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AN AYURVEDIC PERSPECTIVE OF VASCULITIS IN CHILDREN

Dr. Sudha Singh*[1] and Dr. Deepak S. Khawale[2]

[1]Associate Professor, Department of Kaumarbhritya, P.D.E.A. S College of Ayurveda and Research Centre, Nigdi, Pune, Maharashtra- 411044 (India).

[2]Professor & Amp; HOD, Department of Kaumarbhritya, Dr.D.Y.Patil College of Ayurved Research Centre, Pimpri, Pune, Maharashtra – 411044 (India).

***Corresponding Author: Dr. Sudha Singh**

Associate Professor, Department of Kaumarbhritya, P.D.E.A. S College of Ayurveda and Research Centre, Nigdi, Pune, Maharashtra- 411044 (India).

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ABSTRACT

The primary systemic vasculitides in childhood are quite rare and the etiopathogenesis for most of them is not clearly understood. However, it is usually thought that mostly infectious, environmental triggers induce an aggravated inflammatory response in genetically susceptible individuals. As per classification Kawasaki disease and Henoch–Schönlein purpura (HSP) are common vasculitis in children. The modern treatment protocols for acute phase are steroids and symptomatic management. In these kinds of diseases long term preventive management required as explained in ayurveda to reduce the recurrence and protective as well as rejuvenating regimen for end organ. The ayurvedic concept and consideration has been discussed here to manage vasculitides.

KEYWORDS: Vasculitides, Vasculitis, Purpura, Rejuvenating.

INTRODUCTION

Vasculitis is chronic inflammation resulting in necrosis of blood vessels due to narrowing or occlusion of the lumen.Vasculitis may occur as a primary disease (idiopathic) or as a secondary response to an underlying disease e.g., hepatitis B infection. Based on the size of the vessel affected, it can be classified into small-vessel, medium-vessel, or large-vessel vasculitis.[1]

Term “Vasculitides” are a heterogeneous group of autoimmune diseases, all characterized by inflammation of blood vessels (vasculitis) and subsequent ischemia and damage to the organs supplied by these vessels.[2,3,4]

The classification of vasculitis has been a challenging problem for decades.Seven types of established vasculitis are giant cell arteritis (GCA), Takayasu arteritis (TA), Wegener's granulomatosis (WG), polyarteritis nodosa (PAN), Henoch–Schönlein purpura (HSP) and hypersensitivity vasculitis (HSV). Latest classification is as following-

Chapel Hill Consensus Conferences 2012 Classification criteria.[5] (Table-1).

Serial No.	TYPES
I.	**Large-vessel vasculitis (LVV)** • Takayasu arteritis • Giant cell arteritis
II.	**Medium-vessel vasculitis (MVV)** • Polyarteritis nodosa • Kawasaki disease
III.	**Small-vessel vasculitis (SVV)** **A. Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis** • Microscopic polyangiitis • Granulomatosis with polyangiitis (Wegener granulomatosis) • Eosinophilic granulomatosis with polyangiitis (Churg– Strauss syndrome) **B. Immune complex SVV** • Anti-glomerular basement membrane (anti-GBM) disease • Cryoglobulinemic vasculitis • IgA vasculitis (Henoch–Schönlein) • Hypocomplementemic urticarial vasculitis (anti-C1q vasculitis)

IV.	**Variable vessel vasculitis** • Behçet's disease • Cogan's syndrome
V.	**Single-organ vasculitis** • Cutaneous leukocytoclastic angiitis • Cutaneous arteritis • Primary central nervous system vasculitis • Isolated aortitis etc.
VI.	**Vasculitis associated with systemic disease** • Lupus vasculitis • Rheumatoid vasculitis • Sarcoid vasculitis etc.
VII.	**Vasculitis associated with probable etiology** • Hepatitis C virus-associated cryoglobulinemic vasculitis • Hepatitis B virus-associated vasculitis • Syphilis-associated aortitis • Drug-associated immune complex vasculitis • Drug-associated ANCA-associated vasculitis • Cancer-associated vasculitis etc.

Henoch-Schönlein purpura (HSP) also known as Immunoglobulin A vasculitis (IgAV), is one of the most common vasculitis of childhood. It affects predominantly small vessels with the presence of immunoglobulin A1 (IgA1) dominant immune deposits.

HSP is typically a disease of children between the ages of 3 and 10 years. 50% of all cases occur at or before the age of 5 years. Males are affected twice as often as females.The overall incidence in children has been estimated to be 13.5 cases per 100,000.[6]

The cause of HSP is unclear. Studies have suggested the increased serum level of IgA1 anti-endothelial cell antibodies (AECA) as the first hit. It may occur after an upper respiratory infection. Children more frequently present with abdominal pain before the appearance of non-thrombocytopenic palpable purpuric rash.

The rash is especially common on the Gravity dependent areas like lower extremities and buttocks. The main explanation of these symptoms is immune complex deposition in the intestinal vessel walls. Melaena and haematemesis were present.

Diarrhoea is more common in adulthood. It is treated with supportive care and is usually self-limited, although it can cause glomerulonephritis and death. The prognosis is excellent in those cases without renal disease.

Thirty percent of patients who recover from HSP may have recurrent symptoms as late as 7 years after the acute phase, and those with renal involvement may have lifelong problems.The patient should be monitored for rare but more serious complications, such as hemorrhagic involvement of the renal, pulmonary, gastrointestinal, genitourinary, and central nervous systems or the joints, which usually occurs within 4 weeks of initial presentation but may occur as late as 8 weeks.

Laboratory findings are unspecific and include (usually) mildly elevated inflammatory parameters (ESR and CRP), normal or slightly reduced serum C3 and/or C4, the absence of high-titer autoantibodies (particularly ANCA and ANA) and, in 50% of all cases, elevated serum IgA and/or IgM. Fecal occult blood tests and urinary specimens are recommended at diagnosis. Since glomerulonephritis can manifest after the initial phase, regular urinary analysis at least for the first 6 months after disease-onset are recommended.

Treatment

Treatment depends on clinical presentation and organ involvement. Non-steroidal anti-inflammatory drugs (NSAIDs) or acetaminophen /paracetamol can be considered for analgesia in cases with arthritis or arthralgia. For gastrointestinal involvement treatment with corticosteroids (usually 1–2 mg/kg/day for 1 week, followed by taper over 2–3 weeks) can be considered. However, intestinal perforations have been seen under treatment. Substitution of factor XIII can be discussed for gastrointestinal bleedings. Studies indicate that early corticosteroids are not effective in preventing HSP-associated nephritis.

Treatment options for severe renal involvement by HSP include the following:

- High-dose corticosteroids, either alone or combined with immunosuppressive agents such as azathioprine, cyclophosphamide, or cyclosporine;
- High-dose iv immunoglobulins;
- Plasma exchange or plasmapheresis;
- Corticosteroids combined with urokinase and warfarin;
- Renal transplant in case of renal failure.

Ayurvedic View

Disease related references in ayurveda

As per ayurveda we can consider vasculitis as siragata Kupita (Aggravated) vata and after that manifest as tiryak rakta-pitta.

Lakshana of Shiragata Kupitvata

- Sharir mand ruk (body pain)
- Shoth (oedema) or
- sushyati (muscle atrophy)
- spandan (twitching /pulsation)
- siratanvya /mahatya (narrowing /dilatation).[7]

Tiryak Rakta Pitta Lakshan-Asadhya as per acharyas if bleeding from romkupa /skin.
if there is vatanubandh in raktapitta (frequent episodes).
Raktapitta is described in pittaj-nanatmajvikaar.
Vyaas sankoch, shola are karma of vata.[9]

Raktavrita Vayu-burning inside tvak and mamsa with, macular rashes wheals on skin redness.[15]

Samprapti (Pathogenesis) of raktapitta

Dosha-pitta pradhan (vata and kapha)
Dushya rakta dhatu
Marga -raktavahashrotas
Adhisthan-yakrut pleeha raktavahishira,
Udbhav sthan –amashaya.[10]
Madhyamrogmarga-
sira,snayu,kandarashir,hridaya,basti,asthi,sandhi.[15]

Treatment consideration

Madhur Tikta snigdha formulation Should Be consider to reduce the sign and symptoms followed by Deepan pachan dravya to normalize function of agni at dhatu level also .Along with this vishagna formulation should be added for early response considering dushi-visha.Then end organ protective formulations should be continued along with long term nidana pariverjana mainly on ritu sandhi and in pitta prakop kala(sharad ritu).

Herbs and Formulations (Table -2).

Sr.No.	Formulations type	Formulations described for raktapitta in various texts
1	BHASMA	Vaidurya bhasma,Mukta bhasma, Gairik ,Shankha bhasma,Suvarna,bhasma,
2	HERBS (as for raktapitta and rejuvenating for end organ also)	amalaki,Sugandhabala,shatavari,gukshura,vasa,usher.[8] Giloy punarnava,anantmool,Nagabala,Arjun
3	GANA	ushiradigana,Drakshadigana,priyanguadigana.[8]
4	GHRITA	vasa ghrita shatavaryadighrita.[8] For shiragatvata- Dashmooladimajja sneha,[6] Triphaladichatuhsneha,[7] Durvadya Ghrita – Local Application In Romakupa Raktapitta,[11] Tiktak ghritam/mahatiktak ghritam,shirisha etc raktapitta har
5	SIDDHA DUGDH	Vidarigandhadigana siddh cow Dugdha + sugar n honey,[8]
6	BASTI	**Raktapittahar Basti,[12]** 1-Manjistha,Sariva, Ananta,Payasya Jestamadh Siddha Dugdha. 2-Chandan, Draksha, Dhatri, Neelkamal,Sharkara Madhu **Madhukadi Tailam** Indicated For Anuvasana Basti In Rakta Pitta.[13] **Mustadi Rajyapana Basti** Indicated In Raktapitta As Asthapan Basti.[14]
7	Other drug as per concept and practical use	Bilwadi gulika Dushivishari gulika

Preventive measures as per ayurveda

Nidana Parivarjan-all nidan /aharvihar janya causes of raktapitta described in our texts should be avoided like excessive milk consumption in hot weather frequent ingestion of incompatable diet like rohini shak kulathi sevana followed by milk might be precipitate the next bleeding episode.

If there is unexplained anorexia nausea vomiting swarabheda etc then we have to consider these as prodromal symptoms of raktapitta and check the food habit.

When doshas aggravates excessively then raktapitta occur from skin pores of body.

Visha /ama concept

In autoimmune /exaggerated immune response in genetically susceptible individuals the immune complexes can be related with toxins or ama as per ayurvedic concept.

Bilwadi agada like medicines must be prescribed as ayurveda has a very nice concept of Dushi Visha (latent/cumulative poison) said to be responsible for the delayed action and cumulative toxicity on the body.

According to Charaka, latent poison (Dushi Visha) vitiates Raktadhatu (blood) and causes skin diseases such as Kitibha and Kotha. Because of Kapha Dosha Avarana, defective digestion (Agnimandya) and defective metabolism (Dhatwagnimandya) occurs which in turn leads to Apakata of latent poison (Dushi Visha) and stays

for long time in the body without producing any signs and symptoms.[16]

DISCUSSION

Vasculitides" start with inflammation of blood vessels (vasculitis) and subsequent ischemia and damage to the organs supplied by these vessels.Here the cause is exaggerated immune response in genetically susceptible individuals.The disease continued as chronic inflammation and frequent episodes can occur as per type of vasculitis.So longterm management required.

Initial management in acute phase is allopathic medicines as its an medical emergency in children up to first few days and strict intensive care required.By next week ayurvedic pitta pacifying formulations described for raktapitta in ayurvedic texts can be added along with nidanaparivarjan.

As vasculitis can be correlated with siragata Kupita (Aggravated) vata and after that manifest as tiryak rakta-pitta.

Long-term or frequent intake of incompatible diet ,frequent food allergy/intolerance, drug allergy or toxicity with some prolonged using drug, chronic digestive problem or post acute viral infection induced visha (toxins) can be considered as first manifestation and frequent episodes of vasculitis.

Frequent /Daily Agadapana (anti-toxic drugs) should be done with Bilwadi Agad/Dushi Vishari agada[16] with agni pacifying herbs at dhatu level and rasayana dravya for end organ effected in disease.

CONCLUSION

Vasculitis are rare conditions in children and young people, but its an medical emergency Timely and accurate diagnosis by modern tools and initial modern treatment are essential. After acute phase ayurvedic parameter of assessment and indicated formulations as per prakruti of person and involvement of dosha can reduced the frequent episodes and improve the quality of life. As many diseases manifest like this required long term treatment so combined approach is need of current era.

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