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REVIEW ARTICLE

Asthikṣhaya due to Bījaduṣṭi: An Ayurvedic Perspective on Osteogenesis Imperfecta

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ABSTRACT
Osteogenesis imperfecta (OI) is a genetically determined connective tissue disorder, primarily caused by mutations in the genes responsible for Type I collagen synthesis. Clinically, it presents with bone fragility, frequent fractures, ligament laxity, dentinogenesis imperfecta, and varying degrees of skeletal deformity. OI is classified into multiple types, with Types I–IV being autosomal dominant and Types VI–XIII typically autosomal recessive. The pathogenesis revolves around defective collagen production, which impairs the structural integrity of bone. Ayurveda describes a comparable condition under the term Asthikṣhaya, characterized by clinical features such as Asthi Shoola (bone pain), Kesha–Loma–Nakha–Danta Prapatanam (falling of hair, body hair, nails, and teeth), and Sandhi Shaithilya (joint laxity). In congenital or inherited cases, this can be classified as Bījaduṣṭijanya Asthikṣhaya, where defects in Śukra and Śonita (male and female reproductive components) lead to impaired formation of Garbha (embryo), resulting in tissue-level defects manifesting from birth. A conceptual analysis was carried out comparing the pathological mechanisms of OI from a biomedical perspective with the Ayurvedic interpretation of tissue degeneration due to Bīja Doṣa. Classical Ayurvedic texts were reviewed to extract references to Asthi Dhātu, Bījaduṣṭi, and congenital anomalies. Modern literature on OI was analyzed to explore the genetic and structural basis of the disease. The hallmark features of OI—such as fragile bones, ligamentous laxity, and dental abnormalities—align with the Lakṣhaṇas of Asthikṣhaya described in Ayurvedic literature. The congenital nature of OI is conceptually explained through Bījaduṣṭi, where defective parental reproductive tissue contributes to improper Dhātu utpatti (tissue genesis) in the progeny, especially Asthidhātu. This defective tissue development leads to inherent structural weakness, akin to Asthi Saithilya and Bala Hāni (loss of strength). The Ayurvedic concept of Bījaduṣṭijanya Asthikṣhaya provides a framework to understand congenital bone disorders such as OI in terms of Doṣa, Dhātu, and Bīja imbalance. While the biomedical model focuses on genetic mutation and collagen biosynthesis, Ayurveda emphasizes the root disturbance at the level of Bīja, affecting the structural integrity of Asthidhātu. Though management was briefly attempted with Ayurvedic and Yogic interventions in a clinical context, the focus remains on understanding the deeper tissue-level pathology through classical principles.
Keywords: *Bijadushti, Asthikshaya, Osteogenesis Imperfecta, Congenital Disorder*

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INTRODUCTION

Ayurvedic Correlation: Introduction to Asthikṣhaya and Bījaduṣṭijanya Asthikṣhaya

Ayurveda, the ancient Indian system of medicine, emphasizes both preventive and curative approaches to health by understanding the human body as a dynamic balance among Doṣha (bio-energies), Dhātu (body tissues), and Mala (waste products). The primary function of Dhātu is to maintain the structural integrity of the body (Sharira) by providing support and strength [1]. According to the principle of Āśraya-Āśrayī Bhāva described by Acharya Vagbhata, the Asthi Dhātu (bone tissue) serves as the abode of Vāta Doṣha,

establishing a reciprocal relationship where an increase in Vāta leads to a corresponding depletion of Asthi, clinically observed as Asthikṣhaya [2,3].
The pathogenesis of Asthikṣhaya in Ayurveda involves two principal mechanisms. Firstly, there is a deficiency in nutrients essential for nourishing the bone tissue, which may arise from malnutrition or excessive catabolic activity attributed to aggravated Vāta. Secondly, obstruction or Srotoavarodha impedes the proper flow of nutrients through the Asthivāha Srotas (channels supplying bone tissue), often due to impaired digestive fire or Agnimandhya at both the Jatharagni (digestive fire) and Dhātvagni (tissue metabolism), leading to accumulation of Āma (toxins). Asthikṣhaya may result from either one or a combination of these factors. While classical Ayurvedic texts describe Asthi Dhātu's anatomy, physiology, and diseases, detailed descriptions of its etiopathogenesis or Nidana-Panchaka remain limited.
A distinctive contribution of Ayurveda to understanding congenital disorders is the concept of Bījaduṣṭi—genetic or hereditary defects in the reproductive elements (Bīja). The integrity of Bijabhāga (fractions of ova) and Bijabhāgavayava (chromosomal and genetic components) determines normal embryogenesis and inheritance patterns. Defects in these elements disrupt proper organogenesis, leading to congenital anomalies [4].Moreover, Ayurveda acknowledges the role of environmental factors such as improper diet (Ahara) and lifestyle (Vihara) influencing fetal development, causing anatomical and physiological defects (Vikriti) in the Garbha (embryo/fetus) [5]. The vitiation of Vāta Doṣha in the mother during pregnancy is implicated in deformities such as Pangutva (lameness), Kubjatva (kyphosis), and Vamanatva (short stature/dwarfism) [6].
To prevent genetic and congenital disorders, Ayurvedic practice advocates precautionary measures including avoiding consanguineous marriages (Tulyagotra Vivāha), preconception purification of reproductive elements (Bījashuddhi) through Panchakarma therapies, and careful maintenance of maternal health by avoiding factors detrimental to fetal well-being (Garbhopaghātakara Bhāva) and fulfilling maternal cravings (Dauhrida). Despite such measures, classical texts like the Charaka Samhita acknowledge the inherent difficulty in curing diseases caused by Bījaduṣṭi, indicating the incurable nature of many congenital conditions [7].

Osteogenesis Imperfecta

Introduction: Osteogenesis imperfecta (OI), commonly referred to as brittle bone disease, is recognized as the most prevalent inherited disorder affecting connective tissue. The pathology of OI primarily involves both quantitative and qualitative defects in Type I collagen, the predominant structural protein in bone tissue, resulting in fragile bones prone to fractures [8].
History: Evidence of OI has been traced back to ancient times; a partially reconstructed skull of an Egyptian infant mummy exhibited morphological features consistent with OI, such as flattening along the vertical axis and broadening across the transverse axis, characteristic of the "tam-o'-shanter" deformity. Additional findings included abnormal dentition, specifically dentinogenesis imperfecta, and overall thinning of the bones [9]. The earliest clinical recognition and study of OI date to 1788 by Olof Jakob Ekman [10]. Subsequently, in 1833, Jean Lobstein classified what is now known as Osteogenesis Imperfecta Type I as "Lobstein's disease" [10]. Later in the mid-19th century, Willem Vrolik described a related phenotype currently referred to as Vrolik's syndrome.

Rationale for Comparing Osteogenesis Imperfecta with Asthikṣhaya

Osteogenesis imperfecta (OI) is a genetically inherited disorder characterized by defective bone formation due to abnormalities in Type I collagen, resulting in fragile bones, deformities, and increased fracture risk. In Ayurveda, the condition of Asthikṣhaya is described as a depletion or degeneration of Asthi Dhātu (bone tissue), manifesting as bone pain, fragility, structural deformities, and related symptoms such as brittle nails and hair loss. Given the similarity in clinical features and pathological changes between OI and Asthikṣhaya, it becomes pertinent to explore a comparative analysis.
While OI primarily arises from Bījaduṣṭi (genetic defects affecting the quality of reproductive seed or hereditary material), leading to structural anomalies in connective tissue, Ayurveda recognizes Bījaduṣṭi as a fundamental cause of congenital disorders, including those involving skeletal deformities. This conceptual overlap forms the basis for correlating OI with the Ayurvedic entity of Bījaduṣṭijanya Asthikṣhaya—Asthikṣhaya caused by hereditary or genetic faults.
Furthermore, understanding OI through the lens of Ayurvedic principles—such as the role of Vāta Doṣha in causing tissue degeneration, Srotodushti (channel obstruction) impairing nutrient supply to bone, and Agnimandhya (digestive/metabolic impairment)—provides an integrative pathological framework that may enrich comprehension of its complex etiology beyond genetics alone.

Thus, comparing OI with Asthikṣhaya not only facilitates a deeper understanding of hereditary bone disorders within Ayurveda but also underscores the relevance of ancient concepts like Bījaduṣṭi in explaining modern genetic diseases. This correlation potentially opens avenues for Ayurvedic approaches in symptom management and supportive care in OI.

LITERATURE REVIEW

Ayurveda's Concepts of Dhātu, Bīja, and Asthikṣhaya

In Ayurveda, the concept of Dhātu forms the cornerstone of understanding bodily tissues and their functions. Dhātus are the fundamental bodily tissues responsible for structure, function, and vitality. Among the seven Dhātus, Asthi Dhātu (bone tissue) plays a vital role in providing physical support, structure, and protection to the body [1].

The term Bīja in Ayurveda refers to the seed or origin of life, signifying the essence from which tissues, organs, and functions arise. The health of Bīja influences the quality and strength of Dhātus, including Asthi Dhātu [2].

Asthikṣhaya is a classical Ayurvedic disease entity characterized by the depletion or diminution of Asthi Dhātu, leading to compromised bone strength, density, and integrity. It correlates with modern pathological conditions such as osteoporosis, where bone mass loss results in fragility and fractures.

CLASSICAL AYURVEDIC DESCRIPTIONS OF ASTHIKṢHAYA AND ITS NIDĀNA

Ayurvedic texts provide a detailed account of the causative factors (Nidāna) responsible for Samanya Dhātukṣhaya (general tissue depletion) and specific Asthivaha Srotodushti (disturbance of bone-carrying channels), which culminate in Asthikṣhaya [2].

Samanya Dhātukṣhaya Nidāna [9]

Ativyayama: Excessive physical exertion or exercise leading to tissue wear.
Anashana: Fasting or insufficient nutrition causing tissue depletion.
Ati Chinta: Excessive worry or mental strain.
Rukshashana: Intake of dry or rough food.
Alpaashana: Reduced food intake.
Vataatapa Sevana: Exposure to dust and intense sunlight.
Bhaya, Shoka: Excessive fear and grief.
Rukshapana: Consumption of dry liquids such as dry alcoholic beverages.
Prajagara: Disturbed sleep or frequent waking at night.
Ativartana: Excessive activities or functions of Kapha, Rakta, Shukra, and Mala (waste products).
Kala: Age-related factors (Adana Kala – improper time, and Vridhavastha – old age).
Bhutopaghata: Invasion by external entities like spirits or toxins.

Vishishta Nidana with Emphasis on Bījāduṣṭijanya (Seed- or Genetic-Origin Causes) [10]

The Vishishta Nidana or specific causative factors of Asthikṣhaya also emphasize Bījāduṣṭijanya origins — disorders arising from defects or vitiation in the Bīja (seed), which includes hereditary or genetic factors affecting Dhātu formation and integrity.

Sahaja Nidana (Congenital/Hereditary Causes):
Beeja, Beejabhaga, Beejabhagavayava — Defects or vitiation in the genetic seed or germ plasm affecting tissue health
Pitrija Bhava — Paternal hereditary traits contributing to disease predisposition
Kulaja — Family/clan-related hereditary tendencies, including specific ethnic predispositions (e.g., Caucasians)
Prakriti — Particular body constitutions, especially Vata-dominant Prakriti, which may predispose to Asthikṣhaya
Jataja Nidana (Congenital but acquired):
Vatakara Ahara (Vata-aggravating diet) and Vihara (lifestyle) that impact genetically predisposed individuals
Swabhavaja Nidana (Age- and sex-related factors):
More prevalent in women and old age due to natural tissue degeneration and genetic factors.

Samprapti (Pathogenesis) with Bījāduṣṭijanya Influence

Acharyas describe the concept of Ashraya-Ashraya Bhava, which elegantly explains the reciprocal relationship between Doshas and Dhatus. Vata Dosha, being the Ashrayi (supporting factor) of Asthi Dhatu, has a proportional relation, wherein vitiation of Vata invariably leads to Asthi Kshaya.

All Vata Nidanas, including Bījāduṣṭijanya (genetic) causes, contribute directly to Asthikṣhaya. The Bīja factor acts as a predisposing cause that weakens the Dhatu formation capacity from the root (seed), thus affecting the quality and quantity of Asthi Dhatu.
In addition to classical Nidanas related to Vata, Medovaha, Asthivaha, Majjavaha, and Purisavaha Srotas, the genetic predisposition (Bījāduṣṭi) plays a crucial role in the disease manifestation and progression. Proper functioning of Jataragni, Bhutagni, and Dhatwagni is essential for correct Dhatu Utpatti (tissue formation). A vitiated Bīja impacts these Agnis negatively, leading to faulty tissue nutrition and Dhatu Kshaya.
Asthi Dhatu is the primary Dushya in Asthikṣhaya, with its Mala, Nakha, Kesha, and related tissues affected. However, in later stages, other Dhatus including Majja and Shukra also suffer depletion, illustrating systemic involvement—often accelerated by Bījāduṣṭijanya (hereditary weakness).

OSTEOGENESIS IMPERFECTA

Overview of Osteogenesis Imperfecta (OI): Types and Genetic Basis

Osteogenesis Imperfecta (OI) encompasses a group of hereditary disorders primarily characterized by increased bone fragility and decreased bone mass due to defects in type I collagen synthesis. The structural integrity of bone is maintained through the composite interaction between collagen fibers and hydroxyapatite crystals. Collagen, particularly type I, imparts tensile strength and elasticity, whereas hydroxyapatite—embedded in the ground substance between collagen fibers—confers compressive strength and rigidity to the bone matrix. In individuals with OI, there is either a quantitative deficiency or qualitative defect (or both) in type I collagen, rendering bones more susceptible to deformation and fractures compared to unaffected individuals.
The foundational classification of OI was introduced by Sillence et al. in 1979, segmenting the disease into four primary types based on clinical features such as fracture frequency, scleral hue, dentinogenesis imperfecta, and inheritance pattern [12]. Later molecular advancements, especially in DNA diagnostics, elucidated the genetic transmission, confirming that most OI cases—particularly the milder and classical types—follow an autosomal dominant (AD) inheritance pattern, primarily due to mutations in the COL1A1 or COL1A2 genes [10].

Classification and Genetic Mutations

In 2009, the International Nomenclature Committee for Constitutional Disorders of the Skeleton (INCDS) expanded and reorganized the classification of OI based on phenotypic presentation. This system categorizes OI into five major phenotypic groups while preserving the historical Roman numeral system, now supplemented with Arabic numbers to indicate broader phenotypic similarity across types.
Currently, OI is classified into Types I through XIII, based on both clinical and molecular characteristics. Types I to V are typically inherited in an autosomal dominant (AD) manner, associated predominantly with mutations in COL1A1 and COL1A2, which encode the alpha chains of type I collagen. In contrast, Types VI to XIII follow an autosomal recessive (AR) inheritance pattern and result from mutations in genes involved in collagen processing, modification, or bone mineralization [11].

Table 1 - OI Types, Genes, and Inheritance

Type	Gene(s)	Inheritance	Collagen Impact	Clinical Notes
I	COL1A1	AD	Reduced normal collagen	Mildest form, fractures in childhood
II	COL1A1/2	AD	Abnormal collagen	Perinatal lethal
III	COL1A1/2	AD	Abnormal collagen	Severe, deforming
IV	COL1A1/2	AD	Abnormal collagen	Moderate severity
V	IFITM5	AD	Mineralization defect	Hyperplastic callus
VI	SERPINF1	AR	Mineralization defect	Severe bone fragility
VII	CRTAP	AR	Collagen modification defect	Severe, recessive
VIII	LEPRE1	AR	Collagen modification defect	Perinatal lethal
IX	PPIB	AR	Collagen folding defect	Severe
X	SERPINH1	AR	Collagen chaperone defect	Moderate severity
XI	FKBP10	AR	Collagen chaperone defect	Moderate severity
XII	SP7	AR	Osteoblast differentiation defect	Moderate severity
XIII+	Various	AR	Various	Rare and under investigation

Pathophysiology of OI

The core pathological mechanism in OI lies in the defective synthesis or structure of type I collagen. Mutations in COL1A1 and COL1A2 genes result in either reduced production of structurally normal collagen or production of abnormal collagen molecules. These alterations disrupt the triple helical structure necessary for proper fibril formation and mineral deposition. As a result, the bone matrix lacks proper integrity, manifesting clinically as bone fragility, recurrent fractures, deformities, ligamentous laxity, and short stature.

Moreover, because type I collagen is also essential in other connective tissues, patients may present with extra-skeletal manifestations, such as blue sclerae, hearing loss, dentinogenesis imperfecta, and cardiovascular abnormalities. The severity of these manifestations varies widely depending on the specific genetic mutation and its impact on collagen structure or quantity.

Genetics and Molecular Pathology in Brief

Dominant types (I-V) mostly result from mutations in collagen genes themselves (COL1A1 and COL1A2) or IFITM5 affecting mineralization.

Recessive types (VI-XIII) involve mutations in genes essential for collagen post-translational modification (hydroxylation, folding), collagen chaperoning, or osteoblast function.

These diverse genetic causes reflect the complexity of collagen biosynthesis and bone formation pathways and explain the wide clinical spectrum of OI.

MATERIAL AND METHODS

This article employs a dual-framework approach to explore the correlation between Osteogenesis Imperfecta (OI) and the Ayurvedic condition termed *Bījaduṣṭijanya Asthikṣhaya*. The method includes a textual analysis of Ayurvedic classical literature and a critical review of biomedical studies on OI, integrating both paradigms to identify conceptual and clinical overlaps.

Method of Textual Analysis of Ayurvedic Classics

A focused textual analysis was conducted on Ayurvedic compendia, specifically Charaka Samhitā, Sushruta Samhitā, and relevant contemporary Ayurvedic case reports. The aim was to elucidate the Ayurvedic understanding of *Asthi Dhātu*, its pathological depletion (*Asthi Kṣhaya*), and congenital abnormalities due to *Bījaduṣṭi* (defective ovum or sperm).

The term *Bījabhāga* and *Bījabhāgavyava*, as referenced by ancient Acharyas, conceptually align with chromosomes and genes respectively, implicating their structural or functional abnormality in hereditary diseases. Such descriptions are particularly found in disorders marked by dwarfism (*Vāmanatva*), kyphosis (*Kubjatva*), and skeletal deformities (*Asthi Vikṛti*), which parallel the phenotypes seen in OI.

These classical insights were supported by an Ayurvedic case report describing congenital asthi vikāra (skeletal deformity) linked to *Bījaduṣṭi*, wherein the child presented with short stature, skeletal abnormalities, and joint looseness—features that overlap with clinical signs of OI. The study further outlines *Samprāpti* (pathogenesis) based on *Vāta prakopa*, *Agnimandya*, and *Srotoduṣṭi*, leading to improper nutrition of *Asthi Dhātu* and its degeneration [12].

Review and Comparison with Biomedical Literature on OI

A parallel literature review was conducted using peer-reviewed biomedical sources to understand the genetic and clinical spectrum of OI. Osteogenesis Imperfecta is primarily caused by mutations in the COL1A1 and COL1A2 genes, affecting Type I collagen. These mutations lead to quantitative (reduced collagen production) or qualitative (abnormal collagen structure) defects, resulting in impaired bone strength, frequent fractures, and characteristic deformities [13].

Further classifications of OI into Types I–XIII are based on phenotypic expression and genetic findings. In addition to dominant mutations in collagen genes, several autosomal recessive forms involve defects in genes such as CRTAP, LEPRE1, and PPIB, which affect collagen post-translational modifications [13]. The pathophysiology described in modern texts matches the *Asthi Kṣhaya* described in Ayurveda, particularly in terms of clinical markers like bone fragility, scoliosis, dentinogenesis imperfecta, and growth retardation.

Citeria for Correlating Ayurvedic and Biomedical Features

To align the two medical paradigms, the following correlation strategy was applied:

Symptom Alignment: Ayurvedic signs such as *Asthishūla*, *Sandhi Shaithilya*, and *Nakha–Kesha Patana* were compared with OI features like bone pain, joint laxity, and hair/dental abnormalities.

Etiological Equivalence: *Bījaduṣṭi* in Ayurveda was considered analogous to gene mutations in OI.

Pathogenic Similarities: Concepts such as *Vāta prakopa*, *Dhatvagni Vaishamya*, and *Srotoduṣṭi* reflect underlying metabolic insufficiencies akin to defective collagen biosynthesis.

Phenotypic Concordance: Clinical cases of *Bījaduṣṭijanya Asthikṣhaya* with deformities and delayed milestones were juxtaposed with documented OI phenotypes in pediatric patients.

DISCUSSION

Osteogenesis Imperfecta (OI) is a heritable connective tissue disorder primarily caused by mutations in the COL1A1, COL1A2, and IFITM5 genes, resulting in quantitative or qualitative abnormalities of Type I collagen. These genetic abnormalities lead to fragile bones, recurrent fractures, skeletal deformities, dentinogenesis imperfecta, and other systemic manifestations [14].

In Ayurvedic terms, such a condition can be conceptualized under Bījadoṣaja Asthikṣhaya, a state of *Dhātu Kṣhaya* (tissue depletion) arising from *Bīja* (sperm or ovum) defects. According to Ayurvedic principles, Bīja, Bījabhāga, and Bījabhāgavyava represent the foundational components of heredity—functionally akin to modern genetic material. Abnormalities in these hereditary units, especially those affecting the *Asthivaha Srotas* (channels responsible for bone nutrition), are said to manifest as structural and developmental defects of the Asthi Dhātu (bone tissue).

The Ayurvedic etiopathogenesis of OI thus involves Bījadoṣṭi (genetic impairment) leading to impaired *Asthi Dhātu Nirmāṇa* (formation) and *Poshaṇa* (nutrition). This results in Kha Vaiguṇya, or structural vulnerability of the bone-forming channels, and makes them prone to Srotorodha (obstruction). These pathological conditions create a disturbed internal environment in which Vāta, the Dosha responsible for all movement and structural development—including Garbha Vibhajana (embryonic cell division)—is either *obstructed* (*Mārga Āvaraṇa*) or aggravated due to Dhātu depletion.

Charaka emphasizes that a Vāta-vitiating diet or lifestyle (*Āhāra-Vihāra*) in a pregnant mother can lead to Bījadoṣṭi and vitiation of *Asthivaha Srotas*, thereby predisposing the fetus to deformities such as Pāṅgutva (lameness), Kubjatva (kyphosis), and Vāmanatva (dwarfism)—all of which are remarkably similar to clinical features of severe forms of OI.

In OI, the chronic undernourishment of Asthi Dhātu and subsequent Vāta aggravation leads to manifestations such as Sandhi Śotha (joint swelling), Śūla (bone pain), and progressive skeletal deformities including scoliosis, kyphosis, and short stature.

Ayurveda recognizes this as a vicious cycle where Vāta—devoid of anchoring in Sneha (lubricating qualities)—spreads erratically through Riktasrotas (empty channels), further intensifying bone degeneration.

Furthermore, the dual mechanism of Vāta Prakopa through Dhātu Kṣhaya (tissue depletion) and Mārga Āvaraṇa (obstructive pathology) aligns with the progressive nature of OI, where skeletal symptoms may not be evident at birth but worsen over time. This explains the gradual onset of symptoms such as contractures, fragility, and bone pain in OI, similar to late-presenting genetic bone disorders.

This provides a unique pathophysiological interpretation of OI. It emphasizes the role of maternal care, correction of Vāta vitiation, and Dhātu-nourishing interventions in managing congenital bone disorders—even if palliative. The chronic and degenerative nature of OI is also consistent with the Chirakārī Roga Prakṛti (long-lasting disease nature) of Asthikṣhaya, making management Yāpya (manageable but not curable) rather than Sādhyā (curable).

CONCLUSION

Osteogenesis imperfecta (OI) and the Ayurvedic concept of Bījaduṣṭijanya Asthikṣhaya share significant parallels in their congenital origin and clinical manifestations of bone fragility and deformities. While modern medicine attributes OI to genetic mutations affecting Type I collagen synthesis, Ayurveda explains similar bone disorders through the framework of Bīja Doṣa leading to defective tissue genesis and Asthi Dhātu depletion. The Ayurvedic understanding emphasizes the role of Vāta Doṣa, impaired Agni, and Srotodushti in perpetuating bone weakness, offering a holistic perspective that complements the biomedical model. Recognizing this correlation enriches integrative approaches to congenital bone diseases, highlighting the potential of Ayurveda in providing supportive management alongside contemporary treatments.

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