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Review Article

Ashtanindita Purusha – The Clinical Revelation: A Review

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ABSTRACT

Charaka Samhita (*Sutrasthana*, 21st chapter) describes eight distinct physical constitutions termed *Ashtanindita Purusha* (eight undesirable constitutions). These includes *Atideergha*, *Atihraswa*, *Atiloma*, *Aloma*, *Atikrishna*, *Atigaura*, *Atisthoulya* and *Atikrisha*. These individuals are considered "*Nindita*", not due to social bias, but because their extreme physical constitutions predispose them to a wide range of diseases, lower their *Vyadhikshamatva*, and make them difficult to treat [1]. Among the eight, *Atisthoulya* and *Atikrisha* emerge as the most clinically significant, being 'always diseased' and requiring constant management [2]. *Atideergha* and *Atihraswa* correlate with growth hormone disorders such as gigantism/acromegaly and dwarfism (e.g., Achondroplasia, Turner syndrome). *Atiloma* and *Aloma* map to hypertrichosis and alopecia universalis, respectively. *Atikrishna* (hyperpigmentation) and *Atigaura* (hypopigmentation) align with adrenal disorders (Addison's, Cushings), albinism and vitiligo. *Ekadasha Nindita* (additional deformities like *Kubja*, *Kana*, *Bhangura*) are also discussed as localized structural defects [3]. This review aims to bridge the gap between these classical concepts, explore its clinical significance and establish a contemporary correlation of each *Nindita Purusha* with genetic, endocrinological, and metabolic disorders.

Introduction

Ayurveda, the science of life, emphasizes the concept of *Swastha* (health), which is defined as a state of equilibrium of the *Doshas, Agni, Dhatus and Malas,* along with a blissful state of the soul, senses, and mind [4]. Within this framework, Acharya Charaka describes specific physical constitutions that deviate significantly frame this balanced state. These eight constitutions, known as *Ashtanindita Purusha*, are highlighted not for their social condemnation but for clinical significance.

The term *Nindita* signifies that these individuals are prone to a multitude of diseases, possess low *Vyadhikshamatva* and are challenging to manage therapeutically. The eight types are [5],

1. *Atideergha*
2.*Atihraswa*
3.*Atiloma*
4.*Aloma*
5.*Atikrishna*
6.*Atigaura*
7.*Atisthoola*
8.*Atikrisha*

This review aims to provide a modern scientific perspective on these classical entities, correlating them with genetic, endocrinological, and systemic disorders so that this framework will provide an integrated and precise approach to diagnosis and management.

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METHODOLOGY

Literature review was conducted to correlate classical *Ayurvedic* concepts with contemporary clinical conditions. Classical references were sourced from original texts and their commentaries. For contemporary correlations, a systematic search was performed in electronic databases and modern textbooks. Relevant data were extracted, analyzed, and presented in descriptive and tabular format.

EKADASHA NINDITA

In *Charaka Samhita, Sutra Sthana*, commentator Acharya Gangadhara also mentions physical defects such as *Kubja* (hump- backed), *Kana* (one- eyed) and *Bhangura* (crooked). However, these were not included in the primary list of the eight *Ninditas* because they are defects of a specific part of the body (*Ekadesha*), rather than the entire body [6].

Another commentator Acharya Chakrapani, points out that the use of the word "*cha*" (and) in the original Sanskrit text is a linguistic tool to include these other deformities (*Kubjadi*) under the umbrella of '*Nindita*', even if they are not the primary focus of the chapter[7]. In contemporary perspective, this distinguishes between congenital/localized structural deformities and systemic endocrine/metabolic disorders. *Kubja* can be correlated with kyphosis, a spinal deformity caused by osteoporosis, disk degeneration, or injury. *Kana* refers to anophthalmia or severe visual impairment in one eye due to trauma or congenital factors. *Bhangura* (crooked/ bent) can be correlated with scoliosis or various orthopedic contractures that cause a limb or the torso to appear bent [8].

ATIDEERGHA AND *ATIHRASWA*

*Paryaya (*synonyms*): Atikaanga, Heenanga, Vikrutangapraja Atideergha* and *Atihraswa* are primarily linked to disorders of the growth hormone (GH) and skeletal development. Acharya Charaka attributes these two defects in *Beeja* (genetics), *Ashaya* (uterine environment), *Kala* and *Karma* (past deeds) and *Matura Ahara Vihara Dosha* . They can be considered as *Viupa praja* or *Shukrapradoshaja Vyadhi*. This aligns with modern concepts of familial short and tall stature, pathological genetic mutations, congenital anomalies, and hormonal imbalances etc. Eg; Klinefelter syndrome, Marfan syndrome (FBN1 mutation), precocious puberty and Turner syndrome [9], [10].

Atideergha

Caused by *Asthivaha Sroto Dushti Karana*, this can be considered as an *Asthi Pradoshaja Vikara* [11]. The disorder presents with overgrowth of the body compared to normal dimensions. *Atideergha* correlates with gigantism (excess GH before epiphyseal plate closure) and acromegaly (excess GH after closure). Causes include primary GH excess, ectopic or iatrogenic GH excess, and growth hormone-releasing hormone (GHRH) excess [12]. The resulting production of insulin-like growth factor 1 (IGF-1) causes characteristic overgrowth of certain tissues, resulting in coarsening of facial features, enlarged hands and feet, as well as effects on multiple systems (cardiovascular, rheumatologic, neurologic, pulmonary and metabolic).It also has genetic components, such as in Marfan syndrome (FBN1 gene mutation), where tall stature is a characteristic feature [13].

Atihraswa

Atihraswa can be correlated with short body stature, commonly called as dwarfism. This can be classified into:

Proportionate: The individual is smaller than average all over. This includes growth hormone deficiency, dwarfism, primordial dwarfism, Seckel syndrome [14].

Disproportionate: Some body parts are average size while others are shorter than normal. This includes skeletal dysplasias like achondroplasia (FGFR3 gene mutation), the most common form causing a normal-sized torso but short limbs. Other examples are, spondyloepiphyseal dysplasia and diastrophic dysplasia [15].

Causes include familial short stature (FSS) – positive family history, absence of underlying pathological etiologies; constitutional delay of growth and puberty (CDGP) – short height in childhood, but attaining target height by adulthood ("late bloomer"); idiopathic short stature (ISS), endocrine disorders; and genetic disorders such as Turner Syndrome [16].

ATILOMA AND *ALOMA*

Atiloma

A Person with this disorder has more body hair than normal – thicker hair, with more than one *Loma* (hair) arising from a single *lomakupa* (hair follicle) [17].*Atiloma* correlates with hypertrichosis (werewolf syndrome), distinguished from hirsutism. Hypertrichosis can be congenital (genetic syndromes) or acquired [18]. Congenital generalized hypertrichosis includes several rare inherited syndromes in which genetic errors result in dysfunction of proteins involved in hair follicle development. Acquired hypertrichosis lanuginosa is a classic paraneoplastic syndrome, reflecting the physical extremes that indicate deeper systemic imbalance and, in certain instances, precede the diagnosis of cancer [19].

Aloma

Due to a reduced number of *Lomakupa,* the *Sweda* (sweat) and the *Mala* (waste) that are excreted through this pathway cannot be excreted properly, leading to various complications [20]. *Loma* are responsible for temperature perception and sudden touch sensation; thus, reduced sensing ability of *Sparshanendriya* (touch sense) occurs, as hair is also a part of *Twacha* (skin). Hairs also prevent the body from foreign microorganisms and pollutants, their absence marks loss of that protection, which can lead to infections. *Aloma* can be correlated with a body is devoid of hair follicles throughout – i.e., alopecia universalis (AU) - complete loss of hair on the scalp and body [21]. The exact cause of AU is unknown, but it is thought to be an autoimmune condition in which a person's immune system mistakenly attacks the hair follicles [22].

ATIKRISHNA AND *ATIGAURA*

Acharya Sushruta describes the formation of *Garbha Varna* (fetal complexion) as a result of various combinations of the

Panchamahabhuta. The combination of *Agni* and *Prithvi* results in *Krishna Varna*, while *Agni* and *Akasha* produce *Gaura Varna*. *Krishna syava Varna* arises from a mix of *Agni*, *Prithvi* and *Akasha*, whereas *Gaura shyava* is formed by *Agni*, *Jala* and *Akasha* [23]. Any deviation in these permutations and combinations of *Mahabhutas* can lead to alterations in the *Prakruta Varna*, potentially signaling congenital or inherited disorders. In the *Charaka Indriyastana*, skin tones are further categorized into *Prakruta* and *Vaikruta varna*. While *Prakruta varna* includes natural shades like *Krishna* and *Gaura*, *Vikruta varna* includes abnormal shades such as *Shyava* and *Shukla*, which are often considered *Arishta lakshanas*, (signs of poor prognosis) [24].

Atikrishna

Atikrishna Varna is formed by a dominance of *Agni* and *Prithwi Mahabhoota* [25]. It correlates with hyperpigmentation, seen in conditions like melasma or post- inflammatory darkening. Diffuse hyperpigmentation is a hallmark of Addison's disease and Cushing's syndrome, linking complexion directly to adrenal function [26]. Addison's disease is characterized by 'bronze' skin pigmentation, weight loss and weakness. Cushing's disease, caused by excess cortisol, leads to obesity, 'moon face', and purple skin striae [27].

Atigaura

This is the extreme absence of pigment, making the person 'too fair' in an unhealthy way .It represents a group of heritable conditions where there is little to no melanin in the skin, hair, and eyes. The actual skin color of different humans is affected by many substances, but the single most important substance determining human skin color is the pigment melanin, produced within skin cells called melanocytes. Persons with light skin are determined mainly by the bluish- white connective tissue under the dermis and by the hemoglobin circulating in the veins of the skin. *Atigaura* correlates with Albinism (genetic mutations affecting tyrosinase) and vitiligo (autoimmune) [28]. Oculocutaneous Albinism (OCA) is a genetic disorder affecting both eyes and skin. There are seven known types (OCA1 to OCA7), all caused by specific genetic mutations. Characteristic features include white hair, very pale skin, and light colored eyes. Vitiligo is a condition where pigment is lost in patches [29].

ATISTHOOLA AND *ATIKRISHA*

These two highlighted as the most clinically significant, being "always diseased "and requiring constant management [30]. Characterized by a state of constant metabolic imbalance, the *Atisthoola* individual suffers from an overgrowth of fat and muscle that leads to poor energy and a shortened lifespan, while the *Atikrisha* individual is severely depleted, appearing as 'skin and bones' with very little resistance to physical stress or disease.

Atisthoola

The *Samprapti* (pathogenesis) of *Atisthoola* involves *Kapha* and *Meda* vitiation due to sedentary lifestyle, heavy diet and genetics [31]. *Medovaha Srotodushti* causes *Srotorodha* (obstruction of channels), obstructing *Vayu*, which leads to *Atikshudha* (excessive hunger) and *Vishamagni* (irregular digestive fire). This mirrors obesity pathophysiology; adipose tissue dysfunction leads to chronic inflammation, insulin resistance, leptin resistance, resulting in increased hunger and metabolic syndrome [32]. The *Ashtadosha* (eight defects) of *Sthoulya* include reduced lifespan, reduced libido, altered body color and others [33].

Atikrisha

Atikrisha is attributed to *Vata* vitiation and severe nutritional deficiency [34]. *Rasavaha srotodushti* leads to depletion of all *Dhatus*. This correlates with malnutrition, hyperthyroidism, malabsorption syndromes (celiac disease, Crohn's disease), chronic infections (Tuberculosis, HIV), Addison's disease and depression [35]. Susceptibility to *Pleeha* (splenic disorders), *Kasa* (cough) , *Kshaya* (wasing) and *Grahani* (malabsorption syndrome) aligns with modern diagnoses [36].

Comprehensive Table Of *Ashtanindita Purusha* With Ayurvedic Pathophysiology And Modern Correlation

Table 1

Sl.No.	*Nindita Purusha*	Clinical features	*Samprapti*	Modern correlation
1.	*Atideergha*	Enlarged hands/feet, coarse facial features, long limbs, joint hypermobility, cardiovascular involvement	*Asthivaha Srotodushti, Asthi Pradosha Roga, Beejadushti, Garbhashaya Dushti, Kaala , Vata Prakopa*	Gigantism, Acromegaly, Marfan syndrome
2.	Atihraswa	Short stature, Disproportionate limbs (Achondroplasia), delayed puberty (CDGP), webbed neck (Turner's syndrome)	*Beejadosha, Shukra Pradoshaja Vikara,*	Dwarfism (Achondroplasia, CDGP, ISS, Turner Syndrome)

3.	*Atiloma*	Excessive andogen- independent hair growth anywhere on body; may be a paraneoplastic sign	*Lomakupa Vikriti, Kapha Vriddhi , Beeja Dosha, Shukra Dushti*	Hypertrichosis (congenital or acquired)
4.	*Aloma*	Complete loss of hair on scalp and body, auto immune etiology	*Vata Prakopa*	Alopecia Universalis
5.	*Atikrishna*	Diffuse bronzing of skin (especially creases), patches on face; associated with adrenal or metabolic disorders.	*Bhrajaka pitta Prakopa*	Hyperpigmentation Addison's disease, Cushing syndrome, Melasma
6.	*Atigaura*	White hair, pale skin, photophobia (albinism), depigmented patches(vitiligo); genetic or auto immune.	*Kapha* and *Raktakshaya*	Albinism, vitiligo
7.	*Atisthoola*	Pendulous abdomen/buttocks/ breasts, reduced libido, excessive hunger, body odor, reduced lifespan, cardiovascular complications, diabetes	*Medovaha Srotodushti, Srotorodha* leading to *Vayu* obstruction in *Koshta* , *Jataragni* vitiation, *Ashtadosha* of *Sthoulya*	Morbid obesity, metabolic syndrome, Frohlich's syndrome
8.	*Atikrisha*	Dry emaciated body with visible veins and tendons, reduced strength, prone to malabsorption, cough, wasting disease	*Vata prakopa, Rasavaha Srotodushti, Dhatukshaya, Nanatmaja Vatavyadhi*	Malnutrition, Hyperthyroidsm, IBD, Addison's disease, TB,HIV, Depression

Discussion

The fundamental causes for *Ashtanindita Purusha* are multifaceted. Acharya Charaka identifies factors that can be categorized as follows,

Beeja Dosha: Defects in the *Shukra* (sperm) and *Shonita* (ovum) of the parents are a primary cause for conditions like *Atideergha, Atihraswa, Aloma, Atiloma, Atikrishna* and *Atigaura*.

Prakriti: The inherent nature of an individual, determined by the predominance of *Doshas* at the time of conception.

Ahara-Vihara: Improper diet and regimen, especially during pregnancy and early development, contribute significantly.

Poorva Karma- Deeds from previous births are also considered a contributing factor in some contexts.

The clinical significance of this classification lies in its predictive and prognostic value. A patient presenting with *Atisthoola* can be assessed for metabolic syndrome, diabetes, and cardiovascular risk, while on with *Atigaura* can be screened for genetic albinism or autoimmune vitiligo. Similarly, *Atideergha* warrants evaluation for Marfan syndrome or acromegaly, and *Atihraswa* requires growth hormone profiling and skeletal surveys [37].

Recent advances in genomics and molecular medicine have begun to validate many of these classical observations. For instance, the FBN1 gene mutation in Marfan syndrome [38] and FGFR3 mutation in achondroplasia [39] provide genetic correlates for *Atideergha* and *Atihraswa*, respectively. Similarly, the autoimmune basis of alopecia universalis [40] and vitiligo [41] aligns with the *Nindita* classifications emphasis on systemic imbalance rather than isolated cosmetic concerns.

Conclusion

The concept of *Ashtanindita Purusha* in *Ayurveda* is a remarkable classification system that identifies individuals with extreme physical constitutions who are at risk for disease. This review demonstrates a strong correlation between these ancient descriptions and modern genetic, endocrine, and metabolic disorders. *Atideergha* and *Athihraswa* map to growth hormone and skeletal disorders [42]; *Atiloma* and *Aloma* map to hair follicle pathologies [43]; *Atikrisha* and *Atigaura* map to pigmentary and adrenal disorders [44]; and *Atisthoola* and *Atikrisha* map to complex metabolic syndromes like obesity and wasting diseases [45]. Charaka's emphasis on the constant management and poor prognosis of *Atisthoola* and *Atikrisha* underscores the chronic and challenging nature of these conditions [46]. Understanding *Ashtanindita Purusha* through a modern lens not only validates *Ayurveda*, but also opens avenues for integrative management strategies. This integrative approach, combining the holistic view of *Ayurveda* with the precision of modern diagnostics, holds the key to better patient care and underscores the timeless relevance of these classical teachings.

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