



## ROLE OF LABORATORY INVESTIGATIONS IN DIAGNOSIS OF HRIDROGA

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### ABSTRACT

In presence of etiological factors the *Dosha* get vitiated and take refuge in *Hridaya*. Then vitiates *Rasa*, *Hridaya* being seat of *Rasa- Rakta* and produce *Hridroga*. Many people still die prematurely from *Hridroga* or are disabled. To expand the wings of research in Ayurveda, to assess drug efficacy, to monitor disease progression and comprehensive prediction of prognosis, search of reliable laboratory investigations and their utilization in the conditions like *Hridroga* is crucial.

**KEYWORDS:** Rasa, Rakta, Hridroga, Laboratory investigations.

### INTRODUCTION

The terms *Hridaya*, *Hridroga*, *Hridayamaya*, *Hritshoola* have been used in ancient texts. In presence of etiological factors the *Dosha* get vitiated and take refuge in *Hridaya*. Then vitiates *Rasa*, *Hridaya* being seat of *Rasa- Rakta* and produce *Hridroga*. Five types of *Hridroga* wise *Vataja*, *Pittaja*, *Kaphaja*, *Sannipatika*, *Krimi* have been described by the Acharya.<sup>[1]</sup> with their general symptomatology. A keen observation uncovers the similarity, the *Vatika* symptomatology has with that of IHD known in modern medicine like angina pectoris, myocardial infarction (painful conditions). Remaining four types of *Hridroga* features are vague and hence inconclusive.

Although our life expectancy has doubled but too many people still die prematurely from CHD or are disabled. Within the coming decades the disability adjusted life years (DALYs) estimate due to cardiovascular disease which includes IHD, HTN, CVD, CHD and RHD, is expected to rise from a loss of 85 million DALYs in 1990 to a loss of 150 million DALYs globally in 2020, thereby remaining the leading somatic cause of loss of productivity. CHD is likely to become the most common cause of death worldwide.<sup>[2]</sup>

For the diagnosis of *Hridroga* it is important to take aid of modern laboratory investigations together with diagnostic procedures and symptomatology of *Hridroga* given in Ayurveda texts. Also to expand the wings of research in Ayurveda, to assess drug efficacy, to monitor disease progression and comprehensive prediction of prognosis, search of reliable laboratory investigations

and their utilization in the conditions like *Hridroga* is crucial.

### MATERIALS AND METHODS

References related to proposed title are collected from classical texts of Ayurveda, modern pathology books, various publications, Internet.

#### Review

##### CBC

Inflammation has been demonstrated to be an important risk factor for the development of cardiovascular events. An elevated WBC count is a risk factor for coronary heart disease and ischemic stroke incidence and cardiovascular disease mortality. It is a risk factor for atherosclerotic vascular disease. WBC-derived macrophages and other phagocytes are believed to contribute to vascular injury and atherosclerotic progression.

Components of the CBC, such as the RBC, platelet counts, hemoglobin and hematocrit values, also are associated with CHD and can be used in combination with the WBC count to predict coronary risk.<sup>[3]</sup>

##### ESR

It is elevated in a wide range of organic diseases. ESR is not a specific and diagnostic test for any disease. However, it is helpful in differentiating functional from organic disease. It is increased in acute myocardial infarction. It is not significantly raised in angina pectoris. It is decreased in congestive cardiac failure. In emergency situations, ESR may be helpful in

distinguishing angina pectoris from myocardial infarction.

### BSL

DM is a potent risk factor for all forms of atherosclerosis and is often associated with diffuse disease. Macro-angiopathy (Macro-vascular disease) like atherosclerosis of aorta and of medium size arteries (like coronary, cerebral, and peripheral) occurs earlier in life. It can cause myocardial infarction.

### LIPIDS

Lipid disorders are common in clinical practice, and some of them are associated with an increased risk of atherosclerotic cardiovascular disease.

**1. Total Cholesterol:** It includes HDL and LDL cholesterol.

#### HDL- Cholesterol

HDL contains 20-30% of total serum cholesterol. Low HDL-cholesterol is a significant risk factor for coronary artery disease even if total cholesterol level is normal. HDL is involved in "reverse cholesterol transport" (i.e. from peripheral tissues to the liver where it is excreted in bile) and thus decreases cholesterol accumulated in blood vessel walls. Therefore, it is called as cardio-protective

cholesterol. Concentration of HDL-cholesterol is inversely related with the risk of atherosclerotic coronary artery disease. HDL-cholesterol < 40 mg/dl is an important risk factor for coronary artery disease (positive risk factor), while level > 60 mg/dl is cardio-protective (negative risk factor).

#### LDL- Cholesterol

LDL contains about 60% of total serum cholesterol. High LDL-cholesterol is a strong risk factor for atherosclerotic heart disease, and is the major atherogenic lipoprotein. It is the primary lipoprotein that mediates atherosclerotic heart disease.

### 2. Serum Triglycerides

Hypertriglyceridemia is a risk factor for coronary heart disease, Patients with serum triglycerides > 200 mg/dl have risk of atherosclerosis.

#### Reference ranges

##### • Lipid profile

- Serum cholesterol: Desirable level: < 200 mg/dl
- Serum triglycerides: Desirable level: < 150 mg/dl
- HDL cholesterol: > 60 mg/dl
- LDL cholesterol: < 130 mg/dl
- LDL/HDL ratio: 0.5-3.0

**Table 1: Categorization of cardiovascular risk in diabetes mellitus according to lipid levels (American Diabetes Association).**

Category	Low density lipoproteins	High density lipoproteins	Triglycerides
1. High-risk	≥ 130	< 35 (men) < 45 (women)	≥ 400
2. Intermediate risk	100-129	35-45	200-399
3. Low-risk	< 100	> 45(men) > 55(women)	< 200

### Biochemical Cardiac Markers

Myocardial cell necrosis leads to membrane damage and leakage of cell contents into the blood stream. This forms the basis for measurement of biochemical markers of myocardial injury in blood.

#### • Creatine kinase (CK)

- Total CK
- Isoenzymes
- CK-MB (activity)
- CK-MB (mass)

- Aspartate aminotransferase (AST) activity
- Lactate dehydrogenase (LDH)

– LDH activity

– Isoenzymes

#### • Cardiac troponins (cTn)

- cTnT
- cTnI

#### • Myoglobin

Currently, myoglobin (an early marker) and cardiac troponin (a definitive marker) are the recommended markers for diagnosis of MI; if cardiac troponin is not available, CK-MB (mass assay) is the next best alternative.

**Table 2: Timeline of cardiac markers after acute myocardial infarction.**

Markers	Peak	Time for detection	Return to normal
1. Myoglobin	1-3 hr	6-9 hr	1 day
2. CK-MB	3-6 hr	12-24 hr	3 days
3. Troponin	4-8 hr	12-24 hr	5-10 days

### Role of biochemical cardiac markers in acute coronary syndrome

- To confirm or exclude the diagnosis of acute myocardial infarction in cases with sudden onset of chest pain. If diagnosis is uncertain from clinical features and ECG in an emergency setting, biochemical markers are helpful in ruling out myocardial infarction. Serial determinations (at admission, 6-9 hours, and 12-24 hours) are recommended to rule in or rule out diagnosis of acute myocardial infarction. Use of CK-MB (mass) and cardiac troponin is recommended for diagnosis.
- Detection of old (by some days) myocardial infarction by cardiac troponin
- Diagnosis of re-infarction (CK-MB mass assay)
- To assess effectiveness of immediate reperfusion therapy (thrombolysis or percutaneous coronary intervention) in STEMI.
- Risk stratification to determine the likelihood of acute coronary syndrome

### Creatine kinase

Highest activity of CK is present in striated muscle, brain, and heart. Increased CK is caused by disorders of heart like myocardial infarction, myocarditis. CK-BB, CK-MB, and CK-MM are the iso enzymes of CK. CK-BB predominates in brain, CK-MB in cardiac muscle, while CK-MM in skeletal muscle and heart. CKMB is the most cardiac-specific CK iso enzyme, and it is recommended to measure CK-MB mass. Total serum CK and CK-MB are always elevated following MI. However, serum CK and CK-MB can arise from tissues other than heart. Following MI, CK-MB rises within 3-6 hours after the onset of symptoms, peaks within 12-24 hours, and returns to normal level by 48-72 hours.

Relative index or RI ( $[\text{CK-MB}/\text{total CK} \times 100]$ ) is used to distinguish cardiac from skeletal muscle damage; RI above 5% is highly suggestive of acute myocardial infarction. It is recommended to obtain sequential samples (one at presentation and subsequently at 8-hour intervals for 24 hours).

### Myoglobin

Myoglobin is the oxygen-binding low molecular weight protein of cardiac and skeletal muscle cells. Myoglobin rises early after MI (1-3 hours) and is currently the earliest marker. Myoglobin of cardiac muscle cannot be distinguished from that of skeletal muscle. Myoglobin levels are raised following MI, open heart surgery, muscle injury, muscle dystrophy, renal failure, shock, and trauma. Thus, although myoglobin rises early following MI, it is not cardiac-specific. However, non-elevation of myoglobin (in two sequential samples 2-4 hours apart) is helpful for exclusion of early MI in patients presenting with chest pain at emergency department.

### Troponins (Tn)

Cardiac troponin T (cTnT) and cardiac troponin I (cTnI) are the most sensitive and specific of the available markers of myocardial necrosis and are considered ideal markers for definitive diagnosis (either cTnT or cTnI). Troponins regulate the interaction of actin and myosin filaments during myocardial contraction. Following MI, troponins appear in blood at about the same time as CK-MB. If troponins are elevated at least 12 or more hours following onset of chest pain, their diagnostic sensitivity is 100%. TnI is more cardio-specific as it is found only in heart muscle. It is not elevated following skeletal muscle injury. Following myocardial damage, TnI rises 4-8 hours the onset of chest pain, peaks within 12-24 hours, and remains elevated for 7-10 days. Development of assays for TnI and TnT represent a major advance in the diagnosis of MI. As troponins remain elevated for 7-10 days, they are useful in cases presenting late. If onset of chest pain is 9-12 hours before admission, only troponin needs to be measured.

### Reference ranges

- CK-MB: < 5% or < 10 µg/L
- Cardiac Troponin T: < 0.1 µg/L
- Myoglobin: < 90 µg/L

### Natriuretic Peptides

Produced in increased amounts by the heart in response to congestive heart failure. These natriuretic peptides assist in the body's response to heart failure by lowering the pressure in the lungs and increasing the flow of urine. Tests for 2 kinds of natriuretic peptides are currently available for the diagnosis of heart failure: BNP (B-type natriuretic peptide) and pro-BNP (N-terminal pro-B-type natriuretic peptide). Blood levels of both of these substances become elevated in patients with congestive heart failure. Physicians most often use these tests to differentiate patients with congestive heart failure from those with lung problems.<sup>[4]</sup>

### C-reactive protein

It is an acute phase reactant plasma protein synthesized by liver that undergo changes in concentration in response to infection, tissue injury and inflammation. In myocardial infarction, CRP rises over 24-48 hours, peaks at 72 hours, and disappears by 7th day after acute myocardial infarction. Failure to return to normal indicates re-infarction.

The level of CRP has been shown to correlate with future risk as follows.<sup>[4]</sup>

- CRP level less than 1: lowest risk
- CRP levels of 1 to 3: intermediate risk
- CRP greater than 3: highest risk

### PLAQ test

It measures the level of lipoprotein phospholipase A2 (Lp-PLA2). Lp-PLA2 generates oxidized molecules within the blood vessel wall that are more prone to lead to both atherosclerosis and irritability of the

atherosclerotic plaque. Elevations in the levels of Lp-PLA2 have been shown to indicate greater risk of plaque formation and rupture independent of the levels of either lipids or CRP. Patients with elevated levels of Lp-PLA2 seem to be at a greater risk of cardiac events.<sup>[4]</sup>

## DISCUSSION

Acharya Sushruta had mentioned that Hritshoola results due to obstruction to flow of blood in heart thereby aggravated Vata dosha gives rise the pain.<sup>[5]</sup> Acharya Bhela had further clarified this type of pathogenesis, the blood vessels attached to the heart when get obstructed due to Kapha, the nutrients are poorly supplied to the Hridaya.<sup>[6]</sup>

Basically most of the laboratory investigations are done on whole blood and for precision on the plasma extracted from blood. Plasma can precisely be referred as Rasa. Rasa Dhatu gets vitiated by aggravated Tridosha, vitiated Rasa and Dosha traversing through heart and arteries get lodged there and alters functioning of the heart (Hridi Badham),<sup>[5]</sup> to cause a heart disease. Because of obstruction in vessels, flow of Rasa-Rakta to Hridaya is compromised and it is underperfused (Alpa Prano Bhavati) leading to ischemia (Shesho Dhato Na Apyayante).<sup>[7]</sup> thereby aggravating Vata, Pitta, Kapha Dosha leading to severe pain and breathlessness.

Different lab tests can demonstrate Hridaya related tissue injury (Vrana), inflammation (Shotha), necrosis (Kotha) by using Rasa-Rakta as sample material. Vitiated Rasa components are used as predictors of high risk of Hridaya pathology and Hridaya derived biomarkers released in Rasa-Rakta may provide reliable information of Hridaya pathology. Vitiated Dosha cause Vrana, Shotha and Kotha of Hridaya. Rasa residing or circulating through it may show components of damage to Hridaya-Mamsa, Dhamani and Sira by lab tests. **Cardiac troponins and Myoglobin** are released in to Rasa after early Paka and Kotha pradhan injury (Vrana) to Hridaya-Mamsa which is Kaphaja intrinsic. **Creatine Kinase, LDH, AST** are released in to Rasa after damage by Pitta due to Shotha predominant pathogenesis. **C-reactive protein, Natriuretic Peptides and lipoprotein phospholipase A2** are released due to Kapha-Pitta predominant Shotha pathogenesis. **CBC and ESR** high levels in Rasa indicates Shotha and Kotha. Raised levels of **BSL and Lipids** are indicators of Rasa vitiation by Kapha and Meda respectively which increases risk of Hridroga. Vata is predominant dosha to cause this pathology which is aggravated by Kapha, Meda obstruction with Pitta Dosha and to release of their above mentioned components in Rasa which are detected by lab tests.

## CONCLUSION

Based on the conceptual study and discussion the following points of conclusion determine the role of laboratory investigations in diagnosis of Hridroga.

### 1. Risk stratification

Elevations in components like lipids, especially cholesterol, form a potent risk for future Hridroga and critical for cardiac risk factor management. Attention to diet (Pathyapathy), exercise (Vyayam), and drug therapy can improve lipid levels and lower risk.

### 2. Diagnosis of disease

Detection of heart disease can save lives, to detect components that are present in Rasa-Rakta that indicate disease, tests detect components that normally are not present when elevated above normal levels, indicate Hridroga. Also lab tests helps to distinguish Dosha and Dushya involved in pathogenesis of Hridroga.

### 4. Progression

Components released in to Rasa may aid in prediction of disease progression. Exact phases of pathogenesis can be identified by detecting Rasa component at various phases like acute and chronic phases of Hridroga.

### 5. Therapeutic

With the help of investigations done appropriately, treatment goals can be achieved in Hridroga.

### 6. Prognosis

Lab tests play a major role in assessment of the future course of the Hridroga, helps in anticipation of probable consequences of Hridroga and disease outcome.

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