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Ocular manifestations of Pregnancy Induced Hypertension

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Abstract

Pregnancy Induced Hypertension (PIH) can have grave consequences for both mother and foetus. Ocular involvement is common in majority of cases of PIH. Most common symptoms are blurring of vision, photopsias, scotomas, and diplopia. Ocular involvement includes conjunctival vascular anomalies, hypertensive retinopathy, exudative retinal detachment, vitreous and pre-retinal haemorrhages, ischemic optic neuropathy and hypertensive choroidopathy. This review discusses the ophthalmic manifestations of PIH with special emphasis on retinal changes. The ocular vascular changes have been said to correlate with the severity of hypertension and ophthalmic examination is very helpful for evaluation, diagnosis and prompt management.

Keywords: *pregnancy Induced Hypertension (PIH), pre eclampsia, eclampsia, ocular manifestations, fundus changes, ophthalmoscopy*

Ocular manifestations of Pregnancy Induced Hypertension (PIH)

Introduction

Pregnancy Induced Hypertension (PIH) is a challenging stigma in the field of obstetrics and one of the major contributors to maternal and perinatal mortality.[1-7] PIH is classified into Mild PIH, Pre-eclampsia, and Eclampsia.[1-3] Mild PIH is defined as blood pressure of 140/90 mmHg which returns to normal by 12 weeks postpartum. Pre-eclampsia is the presence of hypertension (BP>140/90mmHg) on two occasions with spacing of four hours and significant proteinuria (>300 mg per 24 hrs) and/or edema. Eclampsia is the occurrence of convulsions or coma unrelated to other cerebral conditions, with signs and symptoms of pre- eclampsia. Ocular involvement occurs in a majority of patients of PIH.[5-7] Ocular examination of PIH patients not only helps in diagnosis of eye problems but repeated observations assist in assessing the severity and progression of disease, response to treatment and ultimate outcome or prognosis.[3,5-7] This review discusses the ophthalmic manifestations of PIH with special emphasis on fundus examination. Ophthalmoscope can easily detect the pathological changes in the fundus and the retinal blood vessels in particular that reflect the effect of hypertension throughout the vascular system.[5-9]

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Fundus changes in Pregnancy Induced Hypertension (Clinical Manifestations in PIH)

The clinical course of fundus changes in PIH may be divided into three stages:

(i) Spastic stage: Characterised by spasm of retinal arterioles.[5-9]
(ii) The stage of sclerosis: When pregnancy induced hypertensive changes are superimposed on pre-existing organic sclerotic changes in the vessels.[5-9]
(iii) The stage of retinopathy: Characterised by cotton wool spots, micro aneurysms, flame shaped and splinter haemorrhages, hard exudates, disc edema etc[5-12].

PIH as studied by ophthalmoscopic examination manifests the following characteristics:

Spasm of retinal arterioles

The first change observed in normal retinal arterioles is constriction of the lumen. In some patients this constriction may be localized to a single point, or there can be series of these localized constrictions; some elongated and spindle shaped and usually limited to the first half of the retinal arteriole. Usually the patients showing generalized arteriolar constrictions also exhibit varying degrees of localized constriction. The addition of localized spastic constriction indicates an active progressive disease. The arteriolar constrictions are proved to be spastic in nature when the degree and location seem to vary at subsequent examinations.[5-9] Many patient were examined a few days after delivery when the blood pressure had returned to normal and the formerly constricted anterioles had resume their normal calibre.

Initially angiospasm of the superior nasal arterioles occurs. Sometimes the spasm may start at the periphery and spread towards the disc, becoming generalised. Sometimes fleeting spasm also occurs. Fleeting spasmodic contraction sometimes obliterates lumen of vessels and gives rise to symptoms of amaurosis fugax.[13] Thus, angiospasm is important for diagnostic as well as prognostic purpose. Early detection of angiospastic changes and prompt management, may prevent the onset of more serious retinopathy and generalised progression of the illness.[3,5-9]

Arteriolar Spasms Superimposed on Arteriolar Sclerosis

The main retinal evidences of vasosclerosis associated with PIH are as under:

Three major signs:

(i) Arterio-venous crossing changes
(ii) Focal vascular narrowing
(iii) Generalised attenuation sometimes associated with straightening of the arterioles.

There are other signs of less clinical significance when found in isolation but which may be of value as supporting evidence:

(i) Reduced translucency of vessel walls
(ii) Changes in vascular reflex
(iii) Sheathing.

Arterio -Venous Crossing Changes

In majority of cases, arterioles cross the venules anterior to it. These crossings occur at many places in the fundus and hence are relatively easy to observe.[5-9] In arterial disease, two prominent pathological changes are:
(i) Gunn's sign – an apparent compression of vein
(ii) Salus's sign – deflection in the course of the vein

Compression of the veins (Gunn's sign): On the peripheral side of the crossing, the vein appears to be dilated and swollen as if circulation was, to some extent, impeded; a phenomenon known as "banking"; and occassionally aneurysm – like swellings occur. While in other cases an hour- glass constriction is evident, a dilatation appearing both before and after the crossing.[5-9]

Deflection of the veins out of their normal course (Salus's sign): The arterioles and venules which normally cross at an acute angle, now cross in at an obtuse angle. These phenomena may be combined to form a picture usually described as A-V nicking.[5-9]

Focal vascular narrowing: The appearance is often seen in normal vessels as they emerge from the disc so that the sign should not be considered positive unless it occurs at least half a disc diameter from the edge of the disc.[5-9]

Generalised attenuation and straightening of the retinal arterioles: A generalised narrowing of the retinal arterioles so that they appear much more attenuated than the veins is a sign of extreme value in the diagnosis of both a tonic contracture and also a generalised arteriolar sclerosis. Narrowing of the veins also occurs but is not so much in evidence. There is a significant relationship between the narrowing of the calibre of arterioles and the height of diastolic pressure.[5-9,14]

Translucency of the vessel walls: In the normal eye the translucency of the vessel walls is so nearly perfect that they are invisible but in hypertensive state this property disappears.[5-9]

The vascular reflex

The vascular reflex, which appears as a thin longitudinal bright streak running along the convexity of an arteriole, is due to light reflected partly by the blood column and party by the vessel wall in which the main reflecting element is the media. In sclerotic conditions it is of course the latter element which alters. It may be said, however, that in sclerotic conditions associated with PIH the usual uninterrupted bright streak may suffer significant changes as under:[5-9]

(i) Copper – wire arterioles

There is increase in the brightness of the reflex of the retinal arterioles so that instead of their normal rosy appearance with a thin central bright line, they assume a burnished metallic appearance with broader and softer reflex resembling a copper wire.[5-9]

(ii) Silver - wire arterioles

In more advanced cases, as a result of the still higher refracting power of the arterial wall, the entire vessel reflects light homogenously so that the whole thickness of the artery appears as a bright white reflex like a wire of silver without any evidence of a red blood column.[5-9]

Sheathing of the vessels

In this phenomenon white lines first appear running alongside the walls of vessels (parallel sheathing) and in most extreme cases, the entire vessel is converted into a white fibrous looking cord (pipe-stem sheathing).[5-9] It is important to remember that such an appearance frequently occurs in normal and youthful eyes on the disc and on the retina in its immediate neighbourhood and that its presence is not closely related to age or hypertension. In sclerosis the ensheathing white lines appears first at arterio-venous crossings where the sheaths of arterioles and veins reinforce each other or alternatively, when the same reinforcement is accomplished by a arteriole and vein running closely in parallel, but in more advanced conditions all the vessels may be uniformly ensheathed – a serious pathological feature. In all cases, however, the effect is due to a greater optical density of the vessel wall so that it reflects sufficient light to be demonstrated by the ophthalmoscope.

Aneurysms

Aneurysms of the retinal arterioles are usually of miliary type, they occur particularly in malignant hypertension.[11,15] Larger aneurysms usually appear on smaller arterioles; especially at arterio venous crossings or between localized points of constrictions in the vessel. Aneurysm – like dilatation of venules are common at arterio-venous crossings, but they may occur elsewhere, particularly in small dilated collateral veins or in terminal venous twigs.

Micro aneurysms: Microaneurysms may occur as a part of hypertensive retinopathy, particularly around cotton-wool spots.[10,11,15] While most commonly found in diabetics; they occur next most frequently in patients with hypertensive retinopathy.[15] Aneurysms are often difficult to see. They may be most clearly defined by fluorescein angiography.[16] At this juncture if the pregnancy is allowed to proceed the retinopathy of PIH becomes obvious involving the appearance of edema, exudates and haemorrhages

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which eventually make up the clinical picture typical of hypertensive retinopathy.

The stage of retinopathy
As the retinal arterioles become more constricted there are seen signs of retinal ischemia such as edema of retina, haemorrhages and exudates, etc.

A. Edema of retina:
Edema of retina is usually the first sign of involvement of the retina and it generally makes its appearance at the upper and lower poles of the disc and progresses away from the disc along the course of retinal vessels, which is like the general course of the nerve fibres of retina.[5-9,17,18] In the earliest stage of edema of the retina, the portion involved appears milky and on close examination with the very best focus of the ophthalmoscope and with a gentle and slow to and fro rotation of hand holding the ophthalmoscope the surface shows faint striations running in the direction of nerve fibre layers. The appearance of haemorrhages and exudates in the retina completes the picture of retinopathy.

B. Retinal haemorrhages
Haemorrhages are of two types:

(a) Superficial or flame shaped
The haemorrhages are usually in the posterior one third of fundus, and as a rule appear flame shaped indicating that they are in the superficial nerve fibre layer.[5-9,17,18] Superficially in the nerve fibre layer, blood fans out along the striate course. They are usually situated near the disc or a few disc diameters away from it, arising from retinal capillaries. They might be associated with exudates. It is bright red in colour. It disappears within 6 to 8 weeks.

(b) Deep or Dot and blot
Deeper haemorrhages appear more round and blot like but are not as frequent. In the deeper nuclear or plexiform layers the blood is forced together between the structures resulting in a more round or blot like appearance.[5] When these haemorrhages are present a more severe hypertensive state is suggested.[5-9,17,18] Retinal haemorrhage most likely results from impairment of integrity of capillary endothelium, allowing extravasations past the damaged cell structures.[19] It is round in shape, dark red in colour. It is of venous or capillary origin. It persists for longer time period.

C. Exudates
These are of two types

(a) Cotton wool spots – soft exudates
The soft exudates or "cotton wool spots" are the hallmark of the most severe or "accelerated" type of hypertension.[5-10] These exudates are whitish grey patches, usually 1/3rd to1/2 disc diameter in size, lying superficially in the nerve fibre layer.[17,18] They may be single or multiple; and are seen at the posterior pole in the areas surrounding optic disc but usually sparing the macula and peripheral retina. They may be associated with retinal haemorrhages but may occur without it. In hypertensive retinopathy they are associated with fibrinoid necrosis of the terminal and pre-capillary arterioles.[19]

(b) Hard exudates
They are seen as sharply defined white dots which lie in the deeper layers of the retina i.e. the outer plexiform layer or henle's layer near the macula[5-9,17,18] They are common in the area between the optic disc and the macula. When it surrounds the macula, it may form a partial or complete star figure known as "macular star" or "macular fan". Hard exudates do not arise in the positions occupied by pre-existing soft exudates or haemorrhages. Microscopically they consist of collection of macrophages which are filled with lipid material.

D. Papilloedema (hypertensive optic neuropathy)
In case of severely increasing blood pressure and persistent angiospasm, papilloedema occurs.[5-9,12,15] Initially the upper and lower poles are involved due to density of nerve fibres and then the remaining part. The oedema of the disc may extend into surrounding retina.

Retinal detachment
Retinal detachment is one of the most dramatic and potentially serious ocular complications of PIH.[20-26] Retinal detachment involves sepration of the neurosensory retina from the retinal pigment epithelium. Spontaneous resolution occurs within few weeks. Ischaemic infarcts of choriocapillaris and retinal separation are correlated.[21,22] Choroidal dysfunction, primarily choriocapillaris ischemia, is the underlying mechanism which leads to break down of blood retinal barrier and compromised fluid transport by the RPE, accumulation of subretinal fluid and consequent serous neurosensory detachment. Retinal striae may be the first sign, followed by focal accumulation of sub-retinal or sub-pigment epithelial exudates at the posterior pole. Soon after delivery, spontaneous retinal reattachment occurs and the subretinal fluid is reabsorbed by the RPE and visual acuity should return to pre-detachment levels within weeks. Resolution of retinal detachment is accompanied by an alteration in the form of irregular focal areas of hyper and hypopigmentation, which correspond to the choriocapillaris ischemic stroke-Elschnig's spots. These are non progressive pigmentary disturbances, if discovered later in life, may be mistaken for heredomacular dystrophy.[23]
The development of retinal pigment epithelial tear has been reported after pregnancy complicated by PIH and abruptio placentae.[24-25] Cases of optic atrophy following detachment have also been reported.[5-7,12,26] Spontaneous vitreous haemorrhage and bilateral retinal neovascularization regressing after delivery have been reported in PIH with clotting abnormalities.[27-30] Retinal Detachment should be differentiated from circumscribed neurosensoryretinal detachment of the posterior pole - Central Serous Chorioretinopathy (CSR) associated with pregnancy.[31]

Choroidal Vascular Changes in Pregnancy Induced Hypertension

In accelerated hypertension arteries and arterioles of choroid undergo fibrinoid necrosis, a process that also occurs in other parts of the body.[19] This results in patchy non perfused areas of the choriocapillaries- hypertensive choroidopathy.[21,22,29] The overlying retinal pigment epithelium appears yellow (focal ischemic infarcts) in the acute phase and with time becomes irregularly pigmented with depigmented halos (elschning's spots), usually one sixth to one third disc diameter in size.[22,23] They are typically seen in the mid periphery and in vicinity of optic disc.

Extra Retinal Changes in PIH

Visual disturbances ranging from slight blurring of vision to blindness frequently accompany pre-eclampsia. Visual symptoms such as scotoma, diplopia, dimness of vision, photopsias and cortical blindness have been reported.[5,6,32] Visual disturbances may be precursors of seizure.[33]

The changes are:

Eyelids: Blepharoptosis has been described in patients with severe eclampsia.[34] The blepharoptosis is thought to be of aponeurotic type caused by raised interstitial fluid and hormonal levels.[35] Horner's syndrome is more likely to develop during epidural analgesia during induction of labour in specialized circumstances.[36]

Conjunctiva: Constriction of conjunctival arterioles can occur in PIH.[37] This vasospasm may be mild or may be severe enough to cause local ischemia. Occasionally thrombi can be seen in conjunctival vessels and petechial haemorrhages have been noted in PIH.

Subconjunctival haemorrhages have been reported in patients during labour related to Valsalva's maneuver.[37]

Cornea: The curvature of cornea increases during pregnancy.[38] The curvature returns to normal after cessation of breast feeding.

The most probable etiology of this increase in curvature is development of corneal edema under hormonal influence.

Corneal senstitivity has been reported to decrease during pregnancy. The decrease in sensitivity is due to corneal edema that develops during pregnancy.[39]

Aqueous humor dynamics: The facility of aqueous outflow is increased during pregnancy and it is this factor that is considered to be the major contributing factor for ocular hypotension.[40]

Pupil: Pupillary mydriasis and nystagmus have been described in patients with eclampsia.[41-42]

Lens: No structural change in the lens have been reported during course of pregnancy till date.[5,6]

Field of vision: Some studies report bitemporal contraction; some studies concentric contraction and some studies find no change in most patients.[43]

Amaurosis in pregnancy induced hypertension: Scotomas are usual manifestations of extensive primary, generalized vascular disease or sometimes may be due to multiple cysts like in retinal detachment.[20] They are not common. Some patients with eclamptogenic toxaemia note dimness of vision after coma clears. This usually lasts less than a week. Rarely spots before the eyes, difficulty in reading, diplopia are symptoms complained by patients of toxaemia of pregnancy.[6-8]

Fleeting angiospasm is the basis for these symptoms.[13] These symptoms may last up to 48 hours. The standard methods in treatment of central artery occlusion have no beneficial effects on sight and alleviation can only result from lowering of blood pressure on delivery of foetus.[3,5,44,45] Permanent blindness rarely follows pre-eclampsia. When it does occur, blindness may be the result of primary ocular or cerebrovascular lesion.[46,47] Termination of pregnancy in majority of uncomplicated cases is usually followed by good results, in early cases with a rapid resolution of retinal changes and frequently complete visual recovery. A certain degree of optic atrophy or permanent macular change causing visual loss may remain as a lasting disability in this category.[12,23,48,49]

Other Complications

Other complications reported in literature are proliferative retinopathy, choroidal infarcts, papillophlebitis, retinal artery and vein occlusion, ischemic optic neuropathy, optic atrophy, optic neuritis, thrombosis of the central retinal artery, reversible cortical blindness, extra ocular muscle palsy and purtscher like retinopathy.[5-7,12,,22,26,28,32,33,44-50] The ocular vascular changes in PIH have been found to correlate with the severity of hypertension and this has been used as an indicator to predict adverse feto-maternal outcomes and for termination of pregnancy.

Conclusion

In PIH, ophthalmic examination is very helpful for evaluation of feto-maternal well being and for diagnosis and prompt management.

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