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Conceptual Study of Avascular Necrosis in Ayurveda and Modern Anatomical View

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Abstract

Introduction: Avascular necrosis (AVN) of the femoral head is a progressive degenerative disorder caused by interruption of blood supply, resulting in ischemia, osteocyte death, and eventual collapse of the femoral head. Although AVN is not directly described in classical Ayurvedic literature, its pathogenesis can be interpreted through the concept of *Anukta Vyadhi Sankalpana*. The condition shows similarities with *Asthimajjagata Vata*, *Asthi-Majja Dushti*, and *Srotodushti*. The present study was undertaken to analyze AVN from both modern anatomical and Ayurvedic perspectives.

Methods: This conceptual literary review was carried out using classical Ayurvedic texts, including the *Charaka Samhita*, along with modern anatomy and pathology literature. Relevant published articles were reviewed through peer review articles, the etiopathogenesis, vascular anatomy, and Ayurvedic correlation of AVN of the femoral head. **Results:** The review revealed that the femoral head possesses a delicate vascular supply mainly through the retinacular branches of the medial circumflex femoral artery, making it highly vulnerable to ischemia and necrosis. Ayurvedic analysis suggests that etiological factors such as trauma, alcohol consumption, steroid use, and vascular obstruction lead to *Rakta Dushti*, *Srotorodha*, vitiation of *Vata Dosha*, and

degeneration of *Asthi* and *Majja Dhatu*. Clinical manifestations such as pain, stiffness, limping, and restricted hip movements closely resemble the features of *Asthikshaya* and *Asthimajjagata Vata*.

Discussion: AVN can be understood as a degenerative and ischemic disorder involving *Vata Dosha*, *Rakta*, *Asthi*, and *Majja Dhatu*. Correlating AVN with Ayurvedic concepts provides a scientific basis for understanding its pathogenesis and may help in developing an integrative approach for its management.

Keywords: Avascular Necrosis, *Anukta Vyadhi*, *Rakta Dushti*, *Asthikshaya*, *Asthimajjagata Vata*

Introduction:

The classical texts of Ayurveda include the disease often present in that era of time. As time progressed, there has been change in lifestyle of people along with eating habits, daily regimen, medicine etc. These unavoidable and unfavorable changes in lifestyle lead to manifestation of new diseases. Clinical presentation of many of these diseases cannot be directly co-related with any of the nomenclated classical ailments. Diseases like Sick cell anemia, Dengue, Hypothyroidism, Hyperthyroidism, Cancer, Swine flu, Avascular necrosis, AIDS, COVID-19 pandemic condition, etc. cannot be directly co-related with any of nomenclated classical clinical entity. But to deal with such newly rising clinical conditions Acharyas have given certain guidelines and concepts like *Anukta vyadhi sankalpana*, *Nidan*, *Dosha*, *Dhatu*, *Ama*, *Agni*, *Avarana Shatakriyakala* etc. Hence,

one should initiate the treatment only after acquiring complete knowledge about nature of the disease as well as its pathogenesis, location and etiological factors.^[1] In Ayurveda, there is no any direct reference found about disease resembling Avascular Necrosis. In Avascular Necrosis of femoral head, *Vankshan Sandhishool* is one of the major symptoms. According to the involvement of *Dosha* and *Dushya*, AVN as an *Anukta vyadhi*, an effort is made to understand the possible pathogenesis of Avascular necrosis in terms of factors like *Hetu*, *Dosha*, *Dushya*, *Sthana* etc. which will be useful to initiate a proper line of treatment for the disease. Avascular necrosis (AVN), also known as osteonecrosis, aseptic necrosis or ischemic bone necrosis is a disease that may affect several different bones as a result of temporary or permanent loss of the blood supply to these bones. The ischemia causes the death and eventual collapse of the bone tissue, with its overlying joint surface.^[2] The femoral head is most commonly affected by this disease. Usually, the patients are in their third, fourth or fifth decade of life at the time of diagnosis. Men are more prone to this disease than women. Initially, patients are asymptomatic, but, in time, AVN leads to joint destruction, requiring surgical treatment and in latter stages, total hip replacement (THR).^[3]

Need of study

Although treatment has been facilitated by using a widely accepted international classification system, effective earlier diagnosis using MRI and more aggressive surgical management. No universally

satisfactory therapy has been developed, even for early disease. It is essential that AVN of the femoral head is diagnosed early because delaying this disease by joint preserving measures have a much better prognosis and because the results of joint replacement are poorer in young individuals. [4]

Objectives

To study the concept of avascular Necrosis through modern anatomical and Ayurvedic perspectives.

Specific Objectives

- To understand the etiological factors and pathogenesis of avascular necrosis through Ayurveda perspective.
- To review the Vascular anatomy of the femoral head in AVN.

Materials and Methods

Study Design: the present study is a conceptual and literary review.

Data Collection: classical Ayurvedic text like *Charaka Samhita* were systematically reviewed for *Nidan, Dosha, Dhatu, Ama, Strotodushiti, Agni, Avarana*, etc. Available Modern books were also reviewed for patho physiology and vascular anatomy of AVN. Also relevant articles from google scholar and pubmed were also reviewed for better understand of correlation of AVN with the Ayurvedic concept.

Avascular Necrosis Disease Review

Avascular Necrosis is a degenerative bone condition. It is characterized by the death of

cellular components of the bone secondary to an interruption of the subchondral blood supply. [5] It typically affects the epiphysis of long bones at weight-bearing joints. AVN is also called osteonecrosis, aseptic necrosis, bone infraction and ischemic bone necrosis. [6] In advanced stage of disease, it may result in subchondral collapse which threatens the viability of the joint involved. Osteonecrosis is most common in the hip but also seen in the humerus, knee, and talus and more rarely seen in the smaller bones of the wrist such as the lunate or scaphoid. [7] Avascular necrosis can be caused by trauma or non-traumatic events. Causes are Trauma, Alcoholism, Steroid therapy, Goucher's disease, Caisson's disease – Dysbaric osteonecrosis, A medical condition such as Sick cell anemia or Systemic Lupus Erythematosus, etc.

Avascular necrosis from Ayurveda perspective

There are innumerable diseases as vitiated *Dosha* causes various disorders according to variations in etiology and location. According to Ayurveda a disease pathogenesis should involve the basic *Samprapti* factors like *Dosha, Dhatu, Mala, Agni, Strotas*; without the involvement of these, the disease can't be manifested and hence the detailed knowledge of these basic factors helps to understand the pathology involved and accordingly the treatment can be proposed.

Hetu of AVN

Hetu or *Nidana* means the factors responsible for producing disease i.e., etiological factors. A particular factor can be called as *Nidana* only when it develops a complete disease

process in the body either immediately or after certain period. [8]

Avascular necrosis Hetu can be classified as

- *Nija Hetu* includes causes like: - Cigarette smoking, Heavy alcohol, Steroids treatment etc.
- *Sahaj Hetu*: - Sick cell anemia can be considered as *Sahaj Hetu*.
- *Aagantuja Hetu*: -Trauma, Injury or fracture of bone.
- *Nidanarthakar Roga*: - Vascular defects secondary to disease like Diabetes mellitus.

Samprapti

Complete process of manifestation of disease is called *Samprapti*. The Pathogenesis of AVN can be understood in terms of *Samprapti Ghataka* and *Shatakriyakala*.

The events occurring in Pathogenesis of AVN are as follows

- *Rakta Dushti*
- *Dosha Dushti (Vyan Vayu)*
- *Asthi Dhatu Dushti*
- *Asthivaha Strotas Dushti*.

Rakta Dushti is the first step in the pathogenesis of AVN. *Hetu* like *Aaghata*, *Madyapana*, Fat embolism, Sick cell disease etc. causes *Raktadushti*. *Raktadushti* causes *Strotorodha (Margavrodha)* which lead to vitiation of *Vata Dosha*. *Aahar Hetu* which precipitates the vitiation of *Vata Dosha* is also responsible for *Asthivaha Strotas Dushti and Asthi Dhatu Dushti*. This ultimately causes AVN *Vyadhi*.

Samprapti Ghataka of AVN

Samprapti Ghataka includes *Dosha, Dushya, Strotodushti, Agnimandya, Ama* etc.

1	<i>Dosha:</i>	Predominantly <i>Vata</i>
2	<i>Dushya:</i>	<i>Rakta, Asthi and Majja Dhatu</i>
3	<i>Upadhatu:</i>	<i>Sira</i>
4	<i>Pradhan strotas:</i>	<i>Asthivaha, Raktavaha</i>
5	<i>Srotodushti:</i>	<i>Sanga, Vimarga-gamana</i>
6	<i>Adhishtana:</i>	<i>Vankshansandhi</i>
7	<i>Vyaktisthana:</i>	<i>Vankshansandhi</i>
8	<i>Vyadhi Swabhava:</i>	<i>Chirakari</i>
9	<i>Rogmarga:</i>	<i>Marmasthi sandhi</i>

Lakshana [9]

Sr. No.	Lakshana
1	<i>bhedo-asthiparvanam</i> (breaking type of pain in bones)
2	<i>sandhishula</i> (joint pain)
3	<i>satata ruk</i> (continuous in nature)
4	<i>mamsabalakshaya</i> (loss of strength and muscles weakness)
5	<i>asvapna</i> (disturbed sleep)

Avascular Necrosis from modern perspective

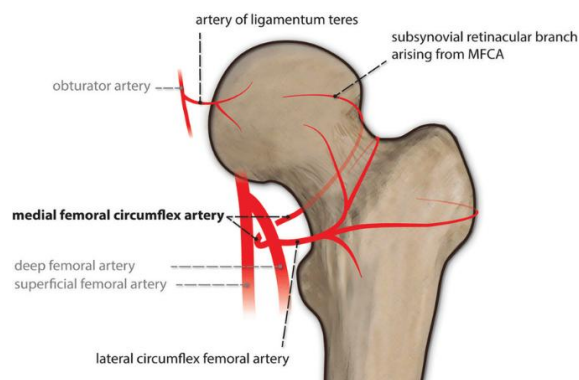
Femoral Head Anatomy

The femur is the body's longest, strongest bone, supporting weight and enabling

mobility through the thigh, hip, and knee joints. It has distinctive curvatures and a head with three regions (superolateral, posteroinferior, and peripheral). The femoral head forms a synovial joint with the acetabulum and receives blood primarily from the medial and lateral femoral circumflex arteries, which create an anastomotic ring around the femoral neck. Avascular necrosis of the femoral head (ANFH) occurs when compromised blood supply causes ischemic injury, leading to osteocyte death and eventual bone collapse.

It can be primary or secondary to other pathology i.e., alcoholism, steroid therapy, sickle cell disease, etc. ^[10] Symptoms of AVN of the femoral head are presented by groin or hip pain radiating to buttocks, anteromedial thigh or knee that is aggravated by weight-bearing and sometimes by coughing. Progressive increase of the pain worsens with time and use; patient experiences pain on rest which may be worse during the night and may have an association with stiffness. ^[11]

Vascular Anatomy of femoral head



The femoral head (FH), the proximal part of the femur, is spherically shaped with a medial depression called the fovea capitis attached to the ligamentum teres. The FH is supplied with blood by numerous arteries, yet it is one of the bone structures most susceptible to the development of avascular necrosis (AVN).

The main blood supply to the FH comes from the medial and lateral circumflex femoral arteries (MCFA and LCFA) that arise from profunda femoris artery (PFA), which originates from the femoral artery (FA). MCFA and LCFA form an anastomotic extracapsular arterial ring at the root of the Femoral Neck from which the retinacular branches pass to the Femoral head ^[12]. In addition to retinacular branches, the head of femur receives another two sources of blood supply. The first is a branch from obturator artery (OA) travelling along the ligament of the FH; however, it is not capable of keeping the head alive if other sources are lost. In most of the population (about two-thirds), the OA arises from the anterior division of the internal iliac artery (IIA). In a small percentage (about one-third), its origin is variable; it may arise from the inferior epigastric artery, followed by the external iliac artery (EIA), and, less commonly, from the iliolumbar artery ^[13]. The last source are twigs arising from the nutrient arteries of the Femoral Shaft. Poor blood supply to the Femoral Head may be due to presence of the cartilaginous epiphyseal plate that can separate blood flow in the bony FS from anastomosing with that of the FH. The FS nutrient arteries are one or two arteries that arise from the PFA and popliteal arteries. These arteries enter the bone through the

nutrient foramina, located along the Linea aspera.^[14]

It can be concluded that both iliac arteries, the IIA and EIA, share the blood supply to FH. The SGA and IGA, as well as the OA, are branches of the IIA, while the MCFA and LCFA as well as the first perforating artery arise from the PFA of FA which is the continuation of EIA in the thigh.

Etiopathogenesis of AVN

Ischemia and Hypoxia: Reduced blood flow leads to inadequate oxygen and nutrient delivery to the bone tissue, leading to ischemia and hypoxia.

Cellular Death: The lack of oxygen and nutrients triggers apoptosis of bone cells. As more cells die, the structural integrity of the bone is compromised.

Inflammatory Response: The death of bone cells initiates an inflammatory response, attracting immune cells and releasing inflammatory mediators, further exacerbating tissue damage.

Bone Resorption: As AVN progresses, the dead bone tissue is gradually broken down by osteoclasts, cells responsible for bone resorption, leading to weakening and collapse of the affected bone.

Structural Collapse: The structural integrity of the bone is compromised due to the loss of bone tissue, leading to deformity and collapse of the joint surface, resulting in pain and limited joint mobility. overall process of impaired blood supply, cellular death,

inflammation, and bone resorption remains central to the development of this condition.

In the beginning, this disease is asymptomatic. As the disease progresses, the hip can become stiffer, which is visible in the gait of the patient when he starts to limp. Symptoms include pain and decreased range of motion in the affected joint. In the early stages, this condition is asymptomatic, which makes diagnosis almost impossible. However early screening with MRI is currently the best modality for diagnosis due to its sensitivity. At a more advanced stage, standard radiograph can confirm the diagnosis. ^[15]

Result

The present review revealed that the femoral head has a delicate and limited blood supply, mainly through the retinacular branches of the medial circumflex femoral artery. Due to poor collateral circulation, interruption of blood flow can easily lead to ischemia and necrosis of bone tissue. The weight-bearing nature of the hip joint further increases the vulnerability of the femoral head to avascular necrosis. The reviewed literature showed that AVN progresses through reduced blood supply, hypoxia, death of bone and marrow cells, weakening of trabecular bone, and eventual collapse of the femoral head. In advanced stages, these changes may result in deformity of the hip joint and restricted movements.

Ayurvedic literature showed similarities between AVN and conditions such as *Asthi-Majjagata Vata* and *Asthikshaya*. Degeneration of *Asthi and Majja Dhatu* described in Ayurveda resembles the

pathological destruction seen in AVN. Symptoms like pain, stiffness, limping, and restricted hip movements correlate with aggravated *Vata Dosha*.

The study also identified correlations between modern etiological factors and Ayurvedic concepts. Trauma may be correlated with *Abhighata*, vascular obstruction with *Srotorodha*, and degenerative changes due to poor nourishment with *Dhatukshaya*. Prolonged steroid use and chronic alcohol intake may contribute to *Dhatu Dushti* and impaired tissue metabolism.

Overall, the reviewed findings suggest that AVN can be understood as a degenerative and ischemic disorder caused by vascular insufficiency in modern anatomy, while Ayurveda explains it mainly through *Vata* vitiation involving *Asthi and Majja Dhatu*.

Discussion

As time progresses, new diseases are emerging due to change in lifestyle and other infections. Ayurveda principles and concepts are endless and its application can be useful in present era as well. *Anukta vyadhi Sankalpana* is one such concepts. “*Anukta*” means any disease or drug which is not explained in Ayurvedic text. It helps not only in the understanding classical concept but also becomes base for understanding new diseases. Due to *Nija, Aagantuja, Sahaj, Anyavyadhi Upadravakruta* or *Purvjanmakruta Hetu, Rakta Dushti* occurs. In *Vatarakta*, vitiated *Vata* along with *Rakta* obstructs the *Sira* and causes pain, burning

sensation and deformities in the joints or other body parts.^[16] This is similar pathophysiology that occurred in avascular necrosis. In AVN, the supply of *Rakta dhatu* to the femoral head is interrupted due to any type of *Margavrodha*, ultimately leading to necrosis. *Margavrodha* is also responsible to aggravate *Vata dosha*.^[17] In the advanced stage, due to continuous *Vata dosha* (due to necrosis) imbalance it is further responsible for causing vitiation of *Pitta and Kapha*. It causes the death of cellular components of the bone and in advanced disease state it may result in subchondral collapse. Due to the aggravated *Vata* traversing along *Sandhi, Asthi, Majja* gives rise to *Khanja & Pangulya*.^[18] Signs and Symptoms of *Asthi* involve breaking type of continuous pain in bone and joint as well as loss of muscle strength.^[19] The '*Ashrayashrayi bhava*' of *Asthi dhatu* and *Vata dosha* helps in understanding the etiology of osteonecrosis. Whenever *Vata dosha* accumulates, *Asthi dhatu* dissipates, because *Vata* and *Asthi* are conversely proportional to each other as per the *Ashrayashrayi bhava*.^[20] As it is *Asthidhatu Dushti Janya Vata Vikar*, it can be considered as *aKashtasadhya Vyadhi*. According to symptoms and signs, avascular necrosis can be correlated with symptoms of *Asthimajjagata vata* but, as we have seen here in pathology involvement of *Rakta dhatu* is also there so from above *samprapti* and discussion it can also be correlated as *Raktadustijanya Asthi*.

Conclusion

In Ayurveda diseases mentioned are classified based on *Nidan, Dosha, Dhatu, Ama, Agni, Avarana* etc. As time progresses new diseases are identified, and the pathology of such newly emerged diseases can be understood by using the principles like *Anuktavyadhi Sankalpna, Shatakriyakala, or Dashavidha Parikshyabhava*, etc and its correlation with *Asthimajjagata vata lakshana*. Understanding the pathology of Avascular necrosis in terms of Ayurveda will help to initiate an appropriate line of treatment for the patient well-being.

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