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A COMPARATIVE STUDY OF RAJYAKSHMA AND PULMONARY TUBERCULOSIS- REVIEW ARTICLE

Vignesh Madaswamy Pillai[1], Kalpana Gholap [2], Ajinkya Deepak Acharekar[3]

[1]Asst. Professor, Dept of Panchakarma, Datta Meghe Ayurvedic Medical College Hospital Research Centre, Nagpur Maharashtra, India
[2]Asso. Professor, Dept of Panchakarma, Dr. G.D.POL Foundation's YMT Ayurvedic Medical College And Hospital, Kharghar, Navi-Mumbai, Maharashtra, India
[3]Assistant Professor, Shalyatantra Department B.R. Harne Ayurvedic Medical College, Karav, Vangani, Thane, Maharashtra, India

Corresponding Author: vigfrndz@gmail.com

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ABSTRACT

Rajyakshma is described as a group of symptoms manifested by indulging *Sahasa, Vegasandharana, Kshaya, and Vishamshana.* In charak Samhita named *'ROGA SAMUHANA'* Rajayakshama is considered a set of chronic respiratory disorders and tuberculosis has been included under it being an infectious disease affecting respiratory organs. **Aim And Objective:- 1.** To know the causes, signs, symptoms, pathogenesis, and treatment of Rajyakshma mentioned in different ayurvedic texts. 2. To know the types, signs symptoms, and treatment of Pulmonary Tuberculosis mentioned in modern texts. 3. To compare Rajayakshma and pulmonary tuberculosis. **Materials And Methods:** All the Ayurvedic and modern literature and contemporary texts including websites required for this review article have been reviewed and documented in the study. **DISCUSSION & CONCLUSION**: - Rajyakshma being considered a group of chronic pulmonary diseases, can be included as one of the aspects of Rajyakshma, which has been revied in this article.

Keywords: *Sahasa, Kshaya, Rajyakshma, Sandharana,* Pulmonary tuberculosis.

INTRODUCTION

The word Rajyakshma is self-explanatory. It is the innovator of the rest of the other diseases. It is the group of diseases that gets manifested with the vitiation of *tridosha* and *saptadhatu*. *Rajyakshma* is considered the king of all diseases. Thus, it is termed as *Raja(king) and Yakshama(decay)*. Various diseases like *Atisara* (diarrhoea), *Shoth* (inflammation), *Pandu* (anaemia), *and Jvara* (fever), emerge as a complication of *shosha*. The prodromal symptoms of the disease are *Pratishyaya*(common cold), *Swaas* (breathlessness), and *Kaas* (cough). The disease whose causal factors are different to understand and whose treatment is not an easy task, such a disease is called '*Mahabalshali*' or disease of great strength by Acharya *Sushruta*. *Rajyakshma* is the group of symptoms that arise due to *Sahasa* (excessive physical activity), *Sandharana* (suppression of natural urges), *Kshaya* (loss of tissues), and *Vishamshana* (Intake of food in an improper manner). *Rajyakshma* is manifested by the vitiation of *Vata and Kapha pradhan tridosha*.

AYURVEDIC CONCEPT OF RAJYAKSHMA: According to Ayurveda, health is defined as "Where physical, mental, and spiritual state is balanced with respect to body and function". The imbalance of all these factors is known as *Dhatu Vaishamya vyadhi*. According to ayurveda, usually, the seven dhatus are converted into OJAS, which is considered a carrier of prana, but in Rajyakshma these dhatus are converted into *mala* and excreted through the body. Ancient Acharya classified the causes of Rajyakshma into four categories.

CHARAK[(1)]*----1) Sahasa 2) Sandharana 3) Kshaya 4) Vishamashana, SUSHRUTA*[(2)] *----1) Aaghata 2) Vegapratighata 3) Kshaya 4) Vishamashana, VAGBHATA*[(3)]*---1) Sahasa 2) Vegasanrodha 3) Kshaya 4) Vishamashana, MADHAVAKARA*[(5)]*--- 1) Vegarodha 2) Kshaya 3) Sahasa 4) Vishamashana*

ETIOLOGY:

SAHASA: Excessive work or fight or exercise beyond one's capacity.

Example: 1) Weak person fights with a strong person. 2) Excessive talkative / Speaks too much. 3) Forceful massage and application of pressure by feet. 4) Runs fast to cover a long distance.5) injury

Reasoning: Due to these the Vata dosha gets vitiated and it affects the kapha in *Urah pradesha* and also vitiates *pitta*. These vitiated doshas move in every direction in the body causing various symptoms.

SANDHARANA: Suppression of natural urges for defecation, micturition, etc. Example: 1) Travelling high or low 2) Near to king 3) While gambling 4) In front of female

KSHAYA: Depletion or discretion. '*Shosha*' is the synonym of Kshaya and explains that the loss of capability of the body to do work is called *Kshaya*. Example: 1) Diminution of rasa. 2) Intake of food in lesser quantity or fasting by persons who are weak by nature. Depletion of dhatus can take place in two ways that can be termed as i) *Anuloma kshaya and ii) Pratiloma kshaya*.

Reasoning: 1) *Anuloma Kshaya* means the depletion of dhatus takes place in the direction of their nourishment i.e., *Rasa then Rakta then Mamsa*, etc. 2) *Pratiloma Kshaya* means the depletion of dhatus takes place in the direction of opposite to their nourishment i.e., *Shukra* then *Majja* then *Asthi* and so on.

VISHAMASHANA: Intake of food without considering the *Ashta Vidhi Vishesha Ayatanani* Example: These are 1) *Prakriti* (nature of food). 2) *Karana* (improper preparation). 3) Samyoga (combination of food). 4) Matra (Quantity). 5) *Desha* (locality). 6) *Kala* (time). 7) Not consuming *Shadrasatmak Bhojana*. 8) *Upayoga samstha* (dietetic rules and wholesomeness). Reasoning: The imbalanced dosha while spreading through the body obstructs the srotas due to which the food taken by the individual does not get converted into dhatu and is excreted through the body in the form of stool.

Rajayakshma Samprapti:- [(6)(7)(8)]

Acharya Charaka explains the pathogenesis of all the types of Rajayakshma in *Nidana sthana* of charak saṃhitā in detail. While explaining the *Samanya Samprapti of Rajyakshma* he states that when Agni is in is pure form, it leads to the formation of *Dhatus* in a true and pure state. These dhatus stay in

their respective *srotas* and with the help of agni form the subsequent dhatu. If there is any obstruction in the *srotamsi*, it leads to depletion of dhatus specially rakta dhatu in this case of Rajyakshma. To explain this *Acharaya chakrapani* says that the obstruction in *srotamsi* is due to dosha which is responsible for the manifestation of the disease. As a result of this, there is no proper availability of Poshaka ras to Rakta and other Dhatus. This also affects the quantum of *dhatvagni*, which is further decreased by Dosha Prabhav. Gangadhara opines that *Dhatvagnimandya* takes place due to the depletion of the *Adharma* i.e., the Dhatu which in turn vitiates the *Adheya* i.e., *Dhatvagni*. In this way, the *Dhatu* depletion and *Dhatvagnimandya* lead to further depletion of Dhatus as well as an increase in *Dhatvagnimandya*. The result is incorrect metabolism of the nutrient leading to more formation of *Kitta* and less formation of *Sara rupa Rasa*. Not only Dhatvagni but also *Bhutagni* gets vitiated. While describing the Samprapti of *Grahani Roga* Acharya Charaka says that *Jatharagni* gets vitiated due to *Nidana sevana*, in return, it is unable to digest a small quantity of food. The undigested gets fermented leading *to Amavisha*. When this *Amavisha* comes in contact with different dhatus of the body it becomes the cause of various diseases. *Amavisha* in contact with *Kapha Dosha* leads to *Prameha, Yakshma, Pinasa*, and other *Kapha diseases.* In this way, we see that *Ama* also acts as an important component of *Samprapti*. Acharya *Charaka and Sushruta* have mentioned two probable ways of manifestation of the disease viz. *Anuloma Kshaya and Pratiloma Kshaya.* Etiological factors also play a crucial role in the provocation of dosha which then spreads through the body. The vitiated *kapha dosha* obstructs the *Srotas* specially *Rasavaha Srotas* leading to malnutrition of further Dhatus and leading to the condition called S*hosha*. According to Acharya *Vaghbhata,* all three vitiated *Doshas* while circulating through the body, enter the sandhi, affecting the *Siras and* leading to obstruction of the Srotas, resulting in dilatation of the *srotas*. Depending on the site where these doshas get settled they produce various symptoms in the person. *Madhava nidana* described the Samprapti of Rajayakshma as similar to Acharya Sushrut. *Vijayarakshita* comments that not only the depletion of dhatus but also the vitiation of srotas is important; otherwise, it will only be Dhatu *Kshaya, not Rajyakshama*. *Anuloma Kshaya* happens not only because of obstruction of *rasavaha srotas* but also other *srotas* due to vitiated kapha dosha. Hence, we conclude that *Vitiated Doshas* and Vitiated Agni are the two main causative factors of Rajayakshma.

MODERN VIEW AND STATUS OF TUBERCULOSIS: -

TUBERCULOSIS remains one of the killer infectious diseases among the adult population in developing countries today. It is a serious bacterial infection caused by a rod-shaped obligate and intracellular bacterium named mycobacterium tuberculosis. It is a slow-spreading chronic infection characterized by gradual weight loss. TB is spread through the air when people lung TB cough, sneeze or spit Effective drugs and vaccines are available even though the disease remains a major threat to life.

Mycobacterium is slender bacteria, difficult to stain i.e., resist deculturation with dilute minerals, and are therefore called AFB. Its a non-motile, non-capsulated, non-spore-forming bacteria. Seven species are responsible for mammalian tuberculosis. M. tuberculosis (human tuberculous bacillus), M.Bovis (bovine tubercle bacilli), M.microti (vole tubercle bacilli),M.africanum(intermediate form between M.tuberculosis &M.Bovis), M.capre, M.camettii & M. pinnipedin.

Morphology

Mycobacterium is slender, straight, curved bacillus with rounded ends, measures 1.4µm × 0.2 µm (average 3 µm – 0.3 µm) in size. Gram Positive bacteria stained only with the Ziehl-Neelsen staining method. Other method of staining is Kinyoun's method of acid-fast staining (except heating is not required hence called cold staining.

Miliary TB: -

1. Blood-borne dissemination gives rise to miliary TB, which may present acutely.

2. Characterised by 2-3 weeks of fever, night sweats, anorexia, weight loss, and a dry 3. Hepatosplenomegaly may develop and the presence of a headache may indicate coexistent tuberculous meningitis.
4. Auscultation of the chest is frequently normal. Although with more advanced diseases widespread crackles are evident.
5. Fundoscopy may show choroidal tubercles.
6. The classical appearances on chest X-rays are of fine 1-2 mm lesions (millet seed) distributed throughout the lung fields, although occasionally the appearances are coarser.
7. Anaemia and leukopenia reflect bone marrow involvement.

Pathogenesis: - [10]

The infection is commonly acquired by inhalation of infected droplets coughed or snored into the air by a patient with pulmonary tuberculosis. In bovine tuberculosis infected cows develop lesions in the udder and bacilli are excreted in the milk which can then infect people who drink raw milk. In developed countries, the pasteurisation of milk has virtually eradicated this organism.

Tubercle bacilli are engulfed by macrophages, but they survive and multiply in macrophages. These lyse the host cell, infect other macrophages and sometimes disseminate to other parts of the lung and elsewhere mechanism in tuberculosis the body. The cell-mediated immunity (CMI) plays an interact with these macrophages whereas the major role to interact humoral immunity appears to be irrelevant, CD4+ helper cells secrete interferon gamma, interleukin 2, tumour exerting different biological Necrosis factors and others exert necrosis effects. It may result in protective immunity or delayed-type hypersensitivity (DTH) reactions. Th-1-dependent type cytokines activate macrophages to kill intracellular mycobacteria and thus result in protective immunity. Th-2 cytokines induce delayed-type hypersensitivity (DTH), tissue destruction, and progressive disease.

Types:- Tuberculosis is divisible into primary and secondary forms:-

1. Primary Tuberculosis:- Inhaled tubercle bacilli are engulfed by alveolar macrophages which replicate to form a lesion called Ghon focus. It is frequently found in the lower lobe or lower part of the upper lobe. Some parts of the bacilli are transported to the hilar lymph nodes. The Ghon focus along with the enlarged hilar lymph nodes is called the primary complex. In the case of M. bors which enters through the mouth, the primary complexes involve the tonsil and cervical lymph nodes or the intestine, often the ileocecal region, and the mesenteric lymph nodes.
2. Secondary (Post-primary) Tuberculosis: - It is caused by reactivation of the primary TB (endogenous) or by exogenous reinfection. Granulomas of secondary tuberculosis occur in the apex of the lungs. The necrotic reaction causes tissue destruction and the formation of a large area of caseation termed tuberculomas. The proliferation of caseous necrosis and cavities are two special features of secondary tuberculosis. Cavities may rupture blood vessels, spreading mycobacteria throughout the body, and break into airways releasing the organism in aerosols and sputum (open tuberculosis).

Clinical features: pulmonary disease

1. Chronic cough
2. Haemoptysis
3. Fever
4. Night sweats
5. Malaise
6. Loss of appetite
7. Loss of weight
8. Unresolved pneumonia
9. Asymptomatic (diagnosis on CXR)
10. General debility
11. Exudative pleural effusion

Diagnosis:-

01. An otherwise unexplained cough lasting more than 2-3 weeks, especially in a location where tuberculosis is common, or typical chest X-ray alterations should prompt further investigation.
02. Direct microscopy of sputum (induced with nebulised hypertonic saline if not expectorating) at least 2 but preferably 3 (early morning samples). The chances of identifying acid-fast bacilli

in sputum are proportional to the bacillary burden (typically positive when 5000-10,000 organisms are present). Tuberculous bacilli are difficult to stain due to their substantial lipid-rich wall The most effective techniques are the Ziehl-Neelsen and rhodamine-auramine stains Rhodamine-auramine stains cause the tuberculosis bacilli to fluoresce against a dark background and are easier to use when numerous specimens need to be examined.

03. A positive smear is sufficient for the presumptive diagnosis of TB, but a definitive Diagnosis requires culture. Smear-negative sputum should also be cultured since culture-positive sputum requires only 10-100 live organisms.

Blood investigations

Raised ESR. CRP, Anaemia, etc.

Tuberculin skin test (low sensitivity/specificity; useful in primary or deep-seated infection)

Treatment: -

The antitubercular drugs include bactericidal agents such as rifampicin (R), isoniazid (H), pyrazinamide (Z), streptomycin (S), and bacteriostatic agents include ethambutol (E), thiacetazone, ethionamide, para-amino salicylic acid (PAS) and cycloserine. Short course regimens of 6-8 months are used. In 2017, the RNTCP adopted a new daily TB treatment regimen. In new TB cases, a combination of four drugs (HRZE) is given daily during an initial intensive phase (IP) for 2 months, followed by three drugs (HRE) daily during the continuation phase (CP) for 4 months. In previously treated TB cases, treatment is given for a total of eight months Streptomycin is also included in IP for 2 months. The intensive phase (IP) is of three months Le HRZES for 2 months and HRZE for one month while the continuation phase (CP) contains HRE for 5 months. As resistant strains emerge readily by mutation and selection, combinations of two or more drugs are used. If the patient is treated with only one antitubercular drug, initially the bacilli die in large numbers, but soon resistant mutants emerge and multiply unchecked. The bacterial resistance may be primary (prior to the start of treatment) or secondary which emerges during the course of treatment.

A serious consequence of unchecked drug resistance has been the emergence of multidrug resistance tuberculosis (MDR-TB). The term multidrug resistance refers to resistance to rifampicin and isoniazid, with or without resistance to one or more other drugs. MDR-TB is a global problem, especially in HIV-infected persons. When first-line drugs are ineffective, second-line drugs are to be used. Quinolones, aminoglycosides, para-amino salicylic acid (PAS), ethambutol, thiacetazone, cycloserine, and capreomycin are being used. Directly observed therapy under supervision (DOTS) is being used to prevent the deterioration of resistance problems by ensuring the patient's compliance. In MDR-TB cases, treatment is given with second-line drugs under DOTS-Plus When the patient does not show improvement, an antibiotic sensitivity test is done to detect resistant strains.

Another serious condition extensively drug-resistant tuberculosis (XDR-TB) has emerged recently, XDR-TB is due to M. tuberculosis strains which are resistant to any fluoroquinolone and at least one of three injectable second-line drugs (capreomycin, kanamycin, and amikacin), in addition to isoniazid and rifampicin.

In recent years, two new terms extremely drug-resistant tuberculosis (XXDR-TB) and drug-resistant tuberculosis (TDR-TB) have been described by different researchers. These terms have been used for M. tuberculosis strains which are resistant to all first-line drugs (isoniazid, ethambutol, pyrazinamide, and streptomycin) and second-line drugs (ofloxacin, moxifloxacin, kanamycin, amikacin, capreomycin, para-aminosalicylic acid and ethionamide). In 2007, two cases of TDR-TB were first reported from Italy. In 2009, Iran was the second country to report 15 cases of XXDR-TB or TDR-TB. Recently in 2011, 12 such cases have been reported from Mumbai, India. However, XXDR-TB and TDR-TB terms are not recognised by WHO. According to WHO these cases are considered

XDR-TB. These new terms are still to be accepted at the international level.

AYURVEDIC MANAGEMENT:

In Ayurveda, there are uncountable drugs that combat the disorder related to the lungs. Today the entire world knows the efficacy and seriousness of our classical regimen in the management of the deadly epidemic disease COVID-19. We know corona viruses that cause illnesses such as the common cold, SARS, and MERS, and these all belong to respiratory tract infections. This itself justifies that if pulmonary TB managed carefully with Ayurvedic medicines could definitely uphold the decline in death rates due to PTB.

TREATMENT:

In the condition of *Shira Shula (headache), and Parshwa Anasa Shula* (pain in the sides of chest and shoulder) the treatment modalities like *Navana, Dhumapana, and Snehapana*, after intake of food, Abhyanga with oil and *Vasti karma* is useful. *Raktamokshana* by using *Shrunga (horn), Alabu (gourd), Jaulauka (leeches), and Siravedh* are indicated. *Prapaundarika, Nirgundi, Padma Kesara, Utpala, Kaseruka, and Payasya* with *Ghrita* is useful in *Shira Shula in Rajyakshma Chandanandi Taila and Satadhatu Ghrita* are used for Abhyanga in Daha condition. Pariseka with milk, *Yashtimadu Kashaya or Mahendra Shita jala*, or the *Chanadanadi Gana Kashaya* are useful in Daha condition. *Mrudu Shodhana, Snehana, and Svedana followed by Vamana and Virechana*.

CONCLUSION

Rajyakshma is the tridoshaja vyadhi with the dominance of vata and kapha dosha and it is an ideal example of *Madhyama Roga Marga*. Here all the *sapta dhatus* are involved in the manifestation of Rajyakshma. All these dhatus are in the state of kshayavastha. The symptoms of *Rajyakshma* are different according to the involvement of *Sahasa, Vegsandharana, Kashaya, and Vishamshana*. Before planning the treatment, it is very much necessary to understand the involvement of causes in the disease and the process of pathogenesis. *Samprapti Vighatana* plays a very important role in the diagnosis of the disease and its treatment.

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