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Critical study of valvular heart diseases W. S. R. to Ayurvedokta Shatkriyakala

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ABSTRACT

Valvular heart diseases leads to definite death by various complications if not treated early. Although it may start as the simple throat infection but eventually it may affect the heart valves if not treated Shatkriya Kala is a unique pathological concept in Ayurveda developed with aim of treating the diseases at various stages of pathogenesis. Ancient Ayurvediac acharyas had stated 6 such stages of therapeutic intervention with reference six to stages pathogenesis namely sanchaya, prakopa, prasara, sthansanshraya, vvakti bheda.. If disease is treated in early stages, it does not lead to complications.

There are vishahra a,d aamhar drugs in Ayurveda which could be potentially used to treat valvular affections. Hence, it is essential to apply the Ayurvedic concept of pathogenesis like 'Shat Kriyakala' to valvular heart disease so as to study it from Ayurvedic pathological point of view and to evolve a strategy to deal with it based on the Ayurvedokta principles of treatment particularly in terms of shatkriyakalas.

The study of valvular heart disease with reference to Shat Kriyakala, showed that the pathogenesis of valvular heart disease can very well be arranged in Ayurvedokta six stages of Shat Kriyakala and treatment could be framed as per different stages.

With the help of modern investigations, these six stages could be diagnostically confirmed.

KEYWORDS – Shatkriyakala, Valvular heart disease

INTRODUCTION:

Valvular heart diseases leads to definite death by various complications if not treated early. Although it may start as the simple throat infection but eventually it may affect the heart valves if not treated early. Shatkriya Kala is a unique pathological concept in Ayurveda developed with aim of treating the diseases at various stages of pathogenesis. Ancient Ayurvediac acharyas had stated 6 such stages of therapeutic intervention with reference to six stages of pathogenesis namely sanchaya, prakopa, prasara, sthansanshraya, vyakti bheda.. If disease is treated in early stages, it does not lead to complications. There are vishahra a,d aamhar drugs in Ayurveda which could be potentially used to treat valvular affections. Hence, it is essential to apply the Ayurvedic concept of pathogenesis like 'Shat Kriyakala' to valvular heart disease so as to study it from Ayurvedic pathological point of view and to evolve a strategy to deal with it based on the Ayurvedokta principles of particularly treatment in terms of shatkriyakalas.

MATERIAL & METHOD

Literary research method is adopted for the present study. Critical study of Ayurvedic as well as modern literature pertaining to the subject is carried out to come to the logical result and conclusion.

Review of Literature

Shat Kriyakala

(Six Stages of pathogenesis and Treatment)

संचयं च प्रकोपं च प्रसरं स्थानसंश्रयम्।। व्यक्तिं भेदं च यो वेत्ति दोषाणां स भवेभ्दिषक्।। सु.सू. 21/36

- 1. Sanchaya (Stage of Accumulation of Doshas)
- 2. Prakopa (Stage of Quantitative increase of Doshas)
- 3. Prasara (Stage of Spread of Doshas)
- 4. Sthansanshraya (Stage of localization of Pathology)
- 5. Vyakti (Stage of Appearance of Features)
- 6. Bheda (Stage of Differentiation, Chronic course)

[Ref. - S.Su. 21/36-37]

संचयेऽपहृता दोषा लभन्ते नोत्तरा गतीः।। ते तूत्तरासु गतिषु भवन्ति बलवत्तराः।। सु. सू. 21/37

As per acharya Sushruta, if the aggravated dosahs are flushed out of the body during stage of accumulation then disease does not advance to the further stage. Hence it is essential to identify

these stages properly and intervene appropriate time.

Valvular Heart Disease (VHD) Infective endocarditis And Valvular Affection

It is a microbial infection of heart valves

or the endocardium in proximity to congenital or acquired cardiac defects. Infection of the endothelial linings of arterial aneurysms or AV fistula produces a similar illness. It is increasingly seen inpatients of VSD, AR, MR, AS, PDA. Endocarditis leads to aggregation of fibrin, platelets, and other blood products at the site of infection. This produces a vegetation which is relatively avascular and tends to isolate the infective organism from the host defense and antimicrobial agents. Valve destruction produces worsening regurgitation and leads to heart endocarditis failure. In caused staphylococcus aureus valve destruction is rapid and local abscess formation occurs commonly. In less aggressive infection (from streptococcus viridians) the progression of disease is slower and large craggy vegetations develop which are prone to form embolism.

Endocarditis occurs at a site where blood flows through narrow orifice and at a high velocity, from a high to low pressure chamber. A decrease in lateral pressure lowers perfusion of the intima resulting in an area more susceptible to infection. This is the location where infective endocarditis initially develops. Hence, it occurs on the right side in VSD and on the pulmonary artery in PDA. Endocarditis does not usually occurs when there is any small pressure gradient as in ASD or when the congenital defect is large enough to abolish the pressure gradient.

Infection occurs along the edges of the heart valves. It is more common on the left side with mitral and aortic regurgitation being the commonest valve lesion complicated by endocarditis. In drug addicts the valves in the right heart are usually affected.

Hypertrophic cardiomyopathy, syphilitic aortic regurgitation, prolapsing mitral valve and atherosclerotic valve lesion may also be rarely complicated by endocarditis.

The extracardiac manifestations result either from the embolization or from the deposition of immune complexes. The later is responsible for arthralgia, Roth spots, focal glomerulonephritis and acute vasculitis.

The pathological process of valvular involvement results after some years in the form of valve thickening, cusp fusion, calcium deposition, a narrowed (stenotic) valve orifice and progressive immobility of the valve cups. When the normal valve orifice area of 5 cm2

is reduced to less than 1 cm2, severe stenosis results. In order to mitral maintain the sufficient cardiac output, the left atrial pressure increases causing left atrial hypertrophy and dilatation. Consequently, pulmonary venous. pulmonary arterial and right heart pressures also increases. The increase in pulmonary capillary pressure followed by the development pulmonary oedema particularly when the get rhythm disturbed fibrillation and tachycardia. This is partially prevented by alveolar capillary thickening and pulmonary arterial vasoconstriction (reactive hypertension). Pulmonary pulmonary hypertension leads to right ventricular hypertrophy, dilatation and failure. Right ventricular dilatation results in tricuspid regurgitation.

SYMPTOMS

- Usually there are no symptoms until the valve orifice is moderately stenosed. Progressive severe dyspnoea because of pulmonary venous hypertension and recurrent infective bronchitis.
- A cough productive of bloodtinged, frothy sputum is quite common, and occasionally frank hemoptysis may occur.

- The development of pulmonary hypertension eventually leads to right heart failure and its symptoms of weakness, fatigue and abdominal or lower limb swelling.
- The large left atrium favours atrial fibrillation, giving rise to symptoms such as palpitations.
- Atrial fibrillation may result in systemic and pulmonary emboli, which give rise to cerebral, mesenteric, renal and pulmonary infarcts.

SIGNS

Face - Severe mitral stenosis with pulmonary hypertension is associated with the characteristic mitral facies or malar flush.

There is a bilateral, cyanotic or dusky pink discoloration over the upper cheeks that is due to arteriovenous anastomoses and vascular stasis.

Pulse

At first the pulse is regular (sinus rhythm) but later the irregular pulse of atrial fibrillation usually develops. The onset of atrial fibrillation often causes a dramatic clinical deterioration.

Jugular veins

If right heart failure develops there is obvious distension of the jugular veins, the 'a' wave will be prominent provided that atrial fibrillation has not supervened.

The severity of mitral stenosis is judged clinically on the basis of criteria such as:

- The time between the closure of the aortic valve and the opening of the mitral valve. Thus the shorter the A2-OS time the more severe the stenosis. This is because it takes less time for the left ventricular pressure to fall to the high left atrial pressure which occurs in severe mitral stenosis.
- As the valve cusps become immobile, the loud first heart sound softens and the opening snap disappears.
- When pulmonary hypertension occurs, the pulmonary component of the second sound is increased in intensity and the mitral diastolic murmur may become quieter because of the reduction of cardiac output.

<u>Potential Ayurvedic drugs for valvular</u> <u>heart diseases :</u>

Ankot [Alangium salvifolium]- It detoxifies the microbial toxins from blood circulation. It also brings down the throat pain. (D.Ni. 1/259)

Yawa-kshara [Potassi carbonas]— It lowers the Kapha by its pungent and hot property. It cures the pain from toxins. It brings out the lysis of urinary calculus. It contains KCl2 (D.Ni. 2/18)

Kantakari [Solanum xanthocarpum] - It lowers the increased coughing and respiratory rate by its bitter, pungent and hot property. It cures the heart diseases [D.Ni. 1/96]

Sarpagandha [Rauwolfia serpentine] – It lowers the Kapha by its bitter, pungent and hot property. It also acts as antimicrobial agent. [D.Ni. 4/92]

Manshila [Arsenic rubrum] - It is pungent, bitter, hot, nullifies the bacterial toxins and their fever attacks. [D.Ni. 3/96]

Gandhaka [Sulphur] – It is pungent, bitter, hot, antimicrobial and kapha lowering agent. [D.Ni. 3/104]

Bhargi [Clerodandrum seratum] - It lowers the Kapha and meda (lipids), Aampachaka, Vishahar . [S.Su. 38/17]

Vacha [Acorus calamus]— It lowers the Kapha by its bitter, pungent and hot property and also acts as diuretic. [D.Ni. 2/8]. It also cures the heart disease. [D.Ni. 2/7]

OBSRVATIONS

- **1. Sanchaya** (Stage of Accumulation wrt VHD)
- This stage characterizes with accumulation of Kapha and fat in the body due to consumption of Kapha aggravating factors.
- This stage characterizes with group A streptococcal pharyngeal

infection. Features of pharyngitis like sore throat, cough , fever develops.

- Investigations To Diagnose The stage:
- CBC/ESR
- Treatment.
- This is the first stage of treatment. Treatment in the form of gargling with Trifla decoction. Tablets of sukshma Trifla PO, Khadiradi and Lavangadi wati for sucking.
- **2. Prakopa** (Stage of Quantitative increase wrt VHD)
 - This stage characterizes with production of pharyngeal exudates reflected as increased spitting tendency and cough.
 - Investigations To Diagnose The stage:
 - Throat swab culture and sensitivity
 - Treatment
 - This is second stage of treatment. Vamana (emesis) should be rid applied to get of the accumulated Kapha. Antibiotics to be given (combination of ampicillin and cloxacillin).
- **3. Prasara** (Stage of Spreading wrt VHD)
 - This stage characterizes with immunological cross reaction between streptococcus antigen and myocardial sarcolema. This occurs

after 2 weeks of initial infection.

Polyarthritis, fever, carditis

(murmurs) may occur.

- Investigations To Diagnose The stage :
- ASO titre
- Treatment.
- This is third stage of treatment.
 Treatment with penicillin group of antibiotics.
- **4. Sthansanshraya** (Stage of localization of pathology wrt VHD)
- This stage characterizes with formation of granulomatous lesion in the subendocardium of the left ventricle. Small warty vegetations develop on the valves may J-R A resulting in little regurgitation. This is fourth stage of treatment. Ankot, Vacha. Yawakshar, Manshil, Gandhak, Bharngi all may be instituted for the treatment at this stage in various forms.
 - Investigations To Diagnose The stage :
 - ASO Titre
 - ECG
 - Treatment
 - This is fourth stage of treatment.
 - Treatment with Injections of Benzyl Penicillin. Supportive Ayurvedic treatment in the form of Ankol Kwatha, Arjuna Kwatha.

- **5. Vyakti** (Stage of development of features wrt VHD)
 - This stage characterizes with valve thickening, cusp fusion and calcium deposition and progressive immobility the valve cusps. It may take years to develop. When the normal orifice area of 5 cm2 is reduced to 1 cm2, severe mitral stenosis occurs resulting in features like exertional dyspnoea, fatigue and features of heart failure. Other features like Soft first heart sound – due to partial closure of valve cusps.

Pansystolic murmur – as regurgitation occurs throughout the systole.

Mid systolic click – due to sudden prolapsed of the valve and tensing of chords.

Third heart sound - because of the sudden rush of blood back into the dilated left ventricle in early diastole.

- Investigations To Diagnose The stage:
- **ECG** - The ECG shows the features of left atrial delay (bifid waves) and left p ventricular hypertrophy as manifest by tall R waves in the left lateral leads, e.g. leads I, aVL and V6, and deep

- waves in the right-sided precordial leads, e.g. leads V1 and V2 (R > S in V1).
- **N-Ray The chest X-ray usually shows a enlarged left atrium with straightening of the left border and a double shadow on the border of the right and left atrium.

 Late in the course of the disease a calcified mitral valve may be seen on lateral view.

 The signs of pulmonary edema or pulmonary hypertension may also be seen with the severity.
- 2-D Echocardiography shows valvular stenosis or regurgitation.

■ Treatment

- Treatment with diuretics provides symptomatic relief. Ayurvedic mutral dravyas like Gokshur, Sahachar, punarnawa may be tried. Vacha, Sarpagandha may be tried. Definitive treatment in the form of surgery like Valvotomy or Valve replacement may be done as per the condition.
 - **6. Bheda** (Stage of complications wrt VHD)
 - This stage characterizes with development of complications of stenotic valves like atrial fibrillation, pulmonary

hypertension, chest infections and embolic infarcts.

- Investigations To Diagnose The stage :
- 2-D The **Echocardiography**movement of the valve cusps and the rate of diastolic filling of the left ventricle may be measured by 2 dimensional echocardiography. Severe mitral stenosis produces immobility of the valve cusps and slow filling the ventricles. Continuous wave (CW) colour doppler used to estimate peak mitral trans-valvular gradient and the valve area. The presence of tricuspid regurgitation can be used to estimate pulmonary arterial The pressure. echocardiogram appearances in deciding helps surgical intervention to be done.

Treatment

■ Treatment of the complications should be done. For. E.g. DC cardioversion for fibrillation, medical cardioversion with tablet digoxin. Oral Warfarin to prevent thromboembolism. Antibiotics as prophylaxis

DISCUSSION

During stage of accumulation, infection of pharynx with group A streptococci leads to local congestion and accumulation of inflammatory exudates. Features of pharyngitis like sore throat, cough, fever develops. Investigations like CBC/ESR could be done at this stage. This is the first stage of treatment. Treatment in the form of gargling with Trifla decoction. Tablets of sukshma Trifla PO, Khadiradi and Lavangadi wati for sucking. **Prakopa** stage characterizes with production of pharyngeal exudates reflected as increased spitting tendency and cough. Throat swab culture and sensitivity could be done at this stage to know the type of microorganism and its sensitivity to the drugs. This is second stage of treatment. Vamana (emesis) should be applied to get rid of the accumulated Kapha. Antibiotics to be given (combination of ampicillin and cloxacillin). Prasara stage characterizes immunological cross reaction between streptococcus antigen and myocardial sarcolema. This occurs after 2 weeks of initial infection. Polyarthritis, fever, carditis (murmurs) may occur. Investigations like ASO titre could be done at this stage. This is third stage of treatment. Treatment with penicillin group of antibiotics. Sthansanshraya stage characterizes with formation of granulomatous the lesion in

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subendocardium of the left ventricle. Small warty vegetations may develop on the valves resulting in little regurgitation. Investigations like ECG could be done at this stage.

This is fourth stage of treatment. Injections of Benzvl Penicillin. Supportive Ayurvedic treatment in the form of Ankol Kwatha, Arjuna Kwatha. Vvakti stage characterizes with valve thickening, cusp fusion and calcium deposition and progressive immobility of the valve cusps. It may take years to develop. When the normal orifice area of 5 cm2 is reduced to 1 cm2, severe mitral stenosis occurs resulting in features like exertional dyspnoea, fatigue and features of heart failure. Investigations like 2-D Echocardiography shows valvular stenosis or regurgitation. This is fifth stage of treatment. Treatment with diuretics provides symptomatic relief. Ayurvedic mutral dravyas like Gokshur, Sahachar, punarnawa may be tried. Definitive treatment in the form of surgery like Valvotomy or replacement may be done as per the condition.

Bheda stage characterizes with development of complications of stenotic valves like atrial fibrillation, pulmonary hypertension, chest infections and embolic infarcts. Treatment of the complications should be done. For. E.g.

DC cardioversion for fibrillation. medical cardioversion with tablet digoxin. Oral Warfarin to prevent thromboembolism. **Antibiotics** as prophylaxis

RESULT

Pathogenesis of Valvular heart disease can very well be arranged in Ayurvedokta six stages of Shat Kriyakala and treatment could be framed as per different stages. Confirmation of these stages could be done with the help of modern investigations.

CONCLUSION

It is concluded that if each stage of

Shat Kriyakala is properly

diagnosed with the help of modern
investigations, then VHD can very
well be managed at very earlier
stage and overt mortality can be
avoided with proper stagewise
intervention.

Abbreviations

D.Ni. – Dhanwantari NighantuS.Su. – Sushrut Sutrasthana

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