SAMPRAPTI VIVECHANA OF CENTRAL RETINAL ARTERY OCCLUSION

Shetty Hemachandra  Amarnath H. K.  Ashwini M. J.
Dept. of Shalakya Tantra, SDM College of Ayurveda and Hospital, Hassan, Karnataka, India

ABSTRACT

Central Retinal Artery Occlusion (CRAO) is one among the major causes of painless loss of vision. It needs acute and intense treatment, otherwise the damage will be irreversible. Review of such a case in Ayurvedic treatise may not yield any specific answer and may usually end up in diagnosing it as Animitta Linganasha. So, it is a real challenge both to evaluate and to plan an effective management. This review is based on clinical presentation, pathogenesis, and prognosis of CRAO and aims to evaluate the involved Samprapti (pathogenesis), Sadhyasadhvata (prognosis) and thus plan an effective treatment.

Keywords: Central Retinal Artery Occlusion (CRAO), Timira, Linganasha, Tejojalashrita patala, Ayurveda, Shalakya

INTRODUCTION

Central Retinal Artery Occlusion is one of the major causes of sudden painless visual loss. It may be unilateral or bilateral. Ischemia following arterial occlusion causes retinal hypoxia and persistence of this state more than 90min can initiate death of retinal photoreceptors, causing visual loss.¹ Such a loss is almost always irreversible unless the management is acute and intense. In most of the instances as there is no noticeable causes for visual loss it usually falls into the category of Animitta Linganasha (visual loss without cause). Though such a case falls into the category of asadhyya vyadh, attempts are to be made to retain the vision. Moreover critical derivation on such a condition can save time and an acute care within Ayurvedic purview may be geared up. This can only be made on understanding its samprapti (pathogenesis) in authoritative way. As a specific pathology is not explained in Ayurveda, modern pathology can be analyzed under Ayurvedic concepts using diagnostic advancements as footsteps and an attempt is made to understand underlining pathology.

Blood supply of Retina

Retina is extremely metabolically active neural tissue with highest Oxygen consumption in comparison with any other body tissue. In humans, retina has dual blood supply. Inner 2/3rd is supplied by Central retinal artery, outer 1/3rd by choroidal circulation. Considerably choroidal circulation has high flow rate (150mm/sec) with low Oxygen exchange in contrary to retinal circulation with low flow rate (25mm/sec) and high Oxygen exchange.¹

Central Retinal Artery

Central retinal artery arises from ophthalinic artery in optic canal about 1cm behind the Eye. It pierces infero- medial aspect of Optic nerve, passes forward in the centre. Then it enters the papilla through constriction at lamina cribrosa. This constriction is the potential site for partial or complete occlusion.³ Then it branches dichotomously superior and inferior, each further subdivided into nasal and temporal branches. These major four
branches are functional end arteries as they supply a sector of retina and no overlap or anastomosis.²

Central Retinal Artery Occlusion

It is the occlusion to the arterial flow at its anatomical constriction at lamina cribrosa. This occurs because of an embolus obstructing the flow and by super added spasm.³This can occur with or without general arterial disorders like atherosclerosis, hypertension, Burgers disease, and diabetic mellitus. Hence immediately there will be no flow to further arias of arterial supply. This leads to sudden and complete ischemia, tissue death and complete visual loss. In some cases it may produce premonitory obscurations, as the embolus dislodges forward, branch retinal artery occlusion may set in affecting a sectoral loss.³

Fundal findings

On fundal examination there will be extensive cloudy retina, resulting from edema that corresponding to area of ischemia. Larger arteries become thinner, thinner becomes invisible. Fovea looks like a cherry red spot reflecting underlining normal choroidal circulation.⁴,⁵

Fundal Fluorescence Angiogram (FFA)

Key observation in case of CRAO is prolonged arm to retina circulation, more than 1min.⁴This will be shown in delayed arterial filling indicating the obstruction in arterial flow. Masking of Choroidal fluorescence by retinal edema may be seen but venous phase will be normal.⁵

Timira samprapti

While explaining the timira samprapti, Acharya Laghu Vagbhata has followed the anatomical description of patala⁶ (~coats of Eye). He says, in Eye, doshas initially invade outer Tejo-jalashrita patala and move inwards involving pishithashrita, medashrita and finally the asthyasritha patala. This view of timira samprapti may not be acceptable as it is not supported by any other Ayurvedic treatise. A contrary view is stated by Acharya Nimi, Sushruta, Vruddha Vagbhata and commentators of various samhitas, who opined the movement of vitiated doshas through upward channels and its localization from abhyantara to bahya akshi patalas.⁷In other words, anatomically Tejo-jalashritha is the first patala, while inverse is true with timira samprapti. Hence, Asthyashrita patala will be the initial occupancy of vitiated doshas, while Tejo-jalashrita (Rasa- Raktashritha) will be the last.⁸ Prognosis of any eye diseases is based on the number as well as the order of patala involved; involvement of deeper patala makes it difficult to cure. Rasadhutu (Tejo-jalashritha patala) being the deepest occupancy in timira samprapti, thus prove its kruchra sadyatha.

Linga nasha⁹

Linganasha is a condition where loss of visual function is due to doshaja or bahya karanas; or a condition which is later to or an advanced stage of timira. According to Acharya Dalhana, Chaturtha Patala is Tejo-jalashritha Patala and Linganasha is a resultant of chaturtha patala gatha timira karaka dosha.

DISCUSSION

Analysis of the above condition requires detailed knowledge of involved srotas (channel), srotodusti prakara (types of impairment in channels), dosha, dhatu and Agni. Srotas are channels which carries parinamamaapadyamana (in process of transfer into other state) dhatus all over the body¹⁰ and their disturbance may disturb margastha (in the channel) dhatus, sthanastha (presently occupied) dhatus and nearby structures leading to impaired physiological functions. This disturbance can be sequential and produce disease symptoms.¹¹Such an impaired functioning
Invasion of vitiated dosha in CRAO can be established. Hence, fundal changes in CRAO and lakshanas seen in rasa pradosa along with the features of raka kshaya seem to be similar. Hence we can presume the role of rasapradosha and resultant raka kshaya in pathogenesis of CRAO.

While discussing the manifestation of ocular diseases the terms Agninasha and tama mentioned in the context of rasapradoshaja vyadhi are usually overlooked.

Eye is a seat of Agni, known as Alochaka pitta. It helps in transcription of visual perseverance by converting the perceived image into rupa jnana. Any interference for its existence can deplete rupa jnanotpatti. Thus, the phrase Agninasha in rasapradoshaja vyadhi can be rightly taken into visual deterioration or timira of CRAO.

The word ‘tama’ has not been elaborated either by the author or by the commentators. Hence, by observing the meaning, in this instance it can be consider as visual loss or linganasha.

After considering the involvement of dosha (Rasa), and srotorodha prakara (sanga) in pathogenesis of CRAO, it is relevant to discuss doshas involved. Aggravated doshas, obstructed in their movement, are the sole reason in the manifestation of any disease. The lakshanas (signs) exhibited in the manifested disease represents the gunas (quality) of the vitiated dosha and thus lead us to the dosha involved. Hence, analysis of dosha has been carried out by critically evaluating the involved gunas in the clinical signs of CRAO like srotorodha, pandutha (kapha prakopa laxana), sada and linganasha (vata prakopa laxana). When Kapha aggravates in the body it will hinder (rodha) the chala guna and gati (movement) of Vata and also counter the ushna and rowing of vessels) and varna hani (defective hue) may be secondary to srotorodha.

Retinal cell death in CRAO is due to prolonged ischemia. Ischemia causes reduced nutritional influx and hypoxia to retinal cells leading to cell death. It is very essential to understand the role of dhatus especially Rasa and Rakta in this context as these dhatus does preehana (nutrition) and jeevana (life supportive) of shareera dhatu through tiryaggata dhamani (transverse channels). Continuous inflow of rasa and rakta does tarpana of all dhatu, supports their physiological functions and thus does dhatu dharana (to sustain). Hence, retinal cell death due to impaired dhatu dharana karma (loss of poshana and jeevana karma) resulting from ischemia in CRAO can be established.

Invasion of vitiated dosha into Rasa dhatu, leads to rasapradoshaja (impaired functioning of rasa) vyadhi. The pathological changes taking place in CRAO, like obstruction to the arterial flow, narrowing of further arterial course, retinal paleness, loss of retinal cell functions and resultant visual loss, can be now rethought off. On keen observation of rasapradoshaja vyadhisa, symptoms like srotorodha (obstruction in channel), pandutha (paleness), sada (sinking in or narrowing of vessels), Agninasha (impaired function of Agni) and tama (deprived or loss of eye sight) are seen. The lakshanas of Rakta kshaya such as sira shaitilya (nar-
tikshna gunas of Agni (Alochaka Pitta) and hence deplete their function. Thus, Kapha is avarodhaka to functions of both Pitta and Vata; and this is true with features like visual deterioration and reduced blood flow to further areas of arterial course which are seen in CRAO.

It can also be established that Vata dosha has a key role in manifestation of most of the signs seen in CRAO. When CRAO sets in, contraction or super added spasm of blood vessels (sira sankocha) are seen immediate next to the site of obstruction along with thinning of vessels (sada) in their further course. Thus these lakshanas are resultant of Vata prakopa caused by Kapha avarodha as a result provoked Vata loses its normal functions like abhiyodhana karma (to carry / grasp knowledge) and leads to linganasha.

Further, exploring into specific type of Vata involved here, it can be understood from the context of Rasa sargas connection that an evident role is played by Vyana Vata in carrying the essential nutrients to the tissues. Hence, avarodha due to Kapha in Rasavaha srotas can lead to gati hani (sanga) of vyana in turn reduction in Rasa gati to further areas of circulation, hampering Rasa tarpana.

Prognosis of any disease is understood based on the primary seat (dhatu) of the disease, dosha involved and current status of the dhatu. In this context, prognosis of timira may be reviewed by observing the involved dhatu (Rasa and Rakta) and its presenting symptom (linganasha). By the review mentioned above it is clear that Tejo-jalashritha patala is the representation of Rasa Rakta dhatu in Eye and in the timira samprapti it will be the end abode of occupancy causing linganasha accounting for its poor prognosis. Hence established involvement of Rasa Rakta in CRAO, make the disease difficult to cure.

CONCLUSION

By above discussion, we can arrive at samprapti of CRAO in Ayurvedic perspective. CRAO is a disease of Rasa pradoshika. Thus, the srotas involved is Rasavaha, with srotodusti prakara as Sanga. It is a tridoshatmaka vyadh wherein Kapha obstructs the flow of Vata, as a result Vata gets prakupita. The initial symptoms observed are panduta and timira (blurred vision) which can be attributed to Kapha pradhana avastha, where there still exists a chance of reverting the pathology and better prognosis. As the time progresses, Vata take an upper hand and visual loss (linganasga) sets in, which may not be reversible making the prognosis poor. The involvement of Pitta (Agni) can be summarized as loss of physiological function of rupa jnana, due to possible Kapha avarodha. Thus, in protocol of managing CRAO treatment mentioned as per Kaphavrittha Vata with due consideration to Pitta may be adopted.

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CORRESPONDING AUTHOR
Dr. Hemachandra Shetty
PG Scholar of Shalakya Tantra
SDM College of Ayurveda & Hospital, Thaninruhalla, Hassan- 573201, Karnataka, India
Email: drhemachandra1985@gmail.com

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