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RESEARCH ARTICLE

SRUNGATAKA MARMA AND ITS VULNERABILITY – A REVIEW.

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Abstract

The ancient thought about body facts are highly appreciable. There are separate channels for carrying various bio-elements in the body. Blood is a very important entity carried through the Sira. These are pervaded throughout the body. These are even constituting the corpus of marma sthan and also nourish the same. Sushruta has explored most vulnerable 107 areas (Marma point) on the human body and cautioned the surgeons community while operating over these marma points. The traumatic effects of such vulnerable points are beautifully described in Ayurvedic applied anatomy. However, there is a need to understand these vital points in terms of morphology and morbidity. Shrungataka Marma is one among them, situated within the skull, vulnerable for injury leads to instant death or death within seven days. In this article an effort is made to understand this marma in the per view of structures, mortality and morbidity by reviewing the classical, contemporary and published clinical data for substantiation.

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Introduction:-

Marma are vital spots on the body injury of which leads to death or severe deformity or excruciating pain. The corpus of these spots are made up of Mamsa(muscular tissue), Sira (vascular tissue), Snayu (ligaments/ nervous tissue), Asthi (bony tissue) , Sandhi (joint)¹. Depending upon the predominance of these structures, marma are classified. and based on effect of trauma they are differentiated as Sadyapranahar (instant death), Kalantarpranaha(delayed death) , Vishalyaghna (death due to foreign body)Vaikalyakar (deformity) and Rujakar (Painful) and these have their own dimension. Structure and traumatic effect is vary according to region. Shrungatak Marma is one among them, located within the head region. It is constituted by confluence of Sira , trauma of these ending into fatality. The location, structural constitution and traumatic effect of this Marma is need to be understood by applied anatomy and clinical observations

Classical review:-

Shrungataka marma is situated within the confluence of Sira; nourishing the Ghrana(nose), Shrota (ear), Akshi(eye) & Jivha (tongue). These are four in number, incorporated under Sadyapranahara category, measures vicinity of four Angula (8cm) circumferences The corpus of this marma is made up of Sira or Dhamani (vascular entity). On injury of these marma, death will occur immediately or within seven days². The Nasya medicaments are going to reach in the shrungatak area and the Dosha residing in the region of oral cavity (Mukha), nose (nasa), eye(Akshi) & tongue (Jivha) are expelled out³.

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Anatomical review:-

The Vascular structures which are having very close relation with the nose, tongue, ear and eyes to be considered here.

Dural Venous Sinuses:-

Dural venous sinuses are a complex of venous channels which lie between the two layers of dura mater, draining blood from the brain and cranial bones. They are lined by endothelium, have no valves, and their walls are devoid of muscular tissue⁴.

Cavernous sinus:-

The cavernous sinus is a large venous plexus that lies on both sides of the body of the sphenoid bone. The sinus extends from the superior orbital fissure to the apex of the petrous temporal bone, with an average length of 2 cm and width of 1 cm. The sphenoidal air sinus and pituitary gland are medial to the cavernous sinus. The trigeminal cave is near the infero-posterior part of its lateral wall, and extends posteriorly beyond it to enclose the trigeminal ganglion. The internal carotid artery, and associated sympathetic plexus, passes forward through the sinus together with the abducent nerve, which lies lateral to the artery. The oculomotor and trochlear nerves and the ophthalmic and maxillary divisions of the trigeminal nerve all lie in the lateral wall of the sinus. Propulsion of blood in the cavernous sinus is partly due to pulsation of the internal carotid artery, but it is also influenced by gravity, and hence by the position of the head.⁴

Tributaries of the cavernous sinus are the superior ophthalmic vein, a branch from the inferior ophthalmic vein (or sometimes the whole vessel), the superficial middle cerebral vein, inferior cerebral veins and sphenoparietal sinus. The central retinal vein and frontal tributary of the middle meningeal vein sometimes drain into it. The sinus drains to the transverse sinus via the superior petrosal sinus, to the internal jugular vein via the inferior petrosal sinus and a plexus of veins on the internal carotid artery, the pterygoid plexus by veins traversing the emissary sphenoidal foramen, foramen ovale and foramen lacerum, and to the facial vein via the superior ophthalmic vein.⁴

Intercavernous sinuses:-

The two cavernous sinuses are connected by anterior and posterior inter-cavernous and the basilar plexus. The intercavernous sinuses lie in the anterior and posterior attached borders of the diaphragm sellae and they thus form a complete circular venous sinus. All connections are valve less and the direction of flow in them is reversible. Small irregular sinuses inferior to the pituitary gland drain into the intercavernous sinuses. Such inferior inter-cavernous sinuses are plexiform in nature and important in a surgical transnasal approach to the pituitary.⁴

Inferior petrosal sinus:-

The inferior petrosal sinus drains the cavernous sinus into the internal jugular vein. It receives labyrinthine veins via the cochlear canaliculus and the vestibular aqueduct.⁴

pterygoid plexus:-

The pterygoid plexus of veins is found partly between temporalis and lateral pterygoid and partly between the two pterygoid muscles. Sphenopalatine, deep temporal, pterygoid, masseteric, buccal, alveolar (dental), greater palatine and middle meningeal veins and a branch or branches from the inferior ophthalmic vein are all tributaries. The plexus connects with the facial vein via the deep facial vein and with the cavernous sinus through veins that pass through the sphenoidal emissary foramen, foramen ovale and foramen lacerum. Its deep temporal tributaries often connect with tributaries of the anterior diploic veins and thus with the middle meningeal veins.

Veins from nose:-

Veins from the posterior part of the nose generally pass to the sphenopalatine vein that runs back through the sphenopalatine foramen to drain into the pterygoid venous plexus. The anterior part of the nose is drained mainly through veins accompanying the anterior ethmoidal arteries, and these veins subsequently pass into the ophthalmic or facial veins. A few veins pass through the cribriform plate to connect with those on the orbital surface of the frontal lobes of the brain. When the foramen caecum is patent, it transmits a vein from the nasal cavity to the superior sagittal sinus⁵. The veins of the hard palate accompany the arteries and drain largely to the pterygoid plexus.⁶ The veins of the soft palate usually drain to the pterygoid venous plexus⁷

Veins from ear- veins associated to external ear drain into the external jugular and maxillary veins and the pterygoid plexus.⁸ middle ear terminate in the pterygoid venous plexus and the superior petrosal sinus.⁹ Inner ear through labyrinthine vein, which ends in the posterior part of the superior petrosal sinus.¹⁰

Veins from eyes -the veins which drain the eyelids are larger and more numerous than the arteries. They pass superficially to veins either on the face and forehead, or deeply to the ophthalmic veins within the orbit. Bulbar conjunctival veins pass to the orbital surfaces of the rectal muscles and join the superior or inferior ophthalmic vein.¹¹ The central retinal vein entering the cavernous sinus or the superior ophthalmic vein¹².

Review of clinical aspects:-

The unique location of the internal carotid artery within a venous structure occasionally gives rise to direct communication between the two structures such that a carotico cavernous sinus fistula (CCF) is established, either as a result of severe head trauma or degenerative or aneurysmal vessel disease. A CCF causes proptosis, which may be pulsatile, together with vascular dilatation in the tissues of the orbit and globe and combinations of third, fourth and sixth cranial nerve palsies. These changes can cause permanent blindness. Any spreading infection involving the upper nasal cavities, paranasal sinuses, cheek (especially near the medial canthus), upper lip, anterior nares or even an upper incisor or canine tooth, may very rarely lead to septic thrombosis of the cavernous sinuses as infected thrombi pass from the facial vein or pterygoid venous complex into the sinus (via either ophthalmic veins or emissary veins that enter the cranial cavity through the foramen ovale). This is a critical medical emergency with a high risk of disseminated cerebritis and cerebral venous thrombosis.⁴

Spread of infection -infection may potentially spread some distance from the infratemporal fossa because the latter lies between the tissue spaces of the face above and the tissue spaces of the neck below. Thus infection may spread to involve the buccal tissue space, or directly around the back of the maxillary tuberosity and into the orbit via the inferior orbital fissure, which may result in a cavernous sinus thrombosis. Once in the orbit, infection may spread directly through the superior orbital fissure into the cranial cavity. Infection may also spread from the infratemporal fossa via the pterygomaxillary fissure to involve the pterygopalatine fossa and its contents, and may spread further via a number of small canals which lead from the fossa into the nose, pharynx and palate.¹³

Cavernous sinus syndrome- is defined by its resultant signs and symptoms: ophthalmoplegia, chemosis, proptosis, Horner syndrome, or trigeminal sensory loss. Infectious or noninfectious inflammatory, vascular, traumatic, and neoplastic processes are the principal causes. Examples of specific entities that may result in cavernous sinus syndrome are myriad and include carotid artery aneurysms, carotid-cavernous fistulas (C-C fistulas) tumors, and Tolosa-Hunt syndrome, to name the most frequently discussed.¹⁴

Cavernous sinus aneurysms- unlike intracranial aneurysms in other anatomic locations, carotid-cavernous aneurysms do not involve a major risk of subarachnoid hemorrhage. However, their rupture can result in direct C-C fistulas, which may lead to cerebral hemorrhage. These aneurysms, which are more frequent in the elderly population, present with an indolent ophthalmoplegia. Although some patients suffer minor disability and do not require treatment, endovascular occlusion of these lesions is often successful and may be attempted in selected patients.

Discussion:-

As per the classical description the Srungataka Marma is It is situated within the confluence of Sira (vessels); nourishing the Ghrana(nose), Shrota (ear), Akshi(eye) & Jivha (tongue). The location of this marma is difficult to understand as the exact landmarks are not mentioned like other marma. The classical description is indicating the only vascular constitution of the Srungataka marma and its structural link with the sense organs. To find its location one has to explore such arrangement within the head having vascular link with Ghrana, Shrota, Akshi & Jivha. In respect to this, only cavernous sinus seems to have vascular connections directly or indirectly with these sense organs.

The literary meaning of the term Srungataka is indicating the cluster or group, joining place of four ways. The word meaning is appropriate for correlating the Srungataka marma with cavernous sinus hence this sinus is having communication with cluster of veins in all four directions therefore the word meaning is relevant.

Actually Cavernous sinus (CS) is a very crucial vascular structure of middle cranial fossa present on either side of sphenoid sinus. It measures 2cm length x 1cm width. The sinus system in the brain is a complex web of veins contains no valves; blood can flow in any direction depending on the prevailing pressure gradients. The Sphenopalatine vein from Nose (Ghnan) are connected to cavernous sinus through pterigoid plexus. Veins of Eye are communicated through the superior, inferior ophthalmic vein and central vein to CS. In case of ear, mastoid emissary vein in the mastoid foramen, which unites the sigmoid sinus with the posterior auricular or occipital veins. In turn sigmoid sinus is in connection with CS. The area of mid face is draining to pterigoid plexus. The inferior petrosal sinus drains the cavernous sinus into the internal jugular vein. All the venous blood of brain eventually goes to internal jugular vein.

The above anatomical discussion reveals the vascular connection of CS with the sense organs . Therefore based on this description, the location of Srungataka marma can be determined on either side of sphenoid sinus in middle cranial fossa. There are two CS connected with each other by anterior & posterior interconnecting sinuses. By considering this, the four numbers of shringatak Marma is substantiated. Other connecting veins cannot be included under enumeration of this Marma as cavernous and intercavernous are situated within the network of these viens. The A-P length of CS is 2 cm , the width of the sphenoid sinus is 1.5 to 2 cm therefore the area of both CS including width of sphenoid sinus measures approximately 6-7 cm which is approximately equal to 4 Angula pramaan. In the four angula circumferencial area many important openings like foramen ovale, foramen spinosum, foramen rotandum, carotid canal, foramen lacerum, superior orbital fissure giving entry to many neuro vascular structures.etc are situated.

Aghataparinaam –according to description it is sadyapranahara Marma. On injury of this instant death can occur or person will die within seven days after the trauma. Consequences and severity of trauma are depending upon the force, direction and region involved. External or internal Injury of cavernous sinus invites the acute thrombosis which is a life threatening condition. The thrombosis of sinus system severely hampers the circulation of CSF which has further life threatening consequences. pressure around the brain may rise, causing papilledema (swelling of the optic disc) which may be experienced as visual obscurations. In severely raised intracranial pressure, the level of consciousness is decreased, the blood pressure rises, the heart rate falls and the patient assumes an abnormal posture. If such conditions are not managed properly death is inevitable.¹⁵ As classical description states the vascular communication with the sense organs; infections of the face including the nose, tonsils, and orbits can spread easily by this route. This intimate juxtaposition of veins, arteries, nerves, meninges, and paranasal sinuses accounts for the characteristic etiology and presentation of cavernous sinus thrombosis (CST).

In the pre antibiotic era infections of the medial third of the face, including the nose, orbits, tonsils, and soft palate, most commonly resulted in septic thrombosis of the cavernous sinus. Bacteria readily entered the facial veins or pterygoid plexus, reaching the cavernous sinus via the superior or inferior ophthalmic veins. In addition, infections of the ear not infrequently spread by emissary veins from the mastoid air cells to the sigmoid sinuses and later reached the cavernous sinuses by way of the inferior petrosal sinuses .Previous antibiotic-era cases have also emphasized the association of facial infections with this complication. Approximately 60% of patients with septic cavernous-sinus thrombosis from 1940-1960 had a preceding facial infection.

In the early antibiotic era, 1940-1945, otitis media was the second most common site of infection predisposing to this complication. In the more recent antibiotic era 1961 to 1984, the sphenoid and ethmoid sinuses have become more frequent primary sites of infection predisposing to septic cavernous sinuses .Bacteria from these air sinuses can spread to the cavernous sinus by small emissary veins or by direct contiguity.¹⁶ Occurrence of cavernous sinus thrombosis (CST) has always been low, with only a few hundred case reports in the medical literature.

Prior to the advent of effective antimicrobial agents, the mortality rate from CST was effectively 100%. Typically, death is due to sepsis or central nervous system (CNS) infection. With aggressive management, the mortality rate is now less than 30%. Morbidity, however, remains high, and complete recovery is rare. Roughly one sixth of patients are left with some degree of visual impairment, and one half have cranial nerve deficits. These mortality and morbidity rates may be due to delayed diagnosis without prompt surgical drainage and antibiotic administration.¹⁷ Frazier stated in his study that, the prognosis in pre-antibiotic days was dismal, since death almost always occurred within days and he estimated only 7% survival.¹⁸

During Sushruta's period the mortality may be 100% and he might have observed severe bleeding from this Marmasthana, that is why he might have categorized this as sadyapranahara. On the basis of recent surgical complications this is possible to infer. Steven Newman has stated that, One of the factors that prevented earlier surgical approach to cavernous sinus lesions was the fear of causing uncontrollable bleeding. The earliest surgery within the cavernous sinus was performed with patients under hypothermia and hypotensive anesthesia or even in cardiac arrest. Although bleeding still may present a challenge during surgery, this issue has turned out to be less of a problem than otherwise expected, given that the lesion itself displaces the venous space.¹⁹

Since the beginning of the modern era of skull base surgery, surgical mortality has been substantially reduced. Dolenc and coworkers²⁰ reported 4 deaths (6.3%) out of 63 patients who had undergone cavernous sinus surgery. More recent series have reported the incidence of death in 2 (3.7%) of 54,²¹ 3 (7.3%) of 41,²² and 2 (9.5%) of 21 postsurgical patients.²³ In the present series, 2 patients died within the perioperative period (2.4%), only 1 with a meningioma (1.75%). A CSF leak is one of the most frequently occurring complications following cavernous sinus surgery and is probably a direct result of surgical aggressiveness. Its incidence may be as high as 28%.²⁴ This above clinical data indicates the proportion of death due to injury or surgical complications to this area. The infections from the sense organs separately can spread to cavernous sinus. There are many case reports however the cases are not ended up in the death but residual morbidity.

Kangsanarak, Jaranet al reported 24,321 patients with otitis media treated at the Otolaryngology Department of Chiang Mai University Hospital from 1978 through 1990, 87 patients had 140 central nervous system complications (0.36%). Multiple complications existed simultaneously in almost 45% of the patients. The group developing the complications was mainly in their second decade of life. Meningitis occurred in 43 patients, brain abscess in 29, perisinus abscess in 23, lateral sinus thrombosis in 16, and extradural abscess in 12 patients.²⁵ Complications arising from otitis media especially intracranially, they tend to carry along with them a high rate of morbidity and mortality. The death rate of lateral and sigmoid sinus thrombosis, for instance, has been reported to be in the range of 5–35%.²⁶ Chirinos JA et al. highlighted Lemierre syndrome, or necrobacillosis, is a severe systemic infection with *F. necrophorum*. The first of the 3 classical stages is primary infection, usually of the throat or ear. Next, the tissue surrounding the lateral pharyngeal space is invaded, and thrombophlebitis of the internal jugular vein develops. Finally, there is metastatic spread of the bacteria, often associated with systemic coagulopathy.²⁷

Jacob L. et al. has opined that *F. necrophorum* is ubiquitous in the normal oral flora, but these anaerobic gram-negative rods are sufficiently virulent to cause infection in immune competent hosts, and, worse, they produce an endotoxin that appears to promote coagulation. Although a recent literature review revealed 116 case reports of *F. necrophorum* infections, occurrence is likely under reported. *F. necrophorum* infections may cause serious complications, including cranial nerve palsies, sinovenous thrombosis predisposing to venous infarction, sepsis and death.²⁸

Dental infections constitute less than 10% of the cases of CST. The dental infection most commonly spreads via the pterygoid venous plexus, where an infected thrombus may extend or disseminate septic emboli. The infection can spread to the contralateral cavernous sinus via intercavernous sinuses, usually within 1 to 2 days of the initial presentation.²⁹ In 1989, Ogundiya et al reported case of a patient with dental infection which got complicated leading to orbital abscess, unilateral blindness, and CST.³⁰ In 1991, Yun et al. reported CST in a 60-year-old diabetic male about 38 days after extraction of an infected upper third molar tooth.³¹

There are multiple important openings like foramen ovale, foramen spinosum, foramen rotundum, carotid canal, foramen lacerum, superior orbital fissure etc are situated in and around area of cavernous sinus. Giving passage to important neurovascular structures. So in skull base fractures also there is possibility of fracture of these foramen and injury to the nerves & vessels. This is one more reason to designate this region as marmasthaan. In above discussion it is observed that the infection from sense organs is possible to spread to cavernous sinus directly or indirectly and if not managed, properly it proves fatal. Therefore sadyapranaharatwa (instant fatality) is relevant in today's era too.

Conclusion:-

Based on the review of anatomical and clinical data it is revealed that, the central part of middle cranial fossa is proved as one of the vulnerable region, as it is ascertained as Shringataka Marma sthaan. The discussion substantiates the constitution of this marma by cavernous sinus, intercavernous sinus, carotid artery with venous

plexus and draining veins(terminal part) in the sinus, body of sphenoid bone with hypophyseal fossa, sphenotemporal, sphenofrontal, sphenoccipital joints, trigeminal ganglion with its three divisions.

Traumatic effect of this marma is instant death or within seven days due to intimate juxtaposition of veins, arteries, nerves, meninges, and paranasal sinuses accounts for the characteristic etiology and presentation of cavernous sinus thrombosis, if not managed properly, affects the CSF circulation. Increased intracranial pressure causes death immediately or within days. Thus, the Ayurvedic ancient thoughts and facts of traumatology signify its relevancy.

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