



Case Study

POTENTIAL THERAPEUTIC BENEFITS OF *BRAHMA RASAYANA* IN THE MANAGEMENT OF TUBERCULOUS CEREBROVASCULAR DISEASE - A CASE REPORT

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ABSTRACT

Tuberculous meningitis (TBM) is an air-borne infectious disease caused by the bacteria *Mycobacterium tuberculosis* that affects the central nervous system (CNS). Among all the incident cases of TB, CNS TB represents approximately 1% with TBM as the most grievous among all. The basic pathology in TBM is the inflammation of the arachnoid membrane, the pia mater and the cerebrospinal fluid (CSF). It typically presents as mild fever, headache, anorexia and general debility that progress over one to two weeks to cause severe headache, fever, vomiting, confusion, meningismus and cranial nerve deficits. The most common complications of TBM include hydrocephalus, optico-chiasmatic arachnoiditis, seizures and stroke. Out of these, tuberculous cerebrovascular disease is a common neurological sequelae. This case study elaborates the treatment line and observations made in a 29 year old male patient who presented with hemiparesis and significant sensory deficit following an event of tuberculous meningitis. MRI brain was suggestive of basilar meningitis, optico-chiasmatic arachnoiditis, infarcts and tuberculoma with chest X-ray revealing increased bronchovascular markings in bilateral lung fields. Initially on admission, *Deepana-pachana* was done followed by *snehapana* with *Shadpala ghrta* and *Virechana* as *Sodhana karma*. *Abhyanga*, *Ushma Sweda*, *Churna pinda sweda* and *Jambeera pinda sweda* were successively done allied with physiotherapy. *Yogavasti* with *Vedanasthapana gana* as *Kashaya* and *Kalka* was done intervened by *Anuvasana vasti*. Succeedingly, *Murdhni taila prayoga* and *Marsha nasya* were also incorporated with periodical neurological, hematological and biochemical assessment. On discharge, *Brahmi kalyanaka ghrta* and *Brahma Rasayana* were advised inclusive of physiotherapy.

INTRODUCTION

TBM is one among the extra-pulmonary presentations of TB that typically affects the CNS. Here, the term meningitis refers to the inflammation of the arachnoid membrane, the pia mater and the intervening cerebrospinal fluid. The inflammatory process can extend into the subarachnoid space and also involve the ventricles. The symptomatology of the disease is characterized by less than two weeks of low-grade fever, anorexia, lethargy and headache that advances to classic clinical syndrome of meningism, confusion and altered sensorium over weeks.

Nuchal rigidity, photophobia and headache constitute the triad of meningism with the Kernig's sign and Brudzinski's sign as the marked clinical signs. Depressed levels of consciousness, diplopia and hemiparesis from focal ischaemia of cerebral arteries are also common occurrences. Physical examination in acute TBM reveals neck stiffness and occasional cranial neuropathy (VI, III, IV, VII in order of frequency) and long-tract signs<sup>[1]</sup>. TBM is often subacute with an initial polymorphonuclear pleocytosis that evolves to lymphocytic predominance rapidly. CSF glucose level less than two-thirds that of the serum glucose level; CSF protein level greater than 50mg/dL and increased WBC count with lymphocyte predominance are classical of TBM<sup>[2]</sup>. CSF culture is definitive in diagnosis with the Gene-Xpert as the extensively utilized primary diagnostic test. Chest radiographs may be consistent with pulmonary or miliary TB<sup>[3]</sup>. CT-head may show

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contrast enhancement over the basilar meninges, hypodense areas consistent with infarcts, hydrocephalus and sometimes focal inflammatory lesions (tuberculomas)<sup>[3]</sup>. CT angiography may show entrapment of vessels or vasculitis<sup>[3]</sup>. Complications include CVA, optico-chiasmatic arachnoiditis, hydrocephalus or seizures. Anti-tubercular treatment (ATT) is to be initiated early on diagnosis.

TBM presents varying symptomatology in its prodromal, meningitic and paralytic phases. Initially manifesting symptoms are often *Mrdu jwara* (low-grade fever), *Shira soola* (headache), *Aruchi* (anorexia) and *Glaani* (general debility). The low-grade fever generally progresses in 1-2 weeks (*Jwara vaishamyā*) with aggravation of headache (*Murdhni vedana*) and altered sensorium (*Moha*) that depict a *Vata-paittika dosha* state with *Aasrayasthana* as *Siras*<sup>[4]</sup>. Among the complications, hemiparesis (*Pakshaghata*) with associated sensory impairment (*Vichetanatwa*) is not uncommon. Here, the treatment principles of *Vatavyadhi* with focus on *Uthamaanga chikitsa* (treatment for diseases in the head and neck region) can be adopted with conservative techniques of physiotherapy.

Latent Tuberculosis Infection (LTBI) is also common in subjects with no marked symptoms but investigations revealing TB infection. LTBI can develop into the disease whenever the bodily immunity is significantly compromised. This can best be considered as a state of *Leenadosha* (the state where *Dosha* remains dormant or concealed) progressing to manifest the disease with the resurge of the specific etiological factors. Among the *Kriyakala* (consecutive stages of manifestation of disease), latency can be considered as the first stage - *Chaya* (stage of accumulation of *Dosha*) that shifts to the state of *Chayapoorvaka prakopa* (stage of aggravation of accumulated *Dosha*) on manifestation of TBM syndrome. Thus enhancing *Vyadhikshamatwa* or host immunity becomes the prime need to resist the infection.

### Case Description

A 29 year old male attended the OPD of *Kayachikitsa*, Government Ayurveda College, Thiruvananthapuram on 19/07/2021 with chief complaints of partial weakness and paraesthesia of the left side of his body since 1 year 5 months. The subject was a previously diagnosed case of tuberculous meningitis (TBM).

### History of Present Illness

The patient with no known co-morbidities and apparently normal before February 2020, gradually started experiencing mild headache of pin-prick type

which aggravated during the evening hours. With regular dull headaches for over a month, medical consultation was taken and primary investigations revealed no abnormalities. No further investigations were advised and done. In a span of one month, the headache progressed in severity associated with evening rise of body temperature, nausea and extreme weakness. There is no history of visual aura, blurriness of vision, vomiting or neck stiffness. Within two days, the condition worsened, the patient was laid up in bed being weak, and was thus shifted to the hospital. Following investigations (MRI brain), tuberculous meningitis (TBM) was diagnosed and ATT was initiated. The patient was only partially conscious during the hospital stay and does not memorize the events during these days. On 12 April 2020, he was stable to get discharged, with resolved symptoms except for residual weakness and paraesthesia of left side of body. With the continuation of ATT, the course of which was 12 months, physiotherapy was also initiated. With the completion of the ATT schedule, the patient visited our OPD and was admitted for the management of the presenting complaints.

### Examination

On inspection, prominent stretch marks over abdomen and mild spastic gait were noted. The patient also experienced poor appetite from disease onset that worsened with ATT.

### Cranial Nerve Examination

V: Reduced sensation to pain, light touch and temperature along the ophthalmic, maxillary and mandibular divisions, VII: Mild facial asymmetry, XI and XII: mild weakness over left were noted.

### Sensory System Examination

Pain, light touch, temperature and vibration were diminished over left half of face, left upper and lower limb (distal more than proximal).

Assessment of the sensory system using the neuropathy analyser -

**Biothesiometry Study (Vibration):** Mild and severe loss on right and left half of the body respectively.

**Cold Perception:** Normal study on right half and mild loss on left half of the body.

**Hot Perception:** Mild loss on right half and moderate loss on left half of the body.

**Motor System Examination:** Revealed no marked difference in muscle bulk bilaterally with grade 1 spasticity of the left lower limb. Muscle power: left upper limb digits- 4<sup>-</sup> on flexion and extension, left foot- 4<sup>-</sup> on dorsiflexion and plantar flexion, 1<sup>st</sup> digit of left foot- 3 on flexion and extension, all other muscle groups- 4<sup>+</sup>.

**Table 1: Deep Tendon Reflex (DTR)**

	Biceps jerk	Triceps jerk	Supinator	Knee jerk	Ankle jerk
<b>Right</b>	2+	2+	2+	3+	1+
<b>Left</b>	3+	3+	3+	4+	Not elicited

**Superficial Reflex:** Extensor plantar response on left with abdominal reflex elicited in all quadrants.

#### MRI Brain

MRI brain revealed extensive thickening of the basal subarachnoid cisterns (suggestive of basilar meningitis), sub-acute infarcts in right hemi pons and right hemi medulla, left P1 PCA infarct, a nodular and ring enhancing lesion along the left cerebellum (suggestive of tuberculoma) and optico-chiasmal arachnoiditis. No dilatation of ventricles or hydrocephalus was elicited.

#### Chest X-ray

Increased broncho-vascular markings in bilateral lung fields.

#### CECT - Thorax

Focal area of sub-segmental atelectasis in the apical segment of right upper lobe.

#### CSF Examination

Total WBC count- 60 cells/cubic mm [differential count on 30 cells: Neutrophils- 10, lymphocytes- 20], CSF protein- 470.40mg/dL and CSF sugar- 36.90mg/dL.

[Characteristic CSF findings of TBM include lymphocytic- predominant pleiocytosis, elevated protein levels (typically between 100 and 500mg/dL) and low glucose (usually less than 45mg/dL)].

#### Blood Investigation

Absolute eosinophil count [AEC]- 850 cells/cumm, FBS- 129mg%, PPBS- 288mg%, Triglycerides- 189mg%, Uric acid- 9.8mg%, SGOT (AST)- 53 IU/L, SGPT (ALT)- 76 IU/L, CRP - 1.4mg/dL

**Diagnosis:** Tuberculous cerebrovascular disease

#### TREATMENT

**Table 2: Intervention**

Date	Treatment
<b>19.07.2021</b>	<b>Internal medicines</b> 1. <i>Saptasaram kashayam</i> - 90ml BD before food with <i>Guda, Saindhava prakshepa</i> 2. <i>Vaiswanara churna</i> - 5gm BD with hot water 3. <i>Dhanwantaram gulika</i> - 1-0-1 4. <i>Abhayarishta</i> - 30ml BD after food
<b>21.07.2021- 15.08.2021</b>	<i>Rooksha sweda</i> with <i>Kolakulathadi churna</i> - 7 days <i>Churna pinda sweda</i> (CPS - <i>Rooksha</i> ) with <i>Kolakulathadi churna</i> and <i>Dhanyamla</i> -10 days <i>Dhanyamla dhara</i> - 7 days <i>Upanaha</i> with <i>Grihadhoomadi churna, Saindhava, dhanyamla</i> and <i>Punarnavadi kashaya</i> over left knee joint <i>Utsadana</i> with <i>Kolakulathadi churna</i> and <i>Dhanyamla</i> - 7 days <i>Thalapothichil</i> with <i>Musta churna, Amalaki churna, Jatamansi churna, Panchagandha churna, Brahmi swarasa</i> - 7 days <i>Thalam</i> with <i>Kachooradi churna</i> and <i>Karpasasthyadi tailam</i> (with <i>thalapothichil</i> )
<b>23.08.2021</b>	<i>Snehapana</i> planned <i>Takrapana</i> prior to <i>snehapana</i> (10gm <i>vaiswanara churna</i> in one litre of <i>Takra</i> taken intermittently per day)- 5 days
<b>28.08.2021</b>	<i>Snehapana</i> with <i>Shadpala ghrta</i> - 6 days ( <i>samyak snigdha lakshana</i> was obtained on sixth day) (maximum dose of intake of <i>ghrta</i> : 250 ml)

03.09.2021	<i>Abhyanga</i> with <i>Shatahwadi taila</i> along with <i>Ushma sweda</i> - 3 days
06.09.2021	<i>Virechana</i> with <i>Gandharva erandatailam</i> – 50 ml with lukewarm milk at morning (after <i>sleshma kala</i> ) <i>Peyadikrama</i> done
09.09.2021	<b>Internal medicines:</b> 1. <i>Maharasnadi kashaya</i> – 90 ml BD before food 2. <i>Ekgangaveera rasa</i> – 1-0-1 after food 3. <i>Brahma Rasayana</i> – 5 gm BD CPS with <i>Satahwadi tailam (Snigdha sweda)</i> - 7 days <i>Thalam</i> with <i>Rasnadi choorna</i> and <i>Ksheerabala tailam</i> (with CPS) <i>Anulomana</i> following CPS with <i>Gandharva erandatailam</i> - 30ml with lukewarm milk at morning
17.09.2021	<i>Shirodhara</i> with <i>Karpasasthyadi tailam</i> - 7 days
24.09.2021	<i>Marsha nasya- Anutaila</i> [2.5ml each nostril] - first 3 days <i>Ksheerabala taila</i> 7 <i>Avartti</i> [2.5ml each nostril]- next 4 days
03.10.2021	<i>Jambeera pinda sweda (JPS)</i> - 7 days <i>Anulomana</i> following JPS with <i>Gandharva erandataila</i> - 30ml with lukewarm milk at morning
11.10.2021	<i>Pizhinjuthadaival</i> (modified type of <i>Snigdha drava sweda</i> ) with <i>Karpasasthyadi taila</i> - 7 days
16.10.2021	<i>Yogavasti</i> - 8 days <i>Anuvasana vasti - Pippalyadi anuvasana taila</i> (100ml with 1 pinch of <i>saindhava</i> ) <i>Kashaya vasti [Vedanasthapana gana kashaya</i> - 480ml, <i>Sahacharadi taila (Madhyama paka)</i> - 120ml, <i>Vedanasthapana gana kalka</i> - 30gm, <i>Madhu</i> - 120ml, <i>Saindhava</i> - 15gm]
24.10.2021	Physiotherapy initiated
28.10.2021	<b>Medicines on Discharge</b> 1. <i>Brahma rasayana</i> – 10gm morning in empty stomach 2. <i>Brahmi kalyanaka ghrta</i> – 5gm HS 3. <i>Mahamasha taila</i> – External application ( <i>Abhyanga</i> and <i>Shiro pichu</i> ) 4. Advised to continue physiotherapy

Following treatment, below mentioned changes were noted:

### 1. Sensory System Examination

**Biothesiometry Study (vibration):** Mild loss on right half and moderate loss on left half of the body.

**Cold Perception:** Normal study on right half and mild loss on left half of the body.

**Hot perception:** Mild loss on right and left half of the body.

### 2. Motor System Examination

Muscle power: left upper limb digits- 4<sup>+</sup> on flexion and extension, left foot – 4<sup>+</sup> on dorsiflexion and plantar flexion, 1<sup>st</sup> digit of left foot – 4<sup>-</sup> on flexion and extension, all other muscle groups – 5 .

### 3. Blood Investigation

Absolute eosinophil count [AEC]- 490 cells/cumm, FBS- 118 mg%, PPBS- 188 mg%, Triglycerides-

138 mg%, Uric acid- 7.2 mg%, SGOT (AST)- 50 IU/L, SGPT (ALT)- 45 IU/L, CRP- 0.8 mg/dL

### DISCUSSION

In Ayurvedic concept, hemiparesis as complication of TBM can be managed following the treatment line of *Pakshaghata*. Here, the *Vata jwara lakshana (Murdhni vedana, Sankha nistoda and Agama-apagama nature of Jwara)* and *Vata - Pitta jwara lakshana (Sirasoola, moha)* can be precisely elicited in the stage of *Purvaroop*. In this case, the chief presentations are weakness and paraesthesia of left half of body which indicates the significant role of *Vatadosha* in *Roga samprapthi*. Owing to the *Rogi lakshana* such as *Jataragni mandya, Balahani* and *Sareera dhaarana ashakti*, the *Dhatus* involved are possibly *Rasa, Rakta, Mamsa, Asthi* and *Majja*. On assessing the disease course, the *Asraya sthaana* of *Dosha* is the *Shiras* with loss of *Sneha saara* in *Mastishka (or Mastulunga majja)*. The resulting

*Dhatukshaya* manifests as *Pakshaghata lakshana*. The biochemical investigations were suggestive of a possible ATT- induced hepatotoxicity as observed from the elevated blood glucose, SGOT and SGPT levels. Significant sensory and motor deficits were also noted. Considering the long term use of ATT, immune compromise and evident *Dosha dushti* in *Shakha*, *Shodhanakarma* was done after adequate *Deepanapachana* and subsequent *Snehapana* with *Shadpala ghrta*. After the elimination of accumulated *Dosha (Sanchita dosha nirharana)*, treatment with regard to *Kevala vata vyadhi*, incorporating *Pakshaghata chikitsa* and *Uthamaanga chikitsa* were implemented. *Brahma rasayana*, with scientifically studied roles as anti-oxidant, anti-inflammatory and immune- modulator was advised on discharge. *Brahmi kalyanaka ghrta* (internally) and *Mahamasha taila* (externally) along with physiotherapy were also instructed.

### CONCLUSION

TBM is the most common among CNS TB that leads to varying complications including neurologic deficits such as stroke. Along with ATT, introducing Ayurvedic management in conjunction aids in early recovery and enhances the quality of life (QOL) of subjects who survive the disease. This case study elaborates the role of *Sodhana*, *Samana* and *Rasayana chikitsa* in the management of hemiparesis and associated sensory deficits as sequelae of TBM.

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