

AN OVERVIEW OF RAKTAPITTA W.S.R. TO VASCULITIS SKIN LESIONS - A CASE STUDY**Dr. Anita Patel***

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ABSTRACT

Vasculitis is a group of disorders characterized by inflammation of vessel walls. Because of rich vasculature, the skin is prone to be frequently affected in vasculitis. Cutaneous involvement in vasculitis may be primary or reflector of fatal systemic disease or evidence of association with some other systemic disorder. Vasculitis represent spectrum of disorders and in most of lesions result from the leakage of blood into skin through inflamed damaged blood vessels. Ayurvedic literature states that in *Raktapitta*, When *pitta* vitiated *Rakta* increases in amount due to *usna guna* of *pitta*, it starts flowing out of the body through different parts- upward, downward or both, or through skin pores. Thus cutaneous lesions in vasculitis are very much similar to *Raktapitta*. A 29 yr old female, who was diagnosed with vasculitis, was successfully treated based on treatment principles of *raktapitta*. After certain *panchkarma* therapies and internal medication, cutaneous lesions were completely disappeared and there is no recurrence in 6 months.

KEYWORDS: Vasculitis is a group of disorders *raktapitta*.**INTRODUCTION**

Vasculitis is group of disorder characterized by inflammation of blood vessel walls. In this disorder there is multi-organ involvement. Due to rich vasculature skin is prone to be frequently affected. Sometimes there may be subtle cutaneous lesions and predominant systemic involvement. And in some entities cutaneous involvement predominates. Misdiagnosis is frequent in these situations; cases of acute abdomen, undergoing emergency exploratory laparotomy, later may be diagnosed as small vessel vasculitis.^[1] Cutaneous vasculitis refers to inflammation of the blood vessels present in dermis and subcutaneous tissue. Hypersensitivity vasculitis is the condition precipitated by use of drugs, serum but in many case no inciting cause can be found. Hypersensitivity vasculitis is usually represented histopathologically as leucocytoclastic vasculitis (LCV). LCV is histopathologic term that defines vasculitis of small vessels in which the inflammatory infiltrate is composed of neutrophil. After de granulation, neutrophil undergo death and breakdown, a process named leucocytoclasia. In some other vasculitis infiltrates are lymphocytes or granulomatous.^[1]

LCV may be acute or chronic. Patients with chronic disease. Patients with chronic disease may experience persistent lesions or intermittent recurrence. LCV may be secondary to medications(), underlying infections (bacterial viral, mycobacterial, parasitic) collagen disorders, malignancies, ulcerative colitis, Crohn's

disease. however half of the cases are idiopathic. LCV may be localized to skin or may be associated with systemic involvement. Internal disease may manifest in the joints, gastrointestinal tract and kidneys.^[3]

The most common presentation of LCV is palpable purpuric (non blanching) lesions occur predominantly on dependent areas, mostly the lower extremities, can on forearms and hands, but unusual to find on upper part of trunk. The patients who are bedridden may develop lesions on the back or on one side. Lesions smaller than 3mm are usually referred to petechiae. Other cutaneous presentations include urticarial wheals, erythematous plaque, bullous hemorrhagic lesions or ulcers, deep skin ulcers and nodules. Systemic inflammatory response from release of chemical mediators from inflamed blood vessels gives rise to various non specific systemic manifestation. They include fever, night sweats, malaise, weight loss, arthralgia, myalgia.^[4]

Pathophysiology: Hypersensitivity vasculitis is thought to be mediated by immune complex deposition. In this form of vasculitis, circulating antigens in the body (produces by factors such as medications, infections, neoplasms) induce antibody formation. These antibodies bind to the circulating antigen and create immune complexes, which then deposit within vessels, activating complement and inducing inflammatory mediators, adhesion molecules, and local factors may affect the endothelial cells and play a role in the manifestations of

this disease. Additionally, auto antibodies, such as anti neutrophil cytoplasmic antibody (ANCA) may be associated with disease manifestations. These antibodies then bind neutrophil adhesion to vessel walls and cellular activation. Overall, however, the exact mechanisms causing hypersensitivity vasculitis remain to be elucidated.^[5]

Diagnosis: In the evaluation of patients with LCV, laboratory tests including a complete blood count, Erythrocyte sedimentation rate (ESR), Liver function test (LFT), Renal function test (LFT) and urine analysis are useful in excluding other systemic diseases. Diagnosis of LCV is confirmed on histological examination of biopsy from affected area that demonstrates perivascular and vascular leucocytic infiltrate along with fibrinoid necrosis.^[6]

Laboratory features such as normocytic, normochromic anaemia, thrombocytosis, raised C reactive protein are also common in the patients. Some patients, especially early in the course of their illness, could present with isolated systemic manifestation posing a diagnostic challenge. Conversely systemic inflammatory response is not seen in most patients with localized lesions.^[7]

Treatment: Most cases predominantly cutaneous vasculitis resolve spontaneously and others remit and relapse before finally remitting completely. In those patients in whom persistent cutaneous diseases evolve, variety of therapeutic regimen have been tried with variable results. In general treatment has not been satisfactory. When an antigenic stimulus is recognized as precipitating factor in vasculitis, it should be removed. If this is microbes, appropriate antimicrobial therapy should be instituted. Glucocorticoid therapy should be instituted, probably prednisolan 1mg/kg/day at rapid tapering where possible. If vasculitis is associated with other underlying disease, treatment of latter is often results in resolution.^[8]

Raktapitta-Bheda

Bheda	Urdhwanga	Adhoga	Tiryagga/Ubhayaga
Hetu	Snigdha, Ushna,	Rooksha, Ushna	Both
Dosha	Kapha	Vata	Tridosha
Sthanam	Amashaya	Pakwashaya	Sarvanga
Marga	7 – Mukh, Nasa 2, Karna 2, Akshi 2	2 – Guda, Mootra	Loma Koopa (Asankhyeya)
Sadhyatwa	Sukhasadhya	Yapya	Asadhya
Shodhanachikitsa	Virechana (good for pitta, but not so good for kapha)	Vamana (not bad for pitta, but contra-indicated for vata)	Vamana, virechana, anarha (Vamana cannot suit Vata and Virechana cannot be given for Kapha)
Shamanachikitsa	Kashaya, Tikta	Madhura	- (Rasa pacifying one dosha vitiates the other and vice versa)
Apunarbhava-upakrama	Kapha samanya upakrama	Vata Samanya Upakrama	Vata – Kapha samanya chikitsa

Generally, In *Raktapradoshaj* diseases, *Virechana* (purgation), *Langhana* (fasting), *raktamokshana* (blood

Ayurvedic review: As the name itself suggests that the disease *raktapitta* is caused by vitiation of *rakta* and *pitta* together due to their *ashrya-ashrayee* relationship. But it is also important to understand that *raktapitta* is different from those group of disorder caused due to vitiation of *rakta* or *pitta* independently. Charaka describes it as *Mahagada* (dreadful), *Mahavega* (rapid onset) & *Asukari* (acute).^[9] It should be treated immediately as disease onset, progress and consequences rapidly sets in.

When *pitta dosha* with increased *usna tikshna gunas* gets more vitiated, it vitiates *rakta* due to *asyaya sthana*. This *pitta* vitiated *rakta* increase in amount and starts flowing out of body, through various outlets. Gananath sen has described disease as bleeding without any trauma or external injury, hence, *nija hetujanya*.^[10]

Charaka has described it immediately after *jwara* as, it occurs due to *santapa* caused by *jwara*, while Sushruta has explained it after *pandu* as both are having common causative factors. *Rakta pitta* is one of the *rakta prodoshaj vyadhi*. In *Vidhisonitiya* chapter Charaka says, all *raktaprodoshaja* diseases should be treated like *raktapitta*.^[11]

The *rakta* vitiated by *pitta* tries to get outside due to increased intra vascular pressure.

When *raktapitta* is associated with *kapha*, vitiated *rakta* comes out through upward direction, When it is associated with *vata*, vitiated *rakta* expels out through downward direction, through anal, urethral and in female vaginal openings. These are *urdhwa* and *adho gatis* of *raktapitta* with stimulation of *udana* and *apana vata* respectively. Apart from these Charaka has explained in *chikitsasthana-4*, *asankheya* or *antiki gati*, where vitiated *rakta* comes out through *lomakoopas*.^[12]

letting) can be administered as per condition of patient and stage of the disease. Specific treatment of *raktapitta*

is *pratimarga doshaharanam* i.e. in *urdhwagati raktapitta*, vitiated *doshas* should be removed through *virechana* (therapeutic purgation) and in *adhogati raktapitta*, *doshas* are expelled out through *vamana* (Therapeutic emesis).

Along with removal of vitiated *doshas*, *tikta, kashaya rasa* medicines are effective in *Urdhwaga raktapitta*.^[13]

MATERIALS AND METHODS

29 yrs old female who was residing at Germany since 3 years after marriage, was admitted in Hospital in Germany for exacerbation of skin lesions- palpable erythematous purpura with severe pain and burning sensation from 26/03/17 to 21/04/17. She was allergic to Novalgin and Arcoxia. Patient was known case of Hypothyroidism and PCOS since 9 yrs and was on medication for it. During the course of admission her skin biopsy investigation diagnosed it as Leucocytoclastic purpura. Among other hematological findings CRP, ESR were raised and Urine analysis also suggested, presence of erythrocytes and leucocytes in urine. Her stool and urine sample suggested possible infections. ANA (Antinuclear antibodies) titre was noticeably erratic. But it was found non conclusive for collagenosis. During hospital stay there was also triggered metrorrhagia. There she was treated with Prednisolan and local therapy with betamethasone.

After discharge she came to India in First week of May and visited Sri Jayendra saraswathy hospital with multiple reddish purplish palpable cutaneous irregular lesions around 1-3 mm in size in scattered manner on extremities and trunk especially on lower limb.(pic-1) But some lesions were barely palpable. In lower limb few urticarial lesions were also observed. Patient was feeling itching and burning sensation in these lesions. Patient was also feeling unusual intolerance for heat and cold. After careful examination, medical history and

Before Treatment



considering *nidanapanchaka*, the case was diagnosed as *Tiryak raktapitta*.

For one week only oral medication was given. Starting with, *Patolkaturohinyadi Kashayam* was given for mild *sodhana*. Next week patient was admitted in patient, oral medications were *Mahatiktakam Kashayam* and *Kumaryasavam* and *Satavari ghritam*, followed by *manjishthadi kashayam* and *Kumaryasavam* and *Satawari ghritam*^[14] During In Patient admission few external *panchkarma* procedures were adopted. During whole period patient was not taking any other allopathic medications.

In Hospital procedures-

1. *Virechana* with *Nimbamritadi eranda taila*-50 ml. *Virechana vegas* seven times
2. *Aragwadha - amalaki siddha takradhara* for 1 week.
3. *Yoga vasti* with *manjishthadi Ksheervasti* and *Mahatiktak ghrita*.

After two weeks procedures cutaneous lesions reduced in size and discolouration got fainted.(Pic-2) Patient's temperature tolerability also increased. Patient went back to Germany in four days. Same oral medications were continued. No new lesions recurred but again patient was feeling same hot and cold intolerance. Again after 4 months patient returned back for treatment as was advised to her. Similar treatment was repeated for two weeks I.e. *Virechana, takradhara and Vasti* except this time *Manjishtha-Guduchi siddha ksheeravasti* was administered.

RESULT

After treatment her cutaneous lesions has greatly reduced and faintly visible. After the treatment since last five months there are no fresh development. Her menstrual cycles are regular and normal. Still she feels occasionally intolerance for temperature.



After Treatment



DISCUSSION

Kumaryasavam is best medicine for blood related ailments such as anemia, *yakrit* and *pleeha vikara* and endocrine deficiency as well as it is hemostatic, hepatoprotective, blood purifier and helps for nourishment of *rakta dhatu*.^[15] *Virechana* has indicated in *prakupita pitta dosha* as well as in *raktapradoshaja vyadh*.^[16] *Virechana karma* is *vatapittashamak*, *lekhana*, *karshneeya*, *srotosudhaikara* which normalises the state of *Agni*. In turn it helps for *raktaprasadana*. *Virechana* may be useful to expel excessive *dravata* of *pitta* and *pitta* present in rest of *avyavas*. *Virechana* eliminates excessive accumulation of *doshas*, and due to *srotosudhi* nutrition to skin is maintained. It also shows significant change in ESR, Improves absorption power suggesting purification of microchannels in the body.^[17] It reduces inflammatory response in the body.^[18]

Takradhar^[19] has antiinflammatory action and regulates cell proliferation. In *Takradhara*, lactic acid in buttermilk helps moistening the skin and good for transdermal absorption. *Basti* is considered as half or whole of entire therapeutic measures and it is quickest and best method of providing strength and immunity.^[20]

(ASK-7/35) *Manjishthadi ksheera basti* has praised in *rakta pitta avarana vata*.^[21]

CONCLUSION

In this case study, cutaneous manifestation of vasculitis has treated as *Tiryaka raktapitta*. Ayurvedic treatment like *Virechana*, *Takradhara* and *basti* has successfully treated the cutaneous manifestation along with some internal medications. It is an effort to understand the concept of *tiryak raktapitta* applied in vasculitis.

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