:

The first report of diabetic retinopathy by V Jaegas 1954 was based on the finding in a 22 year old, gardener. However, most workers have stated that retinopathy is common in patients of middle age group or advance age and rare in younger people and extremely rare below the age of 10 years whatever the duration of diabetes may be (Forsyth and Payne, 1956, Imerslund 1959, Girner 1960, Guest Lample Kessler and Skillman 1965).

Diabetic retinopathy in relation to duration of diabetes:

The duration of diabetes is the most common single factor for the causation of diabetic retinopathy. There is general agreement that the prevalence of retinopathy in the diabetic population is positively associated with the duration of diabetes. In the recent study by Kahn HA and Brodley RW 1975), the prevalence of diabetic retinopathy among patients at Joslin clinic, was 25% in total diabetic population, 7% in patients with diabetes for less than 10 years, 26% in patients with diabetes for 10 to 14 years, and 63% in patients with diabetes for 15 years or more. Retinal changes are rarely seen until the diabetic has been in existence for 3 years, a fact confirmed by all observers (Waite and Beetham 1935; Wagener 1945; Friedenwald 1950; Lawrance et al 1951; Gardes 1953; Scott 1953 and many Others). Patients who develop diabetes before the age of 15 or 16 show a frequency of 10% or less after 5-9 years of diabetes, of
50% after 15 years or so and 80-90% after 26 years or more. Lundbeck (1955) on the initial examination of 246 recently diabetics, in whom the disease had presumably developed above the age of 40 years, found retinopathy present in only 4% whereas Dollfus and Haige (1953) found that 90% of diabetics of over 18 years standing had retinopathy and Dolger 1947 examining cases of 25 years standing, concluded that not a single case had escaped.

Cristiansson's (1961) reported 45.6% of the patients had some form of retinopathy in an average diabetic duration of 16.7 years, a frequency that is in close agreement with the figure given by Kornurup (1955), namely 46.8% in large patients. On the other hand patients with proliferative retinopathy Grade III and IV (Ballantyne 1946) in Christianssens study shows 17.5% of the total case material with an average diabetic duration of 16 years the corresponding figure of Kornurup (1958) are 8.4% with the duration of 16.1 years the higher frequency in Cristianssons study is due to age limit being fixed to 50.

But it is of interest to note that not all the diabetics with long duration of diabetics exhibit the retinopathy. Kinsell (1955) reports that Joslin was able to reward 45 patients with "Quarter centuary victory model" in that they exhibited no signs of "Late diabetes syndrome"
even after 25 years of duration of diabetes. According to Friedenwald (1954), there are about 15-20% of diabetics who do not show diabetic retinopathy after 20-25 years of diabetes.

Khosla et al (1964) found that more severe form of retinopathy was seen as the duration of diabetes increased (especially after 10 year period) but contrary to the usual, the prevalence of diabetic retinopathy is related to the duration of diabetes.

**Sex incidence of Diabetic retinopathy:**

Braun (1937), Hanum (1988), Heinsnun (1939) and many others concluded that diabetic retinopathy is more common in females than males. Duke Elder describes that females-male ratio is 3:2, and also that women are more liable to develop retinopathy. Proportion of female-male ratio with diabetic retinopathy is about 4:3; while in some other studies there is much greater incidence among females; Hanum (1938), for e.g. in 183 cases of diabetic retinopathy found 73% females and 28% males. The larger statistics of Postsmann and Wiese (1954), Keiding et al (1952), Janort et al (1956) and Babel and Rilliet (1958), however did not show a difference between the sexes.
Frequency of Retinopathy and severity of the disease:

Retinal lesions are observed in mild as well as severe cases. Hanum (1938) found retinopathy to be most common in diabetics of mild to moderate severity. Scott (1957) also believes that retinopathy is more common in light diabetes. Donoghass and Drury (1954) in contrast to this found retinopathy to be more common when more insulin was required to control the diabetes. Mohmike also shows that retinopathy occurs earlier and more commonly in severe cases. Waite and Beetham (1935) however, finds no relationship at all between the frequency of retinopathy and the severity of disease.

Effects of control of diabetes on retinopathy:

The clinical and experimental evidence suggests that good control of metabolic aspects of diabetes delays the onset and decreases the severity of retinopathy to prove this prospective study of metabolic control has been performed in experiment with animals (Engerman, Bloodworth 1973). They found that poorly controlled group developed retinopathy while striking reduction in incidence and severity of retinopathy in well controlled group.

Diabetic retinopathy in relation to tyr- of diabetes:

The familiar text book classification of diabetes into 'Juvenile onset' and 'maturity onset' has now been
largely abandoned by diabetologists, the classification adopted by the Americans Diabetic Association and W.:...C. divides the majority of patients into type I (Insulin dependent diabetes mellitus (IDDM)) and type II (Non-insulin dependent diabetics mellitus (NIDDM)). Abundant evidence show that these two have entirely different eitiopathogenesis. Majority of type I patients develop the disease in childhood or in adolescence, but it is by no means confined to this age group. Type II predominantly affects the adults life. Etiology of type I is immune mediated destruction of Islet B cells, while etiology of type II remains mainly unknown.

In Juvenile diabetes, retinopathy rarely occurs before 16-18 years of age (Larson 1960). In patients, who were under 20 at the time of diabetes was discovered, the interval between this and visual loss was on an average 17.4 years (Jertz and Berkov 1968).

Above 20% of Juveniles with diabetes show changes in fundus (Chylinska and Abramowiez 1969) Darnaud et al 1963). The proliferative form appears in about 10% of cases of Juvenile diabetic retinopathy. Prognosis in these cases is poor, about 50% having less than 6/60 vision in both the eyes after 5 years (Deckort et al 1967). This type of Juvenile diabetic retinopathy appears between 10-14
years of age, whole simple or non proliferative type does not appear until 16-29 years (Michaelson 1980). The simple form of retinopathy appears in less than 10% of cases if diabetes has lasted for less than 10 years period; but it is found in at least 70% after 20 years of diabetes (Knowles 1965). Kohner (1977) has suggested that there is an association between proliferative retinopathy and 'Juvenile onset' diabetes and between diabetic maculopathy and 'Maturity onset'. Bodansky, Cudworth, Whitelock and Dobree (1982) also confirms this view. They also report association between male sex and proliferative retinopathy.

The reported incidence of retinopathy in Juvenile onset diabetes is greater than in adult onset group because the patients live long enough for the retinopathy to develop. The highest percentage, 80% occurred in cases of more than 15 years duration of diabetes. The onset of retinopathy in 'Juvenile diabetes' occurs after at least 6-7 year (Borta and Molnar 1970). Terne (1972) reported retinopathy in 67% of cases of Juvenile onset diabetes compared to 43% of adult onset diabetes.

Neuro-ophthalmic association of diabetes:

Diabetes can be the cause of extraocular muscle palsy involving the 3rd (oculomotor), 4th trochlear and 6th abducens cranial nerves. The hallmark of diabetic nerve palsy is that they resolve the recovery or begining of recovery in a few weeks to 4-6 months. Any nerve palsy
which does not show signs of recovery in 6 months may well not be diabetic origin (Brude et al 1985).

The association of optic nerve hypoplasia in a child with a diabetic mother was first noted by Peterson and Walter (1977) and then elaborated by Nelson Elessell and Sadum (1986). The quartet of diabetes mellitus, diabetes insipidus optic atrophy and deafness is referred to Wolfram's syndrome (DIDMOAD) is commonly seen in juvenile diabetes. Lessell and Rosman (1977), Wolfram (1938). An increased occurrence of diabetes mellitus 20% has been noted in patients with anterior ischemic optic neuropathy of the non arteritic variety. Guyer (1985), Ellenberger (1973), Repka (1983).

Burde (1985) used term vasculopathic mononeuropathies involving 3rd nerve with pupillary sparing Rucker (1958), Goldstein (1960). Bell's palsy may precede a diabetic ophthalmoplegia in about one third of cases by several months to years Jaffe (1967).

Patients with diabetes mellitus are known to develop infections processes including orbital cellulitis characterized by proptosis, periorbital swelling and ophthalmoplegia Burde (1985), Rootman J and Lippincott, JB (1988).

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