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ROLE OF *ATICHINTANA* IN THE ETIOPATHOGENESIS OF *HRIDROGA*: AN AYURVEDIC-BIOMEDICAL CONCEPTUAL REVIEW

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ABSTRACT:
Introduction: *Hridroga* is described in Ayurveda as a disorder of the *Hridaya*, an organ that functions as the seat of *Manas*, *Prana*, *Ojas*, *Sadhaka Pitta*, and *Vyana Vayu*, making it highly susceptible to psychological disturbances.
Need of the Study: Among the Manasika Nidanas, *Atichintana* (persistent excessive thinking) is considered as an important psychosomatic factor that disrupts *Sadhaka Pitta* and *Vyana Vayu*, leading to *Rasa-Rakta Dushti*, *Hridaya Khavaigunya*, and the development of *Hridroga* a mechanism that remains underexplored in correlation with modern stress-cardiac pathophysiology.
Aim: This review explores the classical Ayurvedic mechanism linking *Atichintana* with *Hridroga* and correlates it with contemporary evidence on chronic stress, autonomic dysregulation, cortisol excess, endothelial dysfunction, psychogenic hypertension, and stress-induced cardiomyopathy.
Material and Method: A conceptual review was conducted using classical Ayurvedic texts alongside a targeted search of contemporary biomedical literature, synthesising classical Doshic mechanisms with current psychocardiology evidence.
Result: The available evidence suggests that *Atichintana* is a modifiable risk factor for cardiac dysfunction, highlighting the potential role of *Nidana Parivarjana*, *Sattvavajaya Chikitsa*, *Medhya Rasayana*, and *Achara Rasayana* as supportive strategies for cardiovascular health.
Conclusion: These findings support an integrative approach to psychosomatic cardiac disease, combining Ayurvedic mind-body interventions with conventional cardiac care further evidence-based clinical studies are required to validate these concepts.

KEYWORDS-*Atichintana*, *Hridroga*, *Sadhaka Pitta*, *Manasika Nidana*, Psychosomatic Heart Disease, *Vyana Vayu*.

INTRODUCTION

Cardio-vascular disease remains one of the leading causes of morbidity and mortality worldwide, and a substantial and growing body of biomedical evidence now implicates chronic psychological stress as a significant contributor to endothelial dysfunction, autonomic dysregulation, and cardiovascular risk, acting alongside conventional risk factors such as diet, smoking, and physical inactivity. [1,2]
Ayurveda anticipated this mind-heart relationship in its description of *Hridroga*, in which the *Hridaya* is assigned a dual anatomical and psychological identity. Acharya Charaka describes the *Hridaya* as one of the three *Pradhana Marma-Sthanas* (vital points) of the body, along with *Basti* and *Shira*, and as one of the ten *Pranayatanas* (seats of life)[3], while Acharya Sushruta describes it as the origin of the Dhamanis (vessels) through which *Rasa* and *Rakta* are circulated to nourish the entire body[4]. Acharya Vagbhata further specifies the *Hridaya* as the seat of *Sadhaka Pitta*, governing *Buddhi* (cognition), *Medha* (intellect), and *Abhimana* (self-consciousness). [5]
Because the *Hridaya* thus houses both the physical circulatory apparatus and the psychological faculties governed by *Sadhaka Pitta*, it is uniquely vulnerable to disturbances originating in either domain. Among the *Manasika Nidanas* described in classical texts in relation to *Hridroga* including *Chinta*, *Bhaya*, *Krodha*, and *Shoka Atichintana* (excessive/persistent thinking) is noted for causing *Rasavaha Srotodushti* [6] and *Vata* vitiation through its chronic, cumulative nature. Unlike acute emotional shocks such as sudden fear, which classically produce acute *Vataja Hridroga* presentations, *Atichintana* operates as a sustained, low-intensity but continuous stressor more closely resembling the modern concept of chronic occupational or existential stress. This distinction makes *Atichintana* particularly relevant to contemporary lifestyle-related cardiac disease, in which slow, cumulative psychological load rather than a single traumatic event is the dominant pattern.

Aim
To correlate the influence of *Atichintana* on the Etiopathogenacal mechanisms of *Hridroga* by reviewing classical and contemporary literature.

Objective
(1) To review the classical Ayurvedic description of *Hridaya* as the anatomical and psychological seat of *Manas*, and its relevance to *Manasika Nidana*.
(2) To correlate *Atichintana*-induced *Hridroga* with modern concepts of stress cardiomyopathy and psychogenic cardiovascular disease.
(3) To outline Ayurvedic preventive and therapeutic measures directed at *Atichintana* in the context of cardiac health.

MATERIAL & METHOD
This narrative review was conducted through a comprehensive analysis of classical Ayurvedic literature and contemporary biomedical evidence. Classical information on *Hridaya*, *Hridroga*, *Atichintana*, and related *Manasika Nidanas* was collected from Charaka Samhita, Sushruta Samhita, and Ashtanga Hridaya, along with the commentaries of Chakrapanidatta, Dalhana, and Arunadatta. Relevant modern literature on psychological stress, psychocardiology, stress-induced cardiomyopathy, autonomic dysfunction, and neuroendocrine mechanisms was identified through searches of PubMed, Google Scholar, and Scopus. The retrieved evidence was critically reviewed and integrated to explore the possible relationship between *Atichintana* and the pathogenesis of *Hridroga* from both Ayurvedic and contemporary scientific perspectives.

Conceptual Review
Hridaya as the Seat of *Manas* and *Chetana*- Classical Acharyas describe the *Hridaya* as one of the *Trimarma* (three vital seats, along with Shira and Basti) and as one of the ten *Pranayatanas* (vital seats of life)[7] . It is also described as the seat (Sthana) of Manas and related psychological faculties through its function as the *Adhishthana* of *Sadhaka Pitta* . All twenty-four *Dhamanis* (vessels) are said to originate from the *Hridaya*, carrying *Rasa* and *Rakta* to the rest of the body establishing the *Hridaya* simultaneously as a circulatory hub and an emotional-cognitive centre.
Doshic Physiology of the *Hridaya*- Three specific sub-*Doshas* govern the *Hridaya's* combined psycho-circulatory function. *Sadhaka Pitta*, located in the *Hridaya*, governs *Buddhi*, *Medha*, and *Abhimana*, and is directly responsible for the subjective experience of joy, fear, worry, and grief. *Vyana Vayu*, though pervading the whole body, is described as originating in the *Hridaya* and moving throughout the body with great speed, governing movement and rhythmic bodily activity. *Avalambaka Kapha*, located in the *Uras* and *Trika*, provides structural and nourishing support to the *Hridaya*. Disturbance of *Manas* through *Atichintana* directly aggravates *Sadhaka Pitta* and, through it, disturbs *Vyana Vayu* providing the doshic bridge between a purely mental *Nidana* and a cardiac *Vyadhi*.

Samprapti Of Atichintana Janya Hridroga
The following stepwise pathogenesis can be constructed from classical descriptions, specific to the cardiac manifestation of *Atichintana*:
Nidana Sevana: Prolonged occupational, financial, or interpersonal pressures sustain a pattern of repetitive, unresolved *Atichintana*.

Manasika Dosha Prakopa: *Rajas* is persistently aggravated, keeping Manas in a state of restless hyperengagement; secondary *Tamas* may cloud *Dhi* and *Dhriti*, weakening the capacity to disengage from the worrying thought.

Sadhaka Pitta Prakopa: As the *Pitta* directly responsible for emotional processing within the *Hridaya*, *Sadhaka Pitta* is aggravated first and most directly, producing restlessness or a "heavy heart" sensation.

Vyana Vayu Prakopa: Vitiated *Sadhaka Pitta* disturbs the closely associated *Vyana Vayu*, altering the rhythm and force of circulation correlating with palpitation and blood pressure fluctuation.

Rasa-Rakta Dushti: Chronic Doshic disturbance impairs *Agni*, generates *Ama*, and vitiates *Rasa* and *Rakta Dhatu*, the tissues nourishing and circulating through the *Hridaya*.

Hridaya Khavaigunya and Sira-Dhamani Dushti: Cumulative *Dhatu Dushti* localises at the *Hridaya* (a site of *Khavaigunya*), and in its *Dhamanis*, producing conditions conducive to obstruction or functional impairment.

Vyakti (Clinical Manifestation): Depending on the predominant *Dosha*, the disease manifests as *Vataja*, *Pittaja*, *Kaphaja*, *Sannipataja*, or *Krimija Hridroga*.

S.N.	Stages	Ayurvedic Process	Classical Mechanism	Modern Correlate
1	*Nidana Sevana*	Prolonged *Atichintana* exposure	Repetitive, unresolved excessive thinking (occupational/financial/interpersonal)	Chronic psychological stress
2	*Manasika Dosha Prakopa*	*Rajas-Tamas* vitiation	*Rajas* causes restless hyperengagement of *Manas*; secondary *Tamas* clouds *Dhi* and *Dhriti*	HPA-axis and SAM system activation
3	*Sadhaka Pitta Prakopa*	*Pitta*(cognitive-emotional) aggravation	Disturbed emotional processing within *Hridaya*; restlessness, heavy heart sensation	Elevated cortisol, catecholamines
4	*Vyana Vayu Prakopa*	*Vata* (circulatory) aggravation	Altered rhythm and force of circulation	Sympathetic overactivity, reduced HRV, tachycardia
5	*Rasa-Rakta Dushti*	Dhatu vitiation	Impaired *Agni, Ama* generation, vitiated *Rasa-Rakta*	Endothelial dysfunction, reduced nitric oxide, oxidative stress
6	*Hridaya Khavaigunya* and *Sira-Dhamani Dushti*	Structural vulnerability	*Dhatu Dushti* localises at *Hridaya* and its Dhamanis	Chronic inflammation (CRP, IL-6, TNF-alpha), atherosclerotic plaque formation
7	*Vyakti* (Clinical Manifestation)	Dosha-specific *Hridroga*	*Vataja/Pittaja/ Kaphaja / Sannipataja / Krimija Hridroga*	Arrhythmia,hypertension, CAD,Takotsubo cardiomyopathy, heart failure

Correlation with Classical Types of *Hridroga*

***Vataja Hridroga*:** In the early stage of persistent *Atichintana*, continuous psychological stress predominantly aggravates *Vyana Vayu,* resulting in autonomic imbalance, palpitation, anxiety, precordial discomfort, irregular cardiac rhythm, and fluctuations in blood pressure. This closely resembles stress-induced autonomic dysfunction, cardiac arrhythmias, and myocardial ischemia, making *Vataja Hridroga* the most direct clinical manifestation of *Atichintana*.

Pittaja Hridroga: With prolonged emotional stress, excessive activation of the neuroendocrine system and sustained sympathetic stimulation produce features comparable to *Sadhaka Pitta* aggravation, including burning sensation, irritability, excessive thirst, perspiration, and emotional instability. Persistent inflammation, oxidative stress, and endothelial injury observed in chronic stress states may be conceptually correlated with *Pittaja Hridroga.*

Kaphaja Hridroga: Long-standing *Atichintana* associated with sedentary lifestyle and metabolic disturbances gradually leads to heaviness, lethargy, reduced physical activity, and impaired circulation. Modern studies associate chronic psychological stress with obesity, dyslipidaemia, insulin resistance, and accelerated atherosclerosis, which may conceptually correspond to *Kaphaja Hridroga*, where *Kapha* predominance contributes to structural cardiovascular pathology.

Sannipataja Hridroga: When *Atichintana* remains persistent for prolonged periods, all three *Doshas* become involved, producing a complex psychosomatic disorder with combined functional and structural cardiac abnormalities. This stage may be correlated with advanced cardiovascular disease characterized by hypertension, coronary artery disease, heart failure, recurrent arrhythmias, and stress-related cardiac complications involving multiple pathological mechanisms.

Modern Correlation-

Chronic psychological stress is now recognized as an independent and modifiable risk factor for cardiovascular disease. Persistent excessive thinking (*Atichintana*) closely resembles chronic stress and rumination, which continuously activate the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system. Sustained activation of these neuroendocrine pathways leads to excessive secretion of cortisol, adrenaline, and noradrenaline, resulting in prolonged sympathetic overactivity and reduced parasympathetic tone. This autonomic imbalance causes persistent tachycardia, vasoconstriction, elevated blood pressure, and increased myocardial oxygen demand, thereby imposing chronic hemodynamic stress on the heart.[(8)]

Long-term exposure to stress hormones promotes endothelial dysfunction by reducing nitric oxide bioavailability, increasing oxidative stress, and impairing vascular homeostasis. Simultaneously, chronic inflammation develops through elevated levels of inflammatory mediators such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α), which accelerate atherosclerotic plaque formation and vascular injury. Persistent sympathetic stimulation also enhances platelet aggregation and

coagulation activity, increasing the risk of coronary artery disease, myocardial infarction, and thromboembolic events.[9]

Psychological stress additionally affects cardiac electrophysiology by reducing heart rate variability (HRV), increasing sympathetic dominance, and predisposing individuals to atrial and ventricular arrhythmias. Recurrent autonomic imbalance may also contribute to left ventricular hypertrophy, myocardial fibrosis, and progressive cardiac remodelling, ultimately leading to heart failure. Furthermore, acute surges of catecholamines during severe emotional stress may precipitate Takotsubo (stress-induced) cardiomyopathy, a transient left ventricular systolic dysfunction that clinically mimics acute myocardial infarction despite the absence of significant coronary artery obstruction.[10]

Recent evidence in psychocardiology further demonstrates that chronic stress is associated with metabolic disturbances including insulin resistance, dyslipidaemia, visceral obesity, and persistent hypertension, all of which substantially increase long-term cardiovascular morbidity and mortality. These findings establish chronic psychological stress not merely as a behavioural risk factor but as a direct biological contributor to cardiovascular disease through neuroendocrine, inflammatory, metabolic, autonomic, and vascular mechanisms.[11]

From an Ayurvedic perspective, these modern mechanisms may be conceptually interpreted as the physiological consequences of *Atichintana*, which disturbs the normal psycho-cardiac regulation of the *Hridaya*. The classical description of *Sadhaka Pitta*, *Vyana Vayu*, and subsequent impairment of cardiac function offers a traditional framework that closely parallels the modern understanding of the stress–heart axis, thereby supporting an integrative explanation of psychosomatic cardiovascular disorders.

DISCUSSION

This review is among the first to specifically isolate *Atichintana* as distinct from the broader category of *Manasika Nidanas* such as *Chinta, Bhaya, Krodha,* and *Shoka* as an independent Doshic pathway centred on *Sadhaka Pitta* and *Vyana Vayu*, rather than treating psychological causation of *Hridroga* as a homogeneous category attributable to *Vata* alone.

The classical model positions *Atichintana* as a distinct and clinically significant contributor to *Hridroga*, operating through this definable Doshic pathway rather than through *Vata* alone, as is often assumed for *Manasika*-origin cardiac disease. Notably, this classical distinction closely parallels a well-established distinction in contemporary psychocardiology between acute and chronic stressors, in which chronic stress defined as a demanding, distressing experience persisting nearly every day for six months or more is understood to have a greater role than acute stressors in driving long-term disease processes such as atherosclerosis progression. *Atichintana*, as a sustained and cumulative *Manasika Nidana*, corresponds closely to this chronic stress category, distinguishing it mechanistically from acute-onset *Vataja Hridroga* triggered by sudden fear or shock.

The strength of this conceptual model lies in its multi-dimensional Doshic explanation (*Sadhaka Pitta* + *Vyana Vayu*), which offers greater specificity than existing literature broadly attributing *Manasika Hridroga* to *Vata* alone. This specificity finds a biomedical parallel in the cardiovascular reactivity hypothesis, according to which recurrent or chronic activation of neuroendocrine and autonomic response systems produces cumulative, long-term alterations in vascular, immune, and metabolic processes a "wear and tear" phenomenon on the vasculature that accelerates atherosclerosis and triggers cardiovascular events. This modern mechanistic sequence offers a striking structural parallel to the classical Samprapti of *Sadhaka Pitta* and *Vyana Vayu Prakopa* progressing to *Rasa-Rakta Dushti* and *Hridaya Khavaigunya*, lending the classical model added physiological plausibility.

This has direct clinical implications: patients presenting with palpitation, precordial discomfort, or fluctuating blood pressure in the absence of overt structural heart disease should be screened for a chronic pattern of *Atichintana*, and management should extend beyond *Hridya* Dravyas to include *Sattvavajaya Chikitsa*, *Medhya Rasayana*, *Achara Rasayana*, and Yoga-Pranayama, which address the *Rajas*-driven hyperengagement of Manas at its source. This recommendation is consistent with contemporary evidence that stress-management training including cognitive behavioural therapy, meditation, and yoga can mitigate perceived stress and improve cardiovascular risk factors. Early identification of *Atichintana* as a contributing Nidana may allow preventive intervention before *Dhatu Dushti* and *Khavaigunya* become established, consistent with the Ayurvedic emphasis on *Nidana Parivarjana* as the first line of management.

Integration with Conventional Management: These Ayurvedic interventions are proposed as adjunctive, integrative strategies alongside standard cardiological care, and not as substitutes for evidence-based pharmacological or interventional management of established cardiac disease. This aligns with the observation that despite growing evidence linking psychological stress to cardiovascular disease, psychological assessment remains insufficiently integrated into routine clinical cardiology practice, underscoring an opportunity for Ayurvedic Nidana-based screening to complement, rather than replace, conventional risk assessment.

Limitations: Limitations include the difficulty of isolating *Atichintana* from co-occurring *Manasika Nidanas* such as *Chinta* and *Bhaya* in both classical descriptions and clinical presentation, and the absence of controlled clinical studies specifically correlating *Atichintana* (as measured by validated psychometric tools) with objective cardiac biomarkers or imaging findings in Ayurvedic clinical populations. This mirrors a broader limitation acknowledged even in the biomedical literature, where measurement of chronic and cumulative stress remains

methodologically challenging, and evidence linking stress-reduction interventions to hard cardiovascular endpoints remains limited by small trial sizes and short follow-up.
Future Directions: Future studies could employ validated psychometric tools (e.g., the Perceived Stress Scale) alongside Ayurvedic *Nidana* assessment tools, correlated with biomarkers such as serum cortisol, hs-CRP, endothelial function, and echocardiographic parameters, to empirically test the proposed *Atichintana-Hridroga* pathway. The emerging use of wearable devices and ecological momentary assessment in stress research may similarly offer future tools for the objective, real-time assessment of *Atichintana* in Ayurvedic clinical practice.

CONCLUSION

Atichintana contributes to the etiopathogenesis of *Hridroga* through a specific and traceable pathway involving aggravation of *Sadhaka Pitta* and Vyana *Vayu*, secondary *Rasa-Rakta Dushti*, and eventual manifestation at the Hridaya as *Vataja*, *Pittaja*, *Kaphaja*, or *Sannipataja Hridroga* depending on individual constitution. This model closely parallels, and lends classical depth to, the modern biomedical recognition of chronic psychological stress as a cardiovascular risk factor, and supports the integration of mind-directed Ayurvedic interventions *Sattvavajaya Chikitsa*, *Medhya* and *Achara Rasayana*, and Yoga into the comprehensive management of psychosomatic cardiac disease.

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