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# Avascular necrosis of femoral head - An Ayurvedic view

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## ABSTRACT

Avascular necrosis of the bone is the death of osteocytes due to an impaired blood supply. It usually affects people between 30 and 50 years of age. Avascular necrosis is associated with long-term use of high dose steroid medications and excessive alcohol intake. It will be asymptomatic in the early stages, as the condition progress, there will be gradual increase of pain resulting in major morbidity. In Ayurveda, the *Lakshanas* of *Gambheera Vatarakta* can be co-related to the features of AVN. The possible treatment in modern include NSAIDS, core decompression, Bone transplant (grafting) and total joint replacement surgery which have their own complications and adverse effects. Hence, an attempt is made to understand AVN of femoral head as *Gambheera Vatarakta* in Ayurveda and its management is discussed.

**Key words:** Avascular necrosis, *Gambheera Vatarakta*, *Chikitsa*.

## INTRODUCTION

The four most common causes of hip joint pain in the adult are osteoarthritis, inflammatory arthritis, trauma and osteonecrosis. The terms osteonecrosis, ischaemic necrosis, aseptic necrosis and avascular necrosis are considered as synonymous.<sup>[1]</sup> Avascular necrosis (AVN) is defined as cellular death of bone components due to interruption of the blood supply. AVN usually involves the epiphysis such as femoral and humeral heads and the femoral condyles but other bones such as tarsal and scaphoid can also be affected. In children it is known as Legg-Calve-perthes

disease. It usually occurs between the ages of 3 to 12 years old, with the highest rate of occurrence at 5 to 7 years. It affects men more commonly than women, in a 4:1 ratio.<sup>[2]</sup> Avascular necrosis of the femoral head causes significant morbidity up to 20000 people per year.<sup>[3]</sup> It occurs because of an interruption in the blood supply to the femoral head, which causes bone death. This leads to collapse of the femoral head causing secondary osteoarthritis subsequently. AVN can be primary (idiopathic) or secondary or either associated with use of alcohol, glucocorticoids, sickle cell anaemia, thalassemia, polycythaemia, haemophilia) SLE, Caisson's disease, Gaucher's disease, Chronic liver disease, Radiotherapy, Chemotherapy and HIV.<sup>[4]</sup> In about 30% of patients, aetiology of non-traumatic AVN is unclear and hence termed as Idiopathic. It is sometimes seen in twins and in familial clusters suggests that genetic factors maybe involved.<sup>[5]</sup>

## Pathophysiology<sup>[6]</sup>

**Extrasosseous arterial factors** are the most important. The femoral head is at increased risk because the blood supply is an end-organ system with poor collateral development. Blood supply can be

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interrupted by trauma, vasculitis (Raynaud disease) or vasospasm (decompression sickness).

**Intraosseous arterial factors** may block the microcirculation of the femoral head through circulating microemboli. These can occur in sickle cell disease (SCD), fat embolization or air embolization from dysbaric phenomena.

**Intraosseous venous factors** affect the femoral head by reducing venous blood flow and causing stasis. These factors may accompany conditions such as Caisson disease, SCD or enlargement of intramedullary fat cells.

**Intraosseous extravascular factors** affect the hip by the increasing the pressure, resulting in a femoral head compartment syndrome. Fat cells hypertrophy after steroid administration or abnormal cells, such as Gaucher and inflammatory cells, can encroach on intraosseous capillaries, reducing intramedullary circulation and contributing to compartment syndrome. Cytotoxic factors, such as alcoholism and steroid use, have a direct toxic metabolic effect on osteogenic cells.

**Extraosseous extravascular (capsular) factors** involve the tamponade of the lateral epiphyseal vessels located within the synovial membrane through increased intracapsular pressure. This occurs after as trauma, infection and arthritis, causing effusion that may affect the blood supply to the epiphysis.

#### Clinical features

Avascular necrosis of hip usually affects men aged from 35 to 45 years and is bilateral in over 50 percent of patients, it is a pathological state with multiple aetiologies associated with a reduction in the vascular supply to the subchondral bone of the femoral head. This results in osteocyte death and progressive collapse of the articular surface followed by degenerative arthritis of hip joint. The patient is frequently asymptomatic in the early stages. Patients with AVN of the femoral head often report groin or hip pain that can radiate to the buttocks, anteromedial thigh, or knee that is exacerbated by weight bearing and sometimes by coughing. The pain

may initially be mild but progressively worsens over time and with use. Eventually, the pain is present at rest and may be present or even worsen at night, in which case, it may be associated with morning stiffness. As the disease progresses the patient may complain of an ache in the groin and walk with a limp.<sup>[7]</sup> The patient may walk with a limp and may experience loss of range of motion, both active and passive, most frequently in flexion, abduction and internal rotation, especially after collapse of the femoral head. The patient may have tenderness around the affected area. A neurological deficit may be found. The Trendelenburg sign may be positive. A click may be heard when the patient rises from a chair or after external rotation of the abducted hip. Advanced disease leads to joint deformity and muscle wasting.<sup>[8]</sup>

#### Investigations

A weight bearing anteroposterior radiograph of the pelvis along with lateral radiograph will show the classical feature of AVN including increased sclerosis in an early stages, the crescent sign indicating subchondral bone resorption. In the late stages there may be flattening indicating a segmental head collapse. X-ray based radiographic detection are not sensitive enough to detect AVN of femoral head at its onset (stages 0 and 1) while its overall sensitivity for early stage is only about 41%. MRI helps in early diagnosis and predict the prognosis of the disease. Steinberg has classified into seven stages based upon the radiological change in MRI and radiography.<sup>[9]</sup>

#### Steinberg's classification of avascular necrosis of the femoral head<sup>[10]</sup>

Stage	Description
0	Normal or non-diagnostic radiograph, bone scan or MRI
I	Normal radiograph, abnormal MRI or bone scan
II	Sclerosis and cysts
III	Subchondral collapse, crescent sign

IV	Flattening of the head, normal acetabulum
V	Acetabular involvement
VI	Obliteration of joint space

### Differential diagnosis<sup>[11]</sup>

Degenerative disease, Osteoporosis, Arthritis, Inflammatory synovitis, Epiphyseal dysplasia, Epiphyseal stress fracture, Transient osteoporosis of the hip, Osteomyelitis, Malignancy, Hemangioma, Sympathetic dystrophy and Bone marrow edema syndrome.

### Treatment

Conservative treatments such as statin therapy, bisphosphonates or nonsteroidal anti-inflammatory drugs are used alone or in combination, but they rarely provide lasting improvement. Reduced weight bearing, limiting activities or using crutches can slow the damage caused by avascular necrosis and permit natural healing. However, these patients run a risk of 85% of femoral head collapse. Surgical procedures are bone graft, core decompression and total replacement surgery which have their own side-effects.

### According to Ayurveda

The disease which occurs due to derangement of *Vata* and *Rakta* is known as *Vatarakta*. The *Lakshanas* explained in our classics about *Gambheera Vatarakta* similar to description of AVN.

**According to Charaka** - There is a description of *Khuddavata* which infers that *Vatarakta* affects the joints. The aggravated *Doshas* causes *Ruk* (pain) and *Daha* (burning sensation) in the *Ashti* and *Majja*, aggravated *Vatadosha* makes the person lame and paraplegic.<sup>[12]</sup> The site of *Vatarakta* are joints of hands, feet and joints of the body.<sup>[13]</sup>

**According to Sushruta** - Although detail explanation is not available, *Sushruta* mentions that *Mula* of *Gambheera Vatarakta* is *Padmamula* (which can be considered as Inguinal region).

**According to Ashtanga Hrudaya** - In *Gambheera Vatarakta*, *Vata* circulates in the joints, bones and marrow, produces cutting pain and curvatures (of bones and joints) moves all over the body making the person lame by one leg or both the legs.<sup>[14]</sup>

### Nidana

Excessive intake of *Lavana* (Saline), *Amla* (sour), *Katu* (Pungent) or *Snigdha* (Unctous), *Ushna* (hot), *kshara* (alkaline) *Ahara* and *Sura* (alcohol), *Asava* (wine), *Abhighata* (Injury), *Plawana* (swimming), *Vyavaya* (sexual intercourse), *Veganighrana* (suppression of natural urges) and *Hayoshtrayana* (riding on camel and horse) these *Nidanas* vitiate *Vata* and *Rakta* simultaneously.<sup>[15]</sup>

### Samprapti

Due to the respective *Nidana* → *Vata* and *Rakta* gets vitiated → *Prakopa* of *Vata* due to *Margavarodha* by *Rakta* → Leads to further vitiation of *Rakta* → *Vatarakta*.

### Samprapti Ghataka

- *Dosha* - *Tridoshaja*
- *Dushya* - *Rasa, Rakta, Meda, Asthi, Majja*
- *Agni* - *Jatargni, Dhatwagnimandya*
- *Ama* - *Jatargni, Dhatwagnimandyajanya*
- *Srotas* - *Raktavaha, Asthivaha, Majjavaha*
- *Srotodushti* - *Sanga*
- *Udbhava Sthana* - *Pakwashaya*
- *Sanchara Sthana* - *Sarvashareera*
- *Vykthasthana* - *Janu, Jangha, Uru, Kati, Hastapadanga Sandhi*
- *Rogamarga* - *Bahya and Madhyama*
- *Rogaprakriti* - *Chirakari*

### Chikitsa

In *chikitsa* of *Gambiravatarakta*, *Virechana*, *Asthapana Basti* (*Niruha Basti*) and *Snehapana* is indicated. There is no therapeutic measure comparable to *Basti* (medicated enema) for the cure of *vata-rakta*.<sup>[16]</sup> *Saghrityaksheera Basti*<sup>[17]</sup> can be

adopted as *Ghrita* and *Ksheera* helps in *Ashtidhatuposhana* and mitigate *Prakupitavata* and *Rakta*. *Panchatikta Ksheera Basti*<sup>[18]</sup> can also be advised. *Snehapana* can be given to patient of *Vatarakta* by *Guggulutiktaka Ghrita*, *Mahatiktakaghrita* and *Patoladighrita* should be used as all are having *Tikta Rasa Pradhanytha*. After *Snehapana*, *Virechana* has to be given with *Nimbamrutha Eranda Taila* as it is *Vatahara* and *Tikta Rasa Pradana* and does *Asthidhatu Poshana*.

#### Shamanoushadis

- *Kashaya* - *Ashtavarga Kashaya*, *Brihatmanjishtadi Kashaya*, *Guggulutiktaka Kashaya* and *Panchatikta Kashaya*
- *Arishta* - *Ashwagandharishta* and *Nimbamritasava*
- *Churna* - *Shaddaran Churna* and *Triphala Churna*
- *Guggulu* - *Amritha Guggulu*, *Kaishora Guggulu*, *Panchatikta Guggulu* and *Laksha Guggulu*
- *Ghrita* - *Guggulutiktaka Ghrita*, *Panchatiktaka Ghrita*

#### Pathya and Apathya

**Pathya** - *Puranagodhuma*, *Yava* and *Shashtikashali*, Soup of *Adhaki*, *Chanaka*, *Mudga*, *Masura* added with *Ghrita*, *Kshira* of buffalo, goat and cow.<sup>[19]</sup>

**Apathya** - *Divaswapna*, *Vyayama*, *Vyavaya*, *Abhishyandiahara* and intake of *Katu*, *Ushna* and *Guru Ahara*.<sup>[20]</sup>

#### DISCUSSION

Avascular necrosis is cellular death of bone components due to interruption of the blood supply. The *Nidanas* mentioned for *Vatarakta* can be correlated to the etiological factors of AVN and femoral head is affected commonly which can be understood as *Padamula* (Inguinal region). AVN leads to joint destruction, requiring surgical treatment and, in latter stages, total hip replacement. It is essential that AVN of the femoral head is diagnosed and treated early because delaying this disease by joint preserving measures have a much better prognosis. Due to lack

or loss of blood supply it causes deprivation of nutrients to *Ashtidhatu*. The treatment such as *Tikta Ksheera basti* and *Saghrutha Ksheera Basti* mitigates *prakupita Vata* by reaching its *Visheshasthana* and may aid in neovascularization.

#### CONCLUSION

Avascular necrosis is a complex disease. It is important to avoid the etiological factors to prevent further deterioration of the disease. Ayurveda treatment modalities along with daily intake of *Ksheera* and *Ghrita* serves as an effective remedy with improvement in quality of patients life, however once the disease progress to stage III conservative treatment has minimal role to play.

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