

A study of Samprati (Pathology) of Kamala with reference to Ayurveda and modern science

J B Kamble^{1*}, A J Budkuley², N S Chandurkar³

¹Associate Professor, ²PG, ³Professor, Department of Sharir Rachana, Padm. Dr D Y Patil College of Ayurved and Research Centre, Pimpri – Pune, Maharashtra, INDIA.

Email: jkdhemre71@gmail.com

Abstract

Introduction: Ayurveda considers Jaundice (Kamala) as a disorder of raktavahastrotas. Yakrit and pleeha are moolasthana of raktavahastrotas. Vitiated Pitta is the main causative factor in the pathogenesis of kamala. As per Modern View the human liver contains complex parenchymal cells that perform multiple functions which are essential for life. The liver does not easily demonstrate dysfunction at least in its metabolic activities. This is because of enormous reserve capacity and marvelous regenerating power of the liver and only a small portion of the liver is enough to perform all the functions. About 75- 80% of liver, need to be out of function for any of the test to be positive. "a silent organ," The common causes of chronic liver diseases all over the world are infection with hepatitis B virus, hepatitis C virus and alcohol abuse. Bilirubin is the yellow breakdown product of normal heme catabolism caused by body's clearance of aged RBCs which contain haemoglobin. Bilirubin works as cellular antioxidant. Haemoglobin is broken down to heme and globin portion. **Conclusion:** The Samprati (Pathology) of Kamala should be studied in reference to Ayurveda and Modern as these two wisdom are not irreverent to each other or contradictory but complementary to each in understanding various causes and hence the Pathology of Jaundice (Samprati of Kamala) and could be helpful for the management also.

Keywords: Samprati of Kamala, Pathology of Jaundice, Bilirubin.

*Address for Correspondence:

Dr. J B Kamble, Associate Professor, Department of Sharir Rachana, Padm. Dr D Y Patil College of Ayurved and Research Centre, Pimpri – Pune, Maharashtra, INDIA.

Email: jkdhemre71@gmail.com

Received Date: 23/03/2016 Revised Date: 14/04/2016 Accepted Date: 12/05/2016

Access this article online

Quick Response Code:



Website:
www.statperson.com

DOI: 12 June 2016

The common causes of chronic liver diseases all over the world are infection with hepatitis B virus, hepatitis C virus and alcohol abuse. The community prevalence of both hepatitis B virus and hepatitis C virus infections in Nepal are comparatively low³. Alcohol is the most common substance abused in Nepal and a study carried out in 2000 AD found that about 60% of the Nepalese population had experienced alcohol and 41% had taken it during the last 12 months⁴. Alcohol is associated with high morbidity and mortality; about 3.7% of the global deaths⁵. Ayurveda considers Jaundice (Kamala) as a disorder of raktavahastrotas. ⁶Yakrit and pleeha are moolasthana of raktavahastrotas.⁷

INTRODUCTION

The presence of jaundice is usually, but not always, a sign of liver disease. The causes of jaundice are many and range from the common to the rare. The most common causes encountered in Southeast Asian region are; infective hepatitis, obstruction to bile ducts by gall stones or tumors, alcoholic liver disease, drugs, etc¹. Geo-cultural factors influence the prevalence of liver disease of public health importance in any country. Liver disease may vary from country to country and in the same country in different cultural groups and at different periods of time.²

MATERIAL AND METHODS

Vitiated Pitta is the main causative factor in the pathogenesis of kamala.⁸ In the disease under consideration, this vitiated pitta affects the liver in a major way and shows general manifestations in the body. This study should give a deeper insight on the intricate aspects on pathology of bahupitta kamala.: Normal complexion of skin grossly depends on two factors- Teja⁹ and rakta¹⁰ along with snehaguna¹¹ of ojas.¹² Pitta resides

in raktadhatu.¹³ Exposure to dosha vitiating factors leads to vitiation of Pitta, especially its ushna-tikshnaguna, to cause disturbance in the normal physiology of the saumya, sneha quality of ojas which is already in circulation with raktadhatu. Along with this reduced the raktadhatu' sposhakras (required in formation of raktadhatu) is also reduced. Decrease of both raktaand ojas hampers the normal complexion and hence pathological complexion appears. The abnormal complexion varies according to the doshic imbalance. Out of this imbalance of tridosha, aggravated pitta causes the panduvarnata complexion in pandu disease. Moreover, depreciation of the ten qualities of ojas is directly proportional to dhatu daurbalya.⁹ (Many Acharyas agree that the condition of Bahupitta Kamala is preceded by Pandu. Hence, its discussion is invariably important too.) As Described by Gabriel Van Loon in the Text Book CharakaSamhita Handbook on Ayurveda Volume II, 'The patient of panduroga who takes P-aggravating things excessively, his P burns, blood and flesh and thus gives rise to the disorder. His eyes, skin, nails and face become deep yellow, feces and urine as red and yellow and he looks like a frog. His senses and organs lose their functions and he is associated with burning sensation, indigestion, debility, malaise and anorexia. It is due to aggravation of P and is known as located in kostha (belly) or sakha (blood etc.). Kumbhakamala (jaundice located in belly) being established firmly due to chronicity becomes curable with difficulty. Ci16#34-36Prognosis of Kamala: The patient of jaundice succumbs soon to the disease if feces and urine becomes black yellow, there are excessive swelling, blood in eyes, mouth, vomiting, "feces and urine" (what?), fainting, burning sensation, anorexia, thirst, hardness in bowels, drowsiness, confusion, loss of the power of digestion and consciousness. Ci16#37-38¹⁴. The word kamala literally means the loss of desires of doing any work, eating, etc. It can be called as severe anorexia or malaise. Acharyacharak and Harita considered the disease as a type of second stage of Panduroga. Acharyavaghbhat considered it as a separate disease. Although the panduroga is the important etiological factor of the disease the pathogenesis can also take place independently. Bahupittakamala - similar to haemolyticjaundice, Rudhapathakamala - similar to obstructive jaundice **Bahupitta kamala:** Samprapti (Causes and pathogenesis) The patient of panduroga who takes pitta aggravating things and lifestyle then it leads to more pitta prakopa. It leads to burning and It leads to excess secretion from the liver. This secretion spreads all over the body and thereby producing yellowish discoloration on nails eyes skin urine and stools. Thereby leading to Bahupitta karma.Rudhapata Kamala Causes and pathogenesis: Due to intake of dry, cold, heavy, sweet

food items, holding the urges like urine and stools etc. leads to vitiation of vata and kapha. So these vataprakopa makes the kapha dry and thereby leading to the obstruction in pittavahini. The other reasons for this obstruction are gall stone, any tumor in the surrounding area and worms. Due to these obstruction though the formation of pitta is normal. It will not be able to reach the intestines and the colour of stools becomes whitish. Again the obstruction in pittavahini causes the improper movement of pitta producing the symptoms of indigestion, loss of appetite, burning sensation, thirst, debility of body.¹⁵ Kamala has been classified as: KoshtashritaShakhashrita¹⁶In modern science jaundice is classified in three types: Haemolytic (Due to Lysis of RBCS and Excess Bilirubin Production), Obstructive (Obstruction to Common Bile Duct) Hepatocellular (Damage to Liver tissues)¹⁷.On the basis of samprati kamala gets manifested in two forms-koshtaasritaandsakhaasrita kamala. Though in both type vikriti pitta play the role in sakhaasrita type the pitta which has been vitiated does not reach the koshta. So there is an underlying pathology of increased vatadosha leading to saktagati of pitta along with an increased kapha stage crating a srotorodha. Thus not letting the normal pitta come back to koshta. Thus sakhaasraya kamala is a clear form of asayaapakarshajanyavyadhi. Form this total samprapti we can infer that mala ranjana does not occur in sakhaasraya. Kamala due to the absence of pitta in kostha thus, leading to condition of "svetavarchs" and it is a partial obstruction it certainly result in tilapishanibhavarchas. In classical reference another two chronic conditions as 'kumbha kamala' and 'haleemaka' are seen. Kumbhakamala is mentioned by AcharyaChakrapani as a bheda of koshtaasrita kamala, in which patient may present with condition of oedema associated with ascitis and bleeding tendencies. Haleemaka is being explained as vata- pitta dominant condition of kamala by Vaghbhataacharya and it is also known as alasaka.¹⁸ CharakSamhita-According to CharakSamhita, Kamala is a clinical syndrome which develops after the panduroga. When a patient of Panduroga takes excessive paittikahar-vihar develops bahupittakamala. ^{21,23}ShushrutaSamhita²²-According to ShushrutaSamhita, when patient of panduroga or person affected with other diseases consumes amlaraspradhan and apathyakaraha develops kamala. AshtangHriday²³- According to AshtangHriday, when pandurogi or person with excessive pitta consumes pittakaraha develops kamala. Samprapti: Koshtashakhashrita kamala²¹: SampraptiGhataka: 1. Dosha-Pitta. 2. Dushya-Rakta and Mansa.3. Adhisthana-Kostha(Mahasrotasa) and shakha. 4. Srotas- Rasavahasrotas, Raktavahasrotas, Annavahasrotas, Purishvahasrotas. 5. Srotodushti-

Atipravritti, Sanga, Vimargagamana. Shakhshritakamala²² SampraptiGhataka:- 1. Dosha – Pitta 2. Dushya -Rakta and mansa 3. Adhisthana- Kostha (mahasrotas) and shakha 4.Srotas - Rasavaha, raktavaha, annavaha, pureeshvahasrotas 5. Srotodushti – Atipravritti, sanga, vimarggamana.

DISCUSSION

As per Modern View the human liver contains complex parenchymal cells that perform multiple functions which are essential for life. The liver does not easily demonstrate dysfunction at least in its metabolic activities. This is because of enormous reserve capacity and marvelous regenerating power of the liver and only a small portion of the liver is enough to perform all the functions. About 75- 80% of liver, need to be out of function for any of the test to be positive. As is clear from the fact that the liver has been called “a silent organ,” a diseased liver shows relatively few clinical signs unless the disease is severe or advanced. However, simple liver function tests using blood samples are widely available as part of routine health examination, providing opportunities for physicians to find abnormalities in liver function test results in daily clinical practice²⁴ The common causes of chronic liver diseases all over the world are infection with hepatitis B virus, hepatitis C virus and alcohol abuse. Alcohol is the most common substance abused in Nepal and a study carried out in 2000 AD found that about 60% of the Nepalese population had experienced alcohol and 41% had taken it during the last 12 months.⁴ Bilirubin is the yellow breakdown product of normal heme catabolism caused by body's clearance of aged RBCs which contain haemoglobin. Bilirubin works as cellular antioxidant. Haemoglobin is broken down to heme and globin portion. The globin portion is a protein that breaks down into amino acids and plays no role in the pathogenesis of jaundice. The heme, on the other hand, undergoes oxidation reaction catalysed by the enzyme oxygenase to give biliverdine, iron and carbon monoxide. Biliverdine yield a yellow pigment called bilirubin (unconjugated). In the liver, the bilirubin is conjugated with glucuronic acid to give conjugated bilirubin which is water soluble that can be excreted. Bacteria in the intestine convert the bilirubin into urobilinogen. This urobilinogen is then either converted into stercobilinogen or excreted in the feces or it is reabsorbed by the intestinal cells and taken to the kidneys via the blood to be excreted in the urine. In this way, normally the liver metabolizes and excretes the bilirubin in the form of bile. However, if there is disruption in this normal metabolism production of bilirubin, jaundice may results.

CONCLUSION

The Samprati (Pathology) of Kamala should be studied in reference to Ayurveda and Modern as these two wisdom are not irreverent to each other or contradictory but complementary to each in understanding various causes and hence the Pathology of Jaundice (Samprati of Kamala) and could be helpful for the management also.

REFERENCES

1. Gupta M, Patil R, Khan MI and Gupta SK. Retrospective hospital based study of infective causes of jaundice in Tamilnadu, India. Calicut Medical Journal 2011; 9(2):1-4.
2. Shrestha SM. Liver diseases in Nepal. Kathmandu University Medical Journal 2005; 3(2): 178-180.
3. Shrestha SM, Tsuda F, Okamoto H, et al. Hepatitis B virus subtypes and hepatitis C virus genotypes in patients with chronic liver disease in Nepal. Hepatology 1994; 19:805-809.
4. Dhital R. Alcohol and young people in Nepal. Available from: <http://www.ias.org.uk/resources/publications/theglobe/glob20010 3-04>.
5. WHO sixtieth world health assembly. Evidence- based strategies and interventions to reduce alcohol- related harm. Provisional agenda item 12.7. April 5, 2007.
6. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Sutrasthana, VividhashitpitiyaAdhyaya, 28, Shlok 12, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
7. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Vimanasthana, StrotasamvimanAdhyaya, 05, Shlok 08, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
8. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Chikitsasthana, PanduChikitsaAdhyaya, 16, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
9. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Sutrasthana, AtreyabhadrakapiyaAdhyaya, 26, Shlok 11, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
10. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Sutrasthana, VidhishonitiyaAdhyaya, 24, Shlok 04, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
11. Sushruta, Dalhana. SusrutaSamhita. Sutrasthana, Annapaan-vidhiAdhyaya, edited by Vaidya AmbikadattaShashtri, reprint ed. ChaukhambaSankritSansthana, Varanasi, 2010.
12. Agnivesha, Charaka, Dridhabal, Chakrapani. CharakaSamhita. Sutrasthana, VidhishonitiyaAdhyaya, 24, Shlok 31, edited by Vaidya Harish Chandra Singh Kushwaha, reprint ed. ChaukhambaOrientalia, Varanasi, 2011.
13. Vagbhata, AshtangaHridyam, Sutrasthana, DoshabhediyaAdhyaya, 12, edited by

VaidyaBhramhanandTripathi, reprint ed. Chaukhamba Sanskrit Pratisthan, Delhi, 2007.

14. Gabriel Van Loon. CharakaSamhita Handbook on Ayurveda Volume II. P.V. Sharma and ChaukhambhaOrientalia Publishers.2002-2003. Pp-656.

15. ANHC. Kamala [cited 20 April 2016] available at :<http://www.healthandayurveda.com/articles.html>

16. Shastri K.N; Chaturvedi G.N; Charaksamhita 8 ed. Chaukhambhabharti academy Varanasi 1981

17. Fauci; Braunald; Harrisons principle of internal medicine 14ed. MC grew hill book Scompany 1998.

18. AmitkumarSingh. Hepatitis- An AyurvedicPerspective. Asian Journal of Modern and Ayurvedic Medical ScienceJuly-december 2014.3(2).1-5.

19. VaidyaYadavajiTrikanjiAcharya, CharakSamhita, ChakrapaniTika, Reprint 2013, p.528

20. VaidyaYadavajiTrikanjiAcharya, SushrutaSamhita, Nibandhsangraha and NyayachandrikaPanjikatika, ChaukhanbaPrakashan, Varanasi, Reprint 2014, p.729

21. HariSadashivshashtriParadkar, AshtangHriday, Sarvagasundara and AyurvedRasayanatika, ChaukhanbaPrakashana, Varanasi, Reprint 2014, p.519. VaidyaYadavajiTrikanjiAcharya, CharakSamhita, ChakrapaniTika, ChaukhanbaPrakashana, Varanasi, Reprint 2013, p.114

22. Ibid, p.179 6. Ibid, p.528, 532 7. Ibid, p.527

23. Tsubouchi H, Ido A and Mawatari S. New development in treating liver disorders: approaches to liver function test from mild to fulminant disorders. J Med Assoc Japan 2010; 53(4): 218–223.

Source of Support: None Declared

Conflict of Interest: None Declared