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Structural Consideration of *Majjavaha Srotas*

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ABSTRACT

Ancient medical science *Ayurveda* stands on its basic principles like *Panchamahabhuta siddhant*, *Srotas sharir*, *Marma vigyan* etc. *Srotansi* are the inner transporting channels of *dhatu* undergoing transformation. The colour, nature and structure of a *srotas* depend on which *dhatu* they carry. It may be circular, large, or small, straight or reticulated. *Majjavaha srotas* is only defined by *Acharya Charak* but *Acharya Sushruta* has excluded this. *Majja* is described as sixth *dhatu*. It is derived from final extract of *asthi dhatu*. *Majja* is the unctuous part found inside *asthi*. The function is to give moisture, strength, and formation of *shukra dhatu*. *Majja* occupies the pores and cavity of *asthi dhatu*. The *majja dhatu* may compare with yellow bone marrow in modern medical science. Bone marrow basically lies inside flat bones and end of long bones. The *moola* of *majjavaha srotas* are *Asthi* and *Sandhi*. The *moola* may be considered as chief source of that particular *srotas*. As *asthi* is the previous *dhatu*, it may be its *moola*. *Sandhi* is formed by the junction of two or more *asthi*. Mainly bone marrow is seen at the spongy bone situated at the joint forming end part of long bone.

KEYWORDS

Srotas, Majjavaha srotas, Moola sthan, RBCs formation

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INTRODUCTION

Srotansi are the inner transporting channels of *dhatu* undergoing transformation[1]. These are the structural and functional entities identified for the catering of metabolites in the body. Colour of the *Srotas* is in accordance with the colour of *dhatu* which they carry. They are circular (*vrit*), large (*sthul*) or small (*anu*),straight (*dirgha*), or reticulated (*pratan*) in shape[2]. The shape and size of *Srotas* varies from micro to macro channel, so that srotas can carry the *sthul* (macro molecule) as well as *paramasukshmadhatu* (micromolecules). There are thirteen number of *Srotansi* described on the basis of their controlling organs (*moola*) and symptoms manifested by their vitiation[3] (*prakopa*).*Majjavaha Srotas* is defined by*Acharya Charak*[4] and *Acharya Vagbhatta*[5]but *Acharya Sushrut*[6]has excluded this.

Majja is one amongst seven *dhatus*. *Majja* is considered to be the *sara* of *asthi* just like the *sara* found inside the tree according to *Vachaspatyam* and *Sabdakalpadrum*.*Meda* is present in the abdomen and small bones, while in long bones especially it is *majja*, where as in others (flat bones)it is called as *sarakta meda*[7].*Vata* creates hollowness inside the*asthi dhatu* and after which these hollow cavities get filled by the *meda*, which is known as *Majja*[8].The *Majja* are of two types, *pitta majja* (yellow marrow) located at the middle of long bones and *rakta majja* (red bone marrow) located at the end of long bones described by pratyakshya shariram.The chief quality of *majja* is to provide pleasure(*priti*), moisture (*sneha*), strength (*bala*),and filling of bonesand nourishes the *shukra datu*[9].The individuals having the excellence of *majjadhatus* are characterized by softness of organs, strength, unctuous complexion and voice along withfull-bodied long and rounded joints[10].Thesymptoms of decreased *majja* are less production of *shukra dhatu*, pain in joints, cutting pain in the bones, and emptiness in the bone etc[11], but Charak has described there is atrophy of bone tissues, weakness of bones and the patient suffers frequently from*vata*disorders[12].

The *moola* of *Majja vaha srotas* are*asthi* and *sandhi*[13]. *Majjavaha srotas* gets vitiated due to crushing, excessive liquification (*abhisyanda*), injury and compression of bone marrow and intake of mutually contradictory food[14].In context of *majjanugata bhagna*, the bone marrow comes out from its location which causes ultimate loss of *majja dhatu*, it may impair the normal function of *majja dhatu*[15]. Either due to vitiation or loss of

majjadhatu at its place, it may affect the normal function of *Majjavaha srotas*.

All above consideration defines *majja* as bone marrow, present within the bones.If we consider modern point of view, the bone marrow is a highly vascularizedconnective tissue located in the microscopic spaces between trabeculae of spongy bone tissue[16]. It is present chiefly in the axial skeleton, pectoral and pelvic girdles, and the proximal epiphysis of the humerus and femur. In average, bone marrow constitutes 4% of total body mass (approx. 2.5 kg). In newborn, all bone marrow is red and actively participate in blood formation. As an individual grows and in adulthood, the rate of blood formation decreases and the red bone marrow is replaced by yellow bone marrow, basically these are the fat cell. In pathological conditions, yellow marrow revert to red bone marrow and the red bone marrow goes repopulation to pluripotent stem cell.

In embryonic life, primitive nucleated RBCs are produced by yolk sac, in middle trimester mainly from liver,and partly from spleen and lymph nodes. In last trimester and after birth exclusively from bone marrow. Upto 5 years all bones produce RBCs. Proximal ends of humeri and tibiae produces RBCs upto 20 years. After age 20, membranous bones likes vertebrae, sternum, ribs and ilia produce RBCs[17].

Genesis of red blood cell

The blood cells begin their life in the bone marrow from a single type of cell called pluripotential hematopoietic stem cell, from which all the cells of the circulating blood are eventually derived (fig.1).Successive divisions of pluripotential cells form different circulating blood cells. As these cell reproduce, a small portion of them remains exactly like the original cell in the marrow to maintain a supply of these. Although their number diminishes with age. The intermediate stage cells (very much similar to stem cell) are called committed stem cells. The different committed stem cells, when grow in culture will produce colonies of specific types of body cells. A committed stem cell that produces erythrocytes is called a colony forming unit–erythrocyte (CFU-E).Same as colony forming units that form granulocytes and monocytes have the designation CFU-GM and like this. Growth and reproduction of different stem cells are controlled by multiple proteins called growth inducers, like interleukin 3, whereas the others induce growth of only specific type of cells. Growth inducers promote growth only but differentiation of the cells is promoted by differentiation inducers.

These causes one committed cell to differentiate one or more steps towards a final adult blood cell. Formation of growth inducers and differentiation inducers is itself controlled by factors outside the bone marrow. In case of low O_2 for long time causes growth induction and differentiation of erythrocytes like in hemorrhage. In case of infectious diseases, it causes growth; differentiation and formation of specific WBCs. (refer Fig. 1)

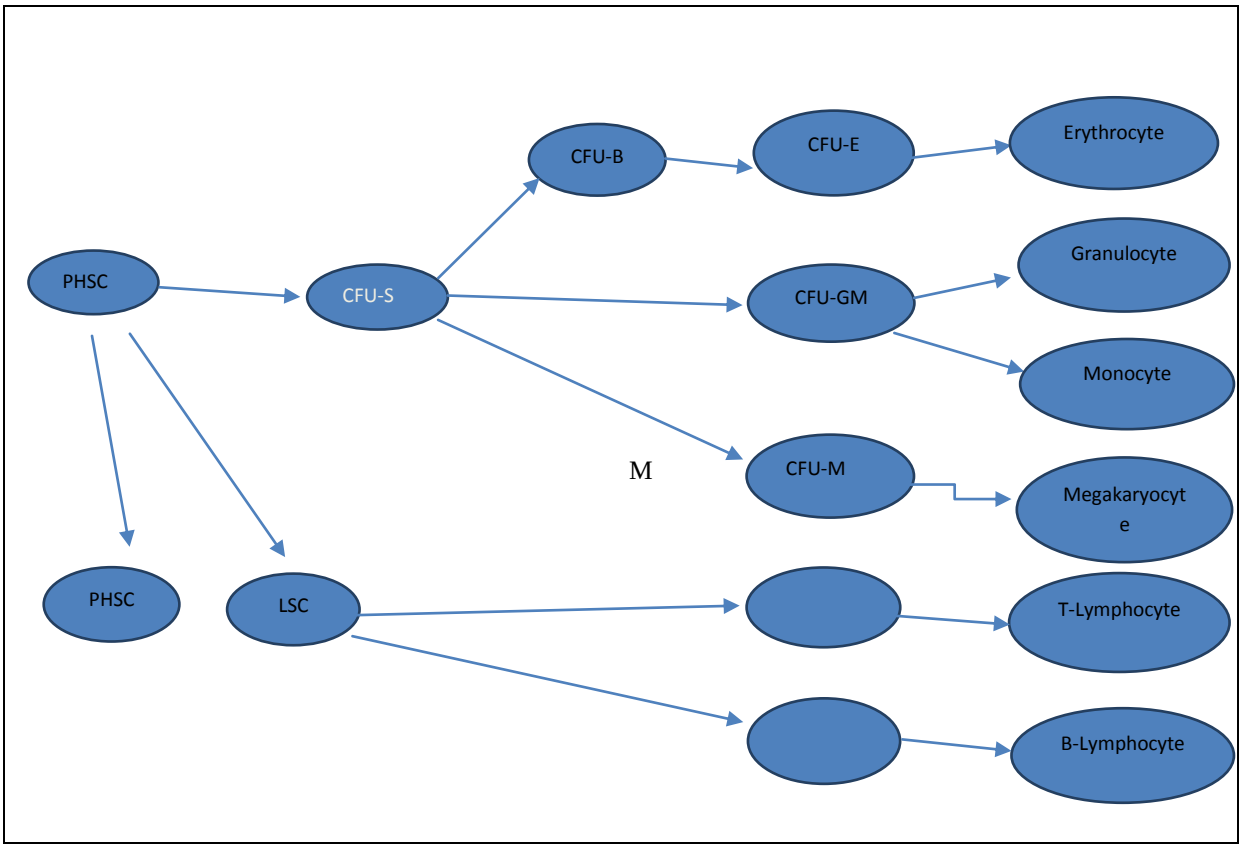

Fig 1 Differentiation and formation of specific WBCs

(PHSC-pluripotent hematopoitic stem cell, CFU-S: Colony forming unit-spleen, CFU-B:Colony forming unit-Blast, CFU-E:Colony forming unit-Erythrocyte, CFU-GMColony forming unit-granulocyte, monocyte, CFU-M:Colony forming unit-Megakaryoblast, LSC-Lymphoid stem cell) (Fig. 1: Formation of multiple different blood cell from pluripotent stem cell)

Stages of differentiation of RBCs

The first cell that can be identified as belonging to RBCs series is the pro-erythroblast under appropriate stimulation, large number of these cells are formed from the CFU-E stem cells. Once the pro-erythroblast has been formed, it divides multiple times, eventually forming many mature RBCs.The first generation cells are called basophil erythroblasts, these cell accumulates very little hemoglobin.Then succeedingthe cells become filled with

hemoglobin to a concentration of 34%,the nucleus condenses to a small size and its final remnant is absorbed from the cell. The endoplasmic reticulum is also reabsorbed. The cell at this stage is called a reticulocyte because it still contains small amount of basophilic material, consisting of remnant of Golgi complex, mitochondria, few other cytoplasmic organelles.During this reticulocyte stage, the cell pass from bone marrow into blood capillaries by diapedesis (through the capillary membrane). The remaining basophilic material in the reticulocyte normally disappears within 1-2 days and the cell is then become mature erythrocyte.

Role of erythropoietin in RBC Production

The principal stimulus for RBCs production in low O_2states is a circulatory hormone called erythropoietin (glycoproteinsecreted by kidney), which stimulate the production of pro-erythroblast from hematopoietic stem cells in red bone marrow.In absence of erythropoietin, hypoxia has little or no effect on stimulation of RBCs production(refer Fig. 2). Normally about 90% of all erythropoietin is formed in the kidneys, remainder is formed in the liver. Only 1/3rd or 1/2 production of RBCs occurs if there is damage of both kidneys.At the other extreme, when large quantities of erythropoietin are formed along with plenty of iron, and other nutrients available, the rate of RBCs production rises to 10 times or more.

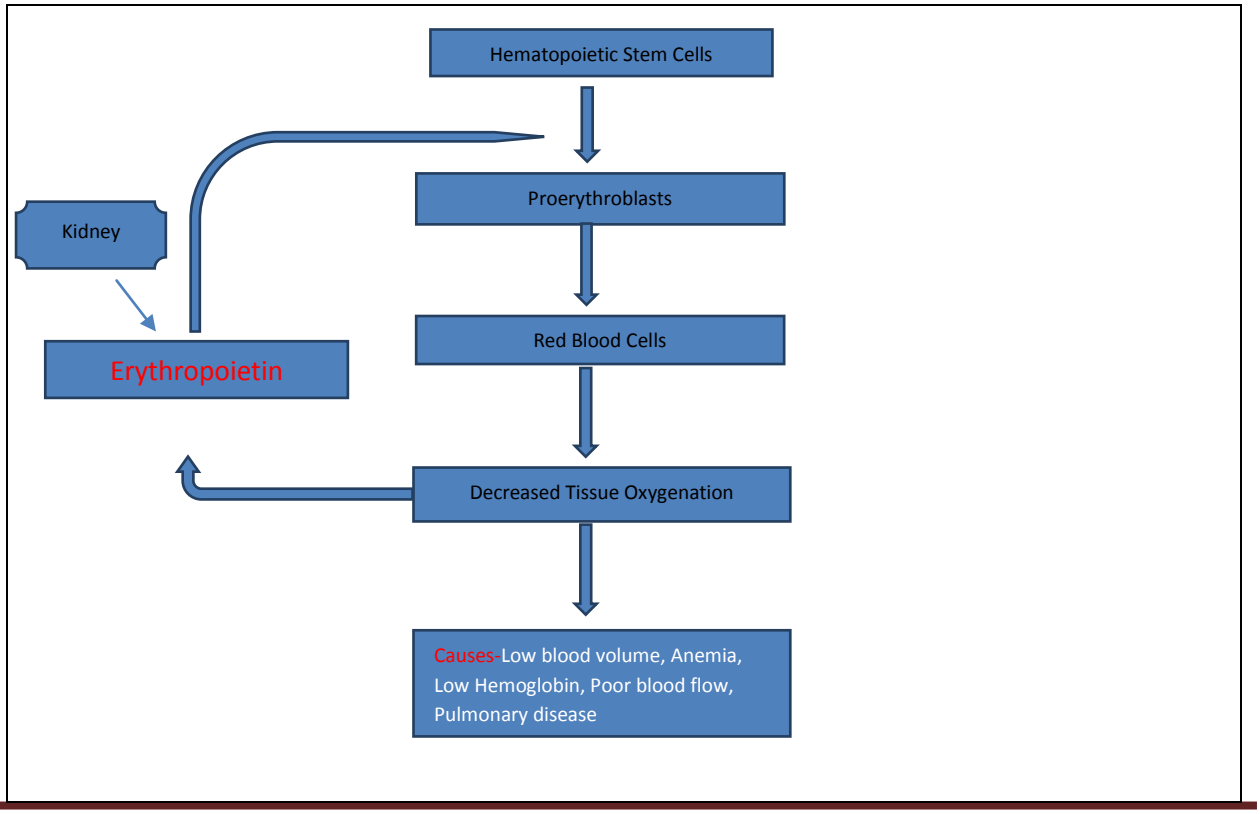

Fig2 Function of the erythropoietin mechanism to increase the production of RBCs when tissue Oxygen decreased

Maturation of RBCs

Vitamin B_{12} and folic acid are very much important for final maturation of RBCs. Both of these are required for synthesis of DNA by forming thymidine triphosphate.Therefore, lack of either Vitamin B12 or folic acid causes abnormal and diminished DNA leads to failure of nuclear maturation and cell division.Once Vitamin B_{12} absorbed from GIT, at first a large quantitystored in liver and then releases slowly as needed by bonemarrow.Folic acid is available in green vegetables, meats, some fruits.Therefore, in many instances of maturation failurethe cause is deficiency of intestinal absorption of folic acid and vitamin B_{12}[18].

If we see the whole anatomy and physiology, we can access that the *majja* as the bone marrow, present within the boneand the bone marrow has primary role towards blood cell formation. As Acharya Charak has described the formation of *majja dhatu* that, *Vata* produces hollowness inside the *asthi dhatu* and after which these hollow cavities get filled by the meda, which is called as *Majja*. On the other hand,*moola* of *meda vaha srotas* is *Vrikka* (*moola* of *meda vahasrotas*) and *Vappabahana*(omentum). As we see from above description that the erythropoietin secreted by kidney has great role towards maturation of RBCs. This show indirect relation of *meda* and *majjavaha srotas*. Vitamin B_{12} and folic acid has great role has important role in DNA synthesis and maturation of RBCs. Vitamin B_{12} is absorbed by the intrinsic factor secreted by parietal cell of stomach, and is stored in sufficient amount in liver which gives supply upto one year or more. These may compare with the *ranjak pitta*, responsible for the colouring of *rakta dhatu*.According to the*khale-kapot* principle, itproves that the required element (Vitamin B_{12}) for maturation of RBCs is used by *majja dhatu* (bonemarrow to mature RBC) from the main source *ahar rasa*.All these materials are utilized by*Majjavaha srotas* for formation of RBCs etc.

CONCLUSION

The*Srotansis*are the inner transporting channels of*dhatu* undergoing transformation. *Majjavaha Srotas*indicates the formationand maturation of RBCswithin the bone marrow of a bone. There are other supportive factors like erythropoietin secreted by kidney, vitamin $B_{12}$and folic acid take active participation towards maturation of RBCs. Vitamin B_{12} is absorbed by intrinsic factor secreted by parietal cell of stomach. Aftermaturation

of erythrocyte,get oxygenated in lungs and circulate to all tissue through systemic circulation, this can be considered under *rakta vahasrotas*. The *moola* of *majja vaha srotas*are *asthi* and s*andhi*, because functional activity of *majja* will be more in *asthi* and *sandhi*. It can be justified clearly because *asthi*are the main site ofbone marrow (red bone marrow is found in flat bones like hip bone, sternum, skull etc. and yellow bone marrow is found in diaphysial portion or shaft of long bones). *Sandhi* as *moola* won't be justified very clearly.The pathological conditions of *majja dhatu* menifests the symptoms like pain in joints, vertigo (*bhrama*), fainting (*murchha*),entering into darkness(*tamasadarsan*) and formation of deep seated abscesses in joint.Joint pain generally occurs because bones are the site of *majja dhatu*. *Bhrama*is due to*raja,pitta*and*vata* can be correlated with vertigo. This is a sensation of whirling and loss of balance, associated with particularly with looking from a height. One of the main causes of vertigo is vascular insufficiency or ischemic condition of brain. This ischemic condition has direct relation with blood cell (RBCs contains haemoglobin which carry Oxygen, necessary for nutrition of tissues) and the hypoxia condition have great role towards stimulation of progenitor marrow cell of red bone marrow for synthesis of RBCs. Same mechanism seen in *murchha* and *tamashadarsana* also.

REFERENCES

1. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika,1st ed. Chaukhambha bharati ecademi;2009. Pp 709.
2. Pandey K, Chaturvedi G, Charak SamhitaVidyatini tika,1st ed. Chaukhambha bharati ecademi;2009.Pp 714
3. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi;2009.Pp 710.
4. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi;2009.Pp 709-715.
5. Triphaty B, Astanga Hridayam, 1st ed.Chaukhamba Sanskrit Pratishthan delhi; 2009. Pp 375.
6. Jadavji Trikamji, Sushrut Samhita Nibandhasangraha tika, 1st ed. Chaukhamba Surabharati Prakashan; 2012. Pp 386.
7. Jadavji Trikamji, Sushrut SamhitaNibandhasangraha tika, 1st ed. Chaukhamba Surabharati Prakashan; 2012. Pp 356.
8. Pandey K, Chaturvedi G, Charak SamhitaVidyatini tika,1st ed. Chaukhambha bharati ecademi;2009.Pp 458.
9. Jadavji Trikamji, Sushrut Samhita Nibandhasangraha tika, 1st ed. Chaukhamba Surabharati Prakashan; 2012. Pp 67.
10. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi;2009.Pp776.
11. Jadavji Trikamji, Sushrut Samhita Nibandhasangraha tika, 1st ed. Chaukhamba Surabharati Prakashan; 2012. Pp 69.
12. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi; 2009.Pp 348.
13. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi; 2009. Pp 711.
14. Pandey K, Chaturvedi G, Charak Samhita Vidyatini tika, 1st ed. Chaukhambha bharati ecademi;2009. Pp 713.
15. Jadavji Trikamji, Sushrut Samhita Nibandhasangraha tika, 1st ed. Chaukhamba Surabharati Prakashan; 2012. Pp 329.
16. Tortora G. J, Derrickson B. H, Principles of anatomy and physiology, 12th ed. 2009. vol.2. Pp693.

17. Hall E. H, Guyton and Hall textbook of Medical Physiology, 12th ed.2012. Pp 414.