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Obesity-Induced Joint Stress and Inflammation in Children: A Narrative Review on the Early Origins of Osteoarthritis

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Abstract

Background: Childhood obesity is emerging as a global health crisis with associated comorbidities. Musculoskeletal complaints in children are one of these comorbidities, with early-onset osteoarthritis (OA) gaining recognition. Early-onset OA in the pediatric population remains underdiagnosed due to the lack of specific diagnostic criteria and overlap with juvenile arthritis symptoms.

Aim: This review aims to explore the most recent evidence regarding early-onset osteoarthritis in obese children, particularly in the knee, highlighting correlations between imaging, biomechanics, and inflammation.

Methods: This narrative review compiles current evidence from imaging, inflammatory biomarkers, clinical, and biomechanical studies to evaluate early degenerative joint changes observed in obese children. Specific keywords were used to collect relevant literature from PubMed and other sources, which were added to Zotero.

Results: Imaging modalities such as Magnetic Resonance Imaging (MRI) and ultrasound showed joint alterations in obese children similar to the early changes seen in adult-onset osteoarthritis. Elevated levels of adipokines, such as leptin, were correlated with these findings. Children who are affected may present with anterior knee pain and limited function, reducing their quality of life.

Conclusion: Mechanical overload and metabolic inflammation in obese children lead to joint degeneration, presenting as a distinct pathology. Diagnosis and intervention are essential to prevent progression to irreversible OA in adults. A refined pediatric diagnostic criterion is needed; this can be accomplished by increasing clinician recognition of the disease in this age group, imaging-guided evaluation, and further longitudinal studies.

Key Words: Childhood obesity, Early-onset osteoarthritis, Joint degeneration, leptin, Metabolic inflammation, Pediatric knee pain.

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1- INTRODUCTION

Childhood obesity, increasingly recognized as a global pandemic, has become one of the major concerns of the 21st century. The World Health Organization estimates that 390 million children and adolescents aged 5-19 were overweight in 2024, with 160 million of them suffering from obesity (1). This reflects the drastic increase in the prevalence of obesity since 1975 among individuals aged 5-19, where obesity affected 5 million girls and 6 million boys then (2). According to the most recent data from 2022, there were over 35 million overweight children under the age of five (1). Despite government efforts and global awareness, an increase in prevalence from 5.4% to 5.7% was also observed over the last two decades (3).

Childhood obesity is a serious medical condition with an array of comorbidities, including metabolic, orthopedic, cardiovascular, pulmonary, hepatic, neurologic, etc (4). Long-standing research has demonstrated the detrimental effects of childhood obesity on the musculoskeletal system, which most frequently manifests as knee pain, lower limb malalignment, elevated risk of fractures, and diffuse musculoskeletal complaints (5). However, the understanding of obesity-related joint problems has evolved to focus on the subclinical degenerative changes in the knee joints of asymptomatic adolescents, as seen on Magnetic Resonance Imaging (MRI), in a cross-sectional study by Widhalm et al. (2011) (6). This finding initiated a new body of research aimed at determining whether these early degenerative changes are the beginning of silent osteoarthritis and whether a causal relationship exists. It expanded the definition of childhood obesity to include the possibility of osteoarthritis developing early, which is a condition usually found in people over 50, rather than merely attributing musculoskeletal pain and abnormal gait to being overweight.

Osteoarthritis (OA) is a chronic, degenerative joint disease with progressive articular cartilage degradation, subchondral bone remodeling, synovial inflammation, and ultimate joint dysfunction (7). Adults with OA can be diagnosed via clinical and radiological findings. In 1986, the American College of Rheumatology (ACR) set criteria for knee OA that consisted of the following: knee pain in conjunction with at least three of six: age >50 years, morning stiffness <30 minutes, crepitus, bony tenderness, bony enlargement, and absence of joint warmth (8). However, given the variety of symptoms and the developing joint structures, this criterion is not validated in pediatrics. As a result, the presence of OA in the pediatric population may go underdiagnosed and mistreated, although there is imaging evidence that suggests early degenerative changes. The absence of pediatric-specific diagnostic standards highlights a deficiency in the evaluation of musculoskeletal disorders in obese children and points to the need to reconsider the diagnosis of osteoarthritis in younger patients.

Obesity and OA are not solely associated with wear and tear and mechanical joint loading (9). However, adipose tissue in obesity acts as a new endocrine organ that secretes adipokines and proinflammatory cytokines, which have been shown to exacerbate joint degeneration, even in non-weight-bearing joints (9). This fact raises a greater concern regarding early-onset osteoarthritis in pediatric populations living with obesity, as depicted in Figure 1.

Given the fact that childhood obesity can be associated with early-onset OA, this review aims to integrate findings from the recent literature on this emerging association. Specifically, it will explore the topic from the following perspectives:

biomechanical and inflammatory mechanisms, imaging findings of early joint degeneration, and clinical features suggesting early, underdiagnosed OA in youth. By highlighting the gaps in diagnosis and new current evidence, this review advocates for the immediate early identification, intervention, and further longitudinal research to determine whether changes occurring in early life continue to develop into clinically relevant osteoarthritis in later life.

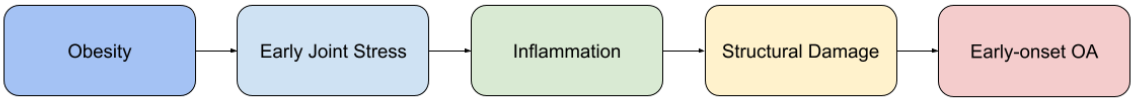

Figure-1: Timeline of pathophysiological progression from childhood obesity to Early-onset osteoarthritis (6,22,27,29,30,36).

2- METHODS

In this review synthesis, evidence regarding the biomechanical and inflammatory mechanisms linking childhood obesity with early osteoarthritis was brought together, highlighting imaging and clinical correlates. A systematic search was conducted on. PubMed/MEDLINE, Scopus, and Google Scholar for articles from database inception to July 2025.

The search strategy combined Medical Subject Headings (MeSH) with free-text terms. The core PubMed string was: ("childhood obesity"[MeSH] OR "pediatric obesity") AND ("osteoarthritis"[MeSH] OR "joint degeneration" OR "cartilage damage") AND ("biomechanics"[MeSH] OR "inflammation"[MeSH] OR "imaging"[MeSH] OR MRI OR ultrasound)

For lifestyle modifiers, additional searches were conducted: ("childhood obesity"[MeSH]) AND ("exercise"[MeSH] OR "physical activity") AND ("bone"[MeSH] OR "joint")

("childhood obesity"[MeSH]) AND ("vitamin D"[MeSH] OR "supplementation")

("caffeine"[MeSH] OR "soft drinks" OR "cola") AND ("adolescents" OR "children")

("breastfeeding"[MeSH]) AND ("childhood obesity"), and ("leptin"[MeSH] OR "adipokines") AND ("osteoarthritis"[MeSH] OR "cartilage")

Peer-reviewed studies on the human pediatric population (adolescents and children) were examined, with biomechanics, imaging, inflammatory pathways, and epidemiological risk factors of interest. Systematic reviews, meta-analyses, and strong mechanistic reviews were also considered. Adult or animal studies were only considered if they contained mechanistic or imaging data of possible application to pediatrics. Non-English language articles, conference abstracts with no accompanying full text, and articles not related to obesity-related joint changes were excluded.

Titles and abstracts were scanned, and then the full text was examined. The final set consisted of 47 papers, which were structured in Zotero and grouped under thematic headings: epidemiology and burden (10 studies), biomechanics (8 studies), imaging (6 studies), inflammatory/adipokine pathways (12 studies), and lifestyle/modifiers like exercise, vitamin D, caffeine, and breastfeeding (11 studies). Additionally,

the reference lists of included papers and reviews were manually searched for other relevant publications.

3-1. Biomechanical Alterations Linking Osteoarthritis and Obesity

Obesity in children alters the mechanical makeup of growing joints, causing unbearable stress on the joints and subjecting the bones and cartilage to unevenly repeated forces (10). These forces increase the wear and tear on the joints, which puts children at risk for OA in the future (11). Even at a young age, this effect is prominent: children and adolescents who are overweight report more musculoskeletal complaints, especially knee pain, than their peers who are of normal weight (5). For example, a large cohort study reviewed data from 227 overweight children vs. 128 non-overweight children and found that 21.4% of overweight youths reported having chronic knee pain compared to 16.7% of non-overweight youths (5). Similar findings were found in a population-based cohort of thirteen-year-olds, who had higher body mass indices, and approximately 8% of them reported having chronic knee pain (12). These results imply that excess adipose tissue puts stress on joints even in children and suggest future degenerative changes (6).

Obesity is associated with significant changes in gait and joint loading. People who are overweight or obese, even at a young age, tend to walk and run with shorter strides, spend more time in the stance phase of gait, and have less flexion of the lower limbs (10,13). Furthermore, during weight-bearing exercises, they tend to adopt a more valgus or abducted knee posture (10,13). Due to this biomechanical variation, the joints are subjected to more forces. For example, obese children walk with nearly twice the patellofemoral (kneecap) contact force as children of normal weight (14). Additionally, they experience higher ground reaction forces and joint moments at the ankle, knee, and hip during dynamic activities (13). As Table 1 highlights, overweight children's unique load profiles and movement patterns place additional mechanical stress on articular cartilage and other joint structures compared to normal-weight children.

Beyond dynamics, skeletal alignment plays a key role. Being overweight during development may eventually lead to malalignment of the lower limbs (15). Additionally, when they reach puberty, obese children often develop genu valgum, or knock-knee alignment, which is a marked increase in knee valgus angulation compared to their healthy-weight peers (16). Such misalignment changes load distribution on the knees, putting strain on the lateral or medial compartments of the articular cartilage (5). Over time, these focal overloads can promote the development of degenerative changes and hasten the deterioration of cartilage in the overloaded areas, as demonstrated in the adult population (17). Therefore, through a combination of alignment-driven load concentration and increased absolute joint forces, obesity has a direct impact on cartilage homeostasis. Repeated small injuries from extra stress can break down the cartilage's collagen and proteoglycan structure. Moreover, when the weight isn't distributed properly due to misalignment, the cartilage becomes more susceptible damage.

While skeletal malalignment clearly contributes to altered load distribution, it is important to note that the included studies only assessed basic anthropometry (age, sex, height, weight, BMI) and did not perform more detailed measurements such as waist-hip ratio, body composition, pubertal stage, or quantitative limb alignment. The absence of these data limits our ability to fully link obesity-related growth changes to the biomechanical pathways described above. Incorporating

such detailed anthropometric assessments in future cohorts would provide a clearer understanding of how fat distribution, maturation, and skeletal geometry interact to accelerate early joint degeneration.

Obesity is not only linked to osteoarthritis through mechanical stress, but it also puts the body in a state of systemic inflammation and triggers local cellular responses that make joints susceptible to inflammatory degeneration (9).

Table-1. Biomechanical alterations in obese pediatric joints vs. normal-weight children (5,6,10,11).

Biomechanical Parameter	Normal-weight children	Obese children
Ground Reaction Force (GRF)	About 1× body weight during walking and running	Increase up to 1.7–2.1× body weight, especially during running
Joint Contact Forces (Knee JCF)	Distributed safely across joint surfaces	Focal overload increases 3–4× forces, especially in the medial knee
Gait Symmetry	Symmetrical stance and swing times	Asymmetrical gait patterns; altered foot placement
Alignment	Neutral axis in the knees	Valgus or varus deformities increase the medial/lateral compartment load
Imaging findings	No abnormalities	Increase in bone marrow edema, cartilage thinning, and meniscal damage

This contrast reveals how childhood obesity alters the mechanics of the joints. Balanced ground reaction forces (≈1× body weight) and symmetrical walking in normal-weight children, compared to greatly elevated loading (up to 2× body weight), increased knee contact forces (3–4× in localized locations), and typical valgus or varus deformities in obese children. These alterations are, in turn, translated directly into abnormally distributed load and correspond to imaging findings of bone marrow edema, cartilage loss, and meniscal injury. Together, the studies indicate that obesity brings with it both quantitative increments in joint force and qualitative alterations in load distribution that enhance the risk for early degeneration compared to normal-weight controls.

3-2. Inflammatory and Metabolic Pathways

3-2-1. Inflammatory Mediators Linking Obesity and Joint Degeneration

Although OA was originally defined as a non-inflammatory arthritis, it is now recognized as being associated with low-grade inflammation, especially in the context of obesity (9,18). Adipokines, such as leptin, and cytokines, including IL-6, TNF-α, and IL-1β, mediate this inflammation (19). One of the most important adipokines in this process is leptin, which is produced by white fat tissue (20). Additionally, it is locally released by chondrocytes, synovium, osteophytes, meniscus, bone, and the infrapatellar fat pad, with higher levels found in synovial and joint tissue (20).

Leptin stimulates chondrocytes to produce inflammatory mediators, including IL-6, IL-1β, IL-8, NO, iNOS, PGE_2, and COX-2, acting as a proinflammatory cytokine (21). Additionally, it upregulates matrix-degrading enzymes (MMP-1, -2, -3, -8, -9, -13, and ADAMTS4/5), which degrade cartilage (see figure 2) (21). In a recent experiment on mice, introducing without leptin fat did not lead to OA in the knee; however, systemic leptin implantation

activated pain and structural OA (22). This demonstrates that leptin, a fat-secreted factor, can actively drive OA (22), supporting the view of OA as not just a localized joint disorder but a systemic disease of adipose tissue (22).

Other adipokines, such as resistin and visfatin, are also produced in greater quantities in obesity (23). These adipokines play a similar role to leptin. For instance, visfatin stimulates synovial fibroblasts to secrete TNFα, IL-1β, IL-6, and chemokines, while resistin induces mononuclear cells to produce the same cytokines (23). However, adiponectin shows dual roles: Some isoforms drive inflammation in cartilage (IL-6, IL-8, NO, MMP-1/3/13, VEGF, MCP-1, VCAM-1), and others are anti-inflammatory via insulin sensitization (23). Although most adipokine research arises from adult studies, the presence of similar biochemical profiles in obese children suggests an urgent need to investigate early metabolic inflammation in juvenile arthritis pathways.

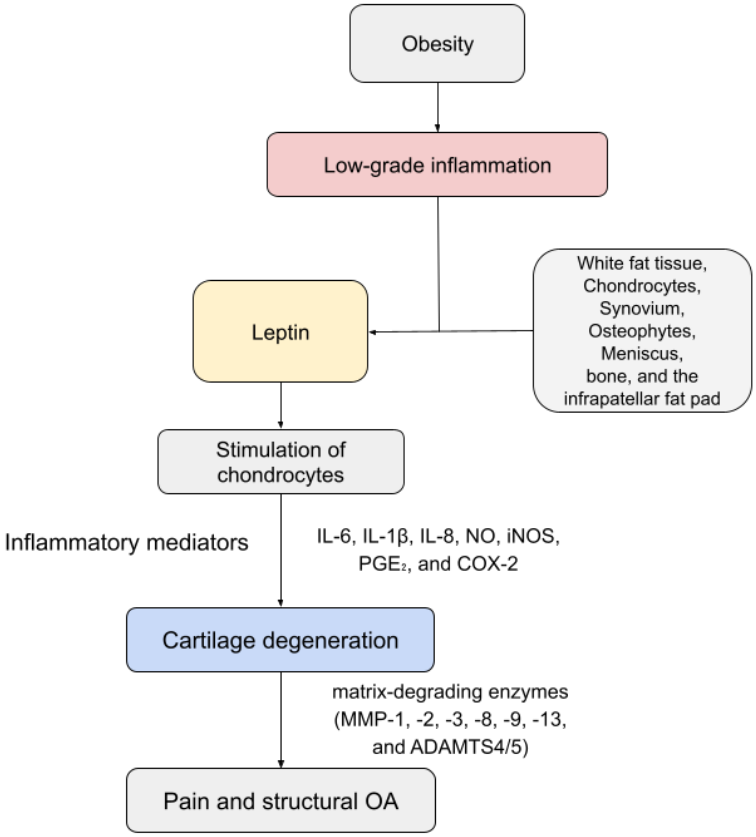

Figure-2: Mechanistic Role of Leptin in Obesity-Induced Joint Degeneration and Early-Onset Osteoarthritis (9,18–22).

Although the inflammation is not a typical synovitis seen in childhood autoimmune arthritis (e.g., idiopathic juvenile arthritis), (24) it plays a pivotal role in cartilage degradation, subchondral bone remodeling, and pain sensitization (25). This provides a clearer view of the fact that obese individuals develop OA even in non-weight-bearing joints (9). Serum leptin levels were considerably higher in adults with primary hand OA than in controls ($p = 0.046$), and leptin was associated with radiographic severity, indicating that adipokine-mediated inflammation plays a part in non-weight-bearing joints (26). Despite strong evidence in adults linking leptin and hand OA, no pediatric studies have yet

evaluated adipokine influence on small-joint degeneration in obese youth, which is an important gap to address.

The typical inflammatory etiology presents with joint swelling, pain, and stiffness persisting for at least six weeks and is used to diagnose juvenile idiopathic arthritis (24). Defined MRI/ultrasound criteria are used to detect evidence of synovitis. Juvenile idiopathic arthritis is the most common chronic pediatric arthritis, affecting approximately 1 in 1,000 children in the U.S., making it heavily studied in pediatric rheumatology (24). By contrast, OA in obese children lacks established diagnostic criteria and is less studied, not taking in to account the ongoing inflammation that occurs locally and systemically. This comparison between early-onset OA associated with obesity and JIA is further explained in Table 2.

Table-2: Comparative features of juvenile idiopathic arthritis (JIA) and obesity-associated osteoarthritis (OA) in pediatric populations (6,19,23,27,32,34,35).

Feature	Juvenile Idiopathic Arthritis (JIA)	Obesity-Associated Osteoarthritis (OA)
Onset Age	Typically, before age 16	Can begin in late childhood/adolescence
Etiology	Autoimmune, idiopathic	Mechanical + Metabolic/Inflammatory (Adipokines)
Joint Involvement	Small and large joints; symmetric or asymmetric	Primarily weight-bearing joints (e.g., knees)
Synovitis	Yes, classic feature	Low-grade, non-classic synovitis
Pain Pattern	Morning stiffness >30 mins; improves with activity	Activity-induced pain worsens with movement
Imaging	MRI/US: Synovial hypertrophy, effusion	MRI: Cartilage loss, meniscal degeneration, edema
Inflammatory Markers	Elevated ESR, CRP, ANA, RF	Mild/moderate CRP, IL-6, TNF-α; leptin is often elevated

This context of comparison establishes the distinctive nature of OA due to obesity in contrast to JIA. Unlike JIA, which is autoimmune in etiology, OA due to obesity arises from mechanical overload superimposed on metabolic and adipokine-mediated inflammation. The clinical presentation differs too: JIA has morning stiffness and systemic inflammatory markers, while OA due to obesity has activity-induced pain, low-grade inflammation, and imaging features suggestive of early cartilage and meniscal damage. Both diseases have in common synovitis, but in obesity, it is less severe and often adipokine-mediated by factors such as leptin and not by traditional autoimmune mechanisms. This underlines the need to recognize obesity-associated OA as a distinct disease within pediatrics that is distinguishable from autoimmune arthritis in terms of etiology, pain profile, and biomarker signature.

3-2-2. Metabolic Inflammation in Pediatric Joints: Imaging and Biomarker Evidence

The systemic and local joint inflammation is not limited to just the obese adult population, but has also been proven in the obese pediatric population (27,28). Researchers conducted a systematic review on ultrasound-detected changes in the infrapatellar (“Hoffa’s”) fat pad of obese children, which found a 30% increased risk of knee inflammation in obese children compared to their non-obese peers (27). The high-resolution ultrasound using B-mode, Doppler, and elastography identified fat pad changes

(27),including hyperechogenicity, Doppler RI < 0.6, and thickness > 10 mm before clinical manifestations (27).

Researchers observed strong correlations between ultrasound findings and IL-6 and TNF-α levels, confirming that Hoffa's fat pad acts as a site of local metabolic inflammation, a component in pediatric joint degeneration (27). This thickened and inflamed Hoffa's fat pad in obese youth points to mechanical overload on anterior knee structures, creating a cyclical stress-inflammation model (27).

A cross-sectional study that explored gait biomechanics and inflammatory biomarkers supported the stress-inflammation paradigm (29), revealing a negative correlation between CRP and sagittal asymmetry and IL-6 and frontal asymmetry (29). This suggests a link between systemic inflammation and uneven biomechanical joint loading (29), putting an obese child in a cycle of mechanical overload and inflammation affecting the developing joints even before the onset of clinical symptoms. Figure 3 illustrates this concept, suggesting that excessive joint loading due to excess body weight initiates localized joint stress, triggering inflammatory signaling, further degrading joint integrity, even in the absence of overt synovitis.

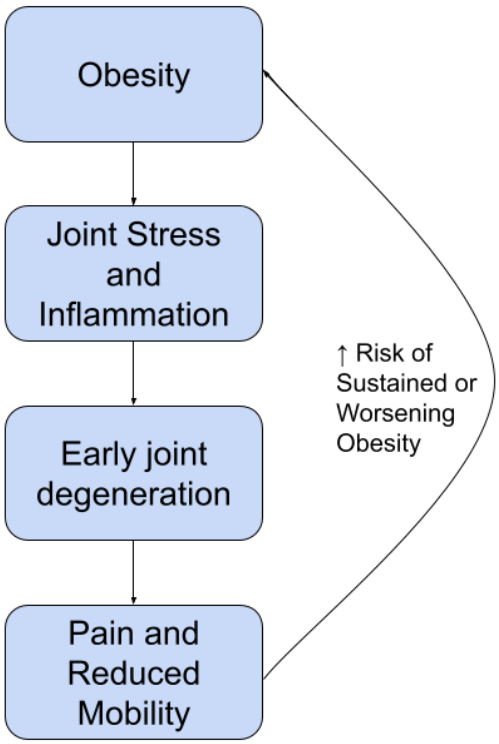

Figure-3: Proposed Cycle of Joint Degeneration in Obese Children (5,6,18,28).

Whether the changes in the joints are reversible remains inconclusive. A study found that after weight loss and anti-inflammatory treatments, ultrasound indicators of fat pad inflammation dropped, suggesting that early changes in childhood obesity may be reversible (27). In contrast, another study found a strong association between synovial fluid leptin levels and cartilage damage, pain, and structural severity of osteoarthritis, even after controlling for BMI (30). Thus, further longitudinal studies are needed to determine whether early treatment of fat pad inflammation can delay or prevent established osteoarthritis. Additionally, more studies are needed to quantify the

risk of developing OA for children who were obese after controlling for their BMI.

3-2-3. Imaging as a Tool for Early Inflammatory Joint Change Detection

Crucially, the inflammatory changes are now detectable through modern imaging techniques, which provide a bridge between molecular pathology and clinical assessment. As previously mentioned, ultrasound imaging can show inflammation in different joint structures. These results highlight the value of imaging in detecting silent joint changes caused by inflammation and other stresses.

3-3. Imaging Evidence of Early Joint Degeneration in Obese Children

3-3-1. Conventional Radiography

In pediatric patients, standard weight-bearing radiographs can provide an initial overview of the knee's osseous alignment and any gross degenerative changes. However, classical osteoarthritic features (e.g., joint-space narrowing, subchondral sclerosis, osteophytes) are usually absent due to the early stage of cartilage degeneration and the developing skeleton. Nevertheless, X-rays are valuable for detecting malalignment of the lower extremities, which is frequently observed in obese children (31).

Notably, excessive weight in children alters the mechanical loading on the developing growth plate, often leading to genu valgum (knock-knee deformity) (31). This valgus alignment is found at high rates in morbidly obese juveniles and can contribute to abnormal load distribution across the knee (31). This, in turn, can explain anterior knee pain since patellofemoral stress and asymmetric cartilage tears are exacerbated. Thus, early identification of malalignment abnormalities on imaging is vital.

In addition, interventions such as guided growth (temporary epiphysiodesis) or orthotic bracing during childhood can correct deformity and prevent progression to irreversible osteoarthritis in adulthood (31). In summary, while radiography may appear "normal" in early obesity-related joint disease, it plays a role in ruling out other bony pathologies and quantifying any skeletal changes (such as varus/valgus angulation) that underlie clinical symptoms.

3-3-2. Magnetic Resonance Imaging (MRI)

MRI is the cornerstone imaging modality for evaluating obesity-related knee damage in children, owing to its superior soft-tissue contrast and ability to visualize cartilage, menisci, and bone marrow directly. MRI has proven highly sensitive in detecting early degenerative changes that are invisible on X-ray (6). Unlike ultrasound, which was detailed earlier as a first-line tool for assessing superficial structures, MRI can comprehensively assess deep intra-articular tissues without being limited by acoustic windows (6).

Importantly, studies using MRI have revealed that morbidly obese children often already exhibit the hallmarks of osteoarthritis in their knees. For instance, Widhalm et al. examined 24 knees of 20 obese adolescents, and cartilage lesions were observed in every knee, with multiple regions affected in most cases (6). The patellofemoral compartment was involved in the vast majority, with retropatellar cartilage damage noted in 19 of 24 knees, and weight-bearing compartments showing a range of chondral lesions, some knees demonstrating grade II or even grade III cartilage lesions in the medial compartment (6). Such findings are striking, as grade II and III lesions indicate substantial cartilage fibrillation or defects that one would only expect in much older patients under normal circumstances. In addition to cartilage loss, meniscal changes were present in most of these obese

patients' knees, including signs of early meniscal degeneration or small tears (6).

Moreover, MRI frequently detects subchondral bone alterations as well as bone marrow edema or stress-related changes, reflecting the osteoarticular impact of excessive weight (32). Indeed, one review noted that in a series of obese children (ages 9–19) with knee pain, all had cartilage and bone alterations evident on MRI (32). This comprehensive view provided by MRI highlights that pediatric obesity can initiate a constellation of structural joint changes analogous to adult osteoarthritis (32). By confirming the presence of cartilage defects, meniscal injury, or bone stress, MRI not only documents the extent of an obese child's joint damage but also guides the aggressiveness of management needed.

3-3-3. Imaging–Clinical Correlation

The advanced imaging findings closely correlate with clinical symptoms, validating that knee pain in obese children is not simply "growing pains" but is rooted in structural joint damage. For instance, joint effusions seen on US or MRI indicate synovitis, which often corresponds to episodes of swelling, stiffness, and pain after activity (11). Widhalm et al. observed that constant knee pain was correlated with more cartilage lesions in obese adolescents (on average, 3.7 lesions per knee) compared to obese peers without constant pain (≈2.8 lesions) (11). In contrast, normal-weight controls had almost no cartilage defects (mean <1) and reported minimal knee issues (11). Furthermore, clinical knee function scores (IKDC, KOOS) were worse in the obese-pain group and progressively better in the pain-free obese and lean groups, paralleling the MRI findings (11).

Imaging also reveals factors that can exacerbate symptoms: for example, an inflamed Hoffa's fat pad (seen as hyperechoic and swollen on US) can cause anterior knee pain and impingement (27), while meniscal tears seen on MRI can lead to clicking or locking sensations (33). Importantly, these objective imaging signs lend credibility to the child's complaints. Indeed, one study concluded that morbidly obese youths with knee pain show significant lesions of the cartilage, abnormal meniscal features, and effusions, which likely explain their substantial pain and reduced quality of life (34). Recognizing this imaging-symptom correlation is critical for clinicians as it highlights that obesity-induced joint changes are already underway in childhood, driving real clinical symptoms.

3-4. Clinical Manifestation

Chronic knee pain and functional limitation are common complications in obese pediatric populations with degenerative knee changes. A study of 13-year-olds showed that 8% of them had knee pain that lasted more than three months (12). Daily pain was present in 37.5% of those who had knee pain, and almost all the pain was localized to the anterior knee (12). This knee pain was strongly associated with excess body weight; pain-free adolescents had lower BMI (body mass index) compared to those with pain (12).

Clinically, the CDC (Centers for Disease Control and Prevention) reported more than 220,000 children with arthritis (35). Although this estimation included parent reports and did not differentiate between OA and JIA, it was strongly associated with obesity and overweight (35). The child experiences knee pain that is exacerbated by activity (running or climbing stairs), with notable stiffness after effort, and it accounts for approximately 15% of general pediatrician visits (32). The chronic knee pain reduces the quality of life of the child and occurs with physical inactivity, depression, and anxiety (35). This shows how this

discomfort affects more than just health and well-being.

Even with weight loss, these changes affect those children's future knees. A 25-year longitudinal cohort study found that people who were overweight as children were much more likely to have knee pain, stiffness, and loss of function in adulthood, even after taking into account their adult weight (36). This suggests that being overweight as a child can permanently set the stage for osteoarthritis in the knees. In short, the clinical signs of knee injury in children who are overweight are the same as those of early osteoarthritis. Recognizing this early arthritis in childhood is important so that steps can be taken to slow down the damage to the joints and stop it from getting worse and turning into severe osteoarthritis in adulthood.

3-5. Modifiers of Risk and Methodological Considerations,

Across the pediatric joint–focused articles included in this review, MRI cohorts of obese youth with early cartilage/meniscal change (6,11). ultrasound evidence of Hoffa's fat pad inflammation (27), and gait–inflammation coupling (29), none prospectively evaluated supplement regimens (e.g., vitamin D, calcium) or prescribed exercise programs (type, intensity, session length, weekly frequency) as exposures/interventions; "activity" appeared only within gait/biomechanics testing. Daily caffeine/cola intake was not captured. Demographics (age, sex, BMI/obesity) were consistently reported, but pubertal stage, infant feeding history, genetic markers, and supplement use were not. Major inflammatory/orthopedic diseases were typically excluded, and comorbidities were variably listed. This consistent absence of reporting across multiple studies highlights a systematic evidence gap that should be addressed in future work.

3-5-1. Supplement Use and Exercise (type and duration)

The lack of information on supplement use and structured exercise regimens in the included studies makes it impossible to conclude their role in preventing or modifying early joint degeneration in obese children. However supporting evidence from pediatric bone health studies shows a connection. Recent trials reveal that supervised, mixed-mode, weight-bearing exercise interventions lasting 12-20 weeks, given roughly three times a week for 60-90 minutes per session, are linked with increased bone accrual in obese children (37–39). Calcium and vitamin D supplementation during growth has also been connected with improved bone outcomes, though these trials have not assessed joint- or cartilage-specific outcomes. Together, this evidence supports the biological potential that exercise and adequate micronutrient intake may reduce musculoskeletal susceptibility, but reinforces the imperative for joint-focused pediatric research.

3-5-2. Daily Caffeine and Cola Drinks

None of the pediatric OA studies included in this review examined caffeine or cola consumption, so no conclusions can be drawn from joint-specific data. The broader pediatric evidence is mixed. A large NHANES-based analysis found no causal relationship between caffeine intake and bone mineral density (BMD) in children and adolescents (40).Similarly, a Danish longitudinal cohort study found no measurable effect of soft drink intake on bone health or fracture risk (41). However, observational studies in Asian adolescents have linked high soft drink or cola consumption with lower BMD (42,43). While these findings are bone-focused rather than joint-specific, they suggest that excessive cola consumption may negatively affect skeletal quality and should be investigated in relation to early joint degeneration in obese youth.

3-5-3. Demographics, Infant Nutrition, Genetics, and Comorbidities

Demographic factors, infant nutrition, genetic predisposition, comorbidities, and supplement use were incompletely captured in the included studies. Only age, sex, and BMI were consistently reported, while pubertal stage, breastfeeding history, genetics, and supplement use were absent. Broader pediatric evidence emphasizes the importance of these variables. For example, NHANES 2009–2018 analyses show that longer breastfeeding duration is associated with lower BMI in early childhood (44), which indirectly reduces mechanical stress on developing joints. The absence of these data from pediatric OA studies represents a major limitation for risk stratification and highlights the need for standardized reporting of these modifiers in future research.

3-5-4. Vitamin D and Leptin

Vitamin D deficiency and elevated leptin levels are two biologically plausible pathways through which obesity may accelerate joint damage, yet neither was measured in the pediatric OA cohorts we reviewed. Recent meta-analyses of vitamin D supplementation have shown little to no clinically meaningful effect on BMD in children and adolescents, even in subgroups who are deficient (45). Conversely, pediatric exercise trials consistently demonstrate reductions in circulating leptin following formal intervention in obese children (46), and translational studies in adults suggest adipose-derived leptin is a mediator of cartilage breakdown and osteoarthritic pain (22). Although not measured in the series of studies reviewed, such biomarkers require attention in future work for pediatric OA, ideally through prospective imaging cohorts that include biochemical profiling.

3-5-5. Risk of Bias

Concerning bias, the included studies attempted to minimize selection bias by excluding children with inflammatory rheumatologic or congenital orthopedic disease and by clearly defining obesity through age- and BMI-based cutoffs (6,11,27,29). Confounding was partially addressed by reporting and comparing age, sex, and BMI, though important modifiers such as pubertal stage, physical activity levels, metabolic comorbidities, and supplement use were inconsistently captured, leaving residual confounding likely. Measurement bias was reduced by the use of standardized MRI and ultrasound descriptors, but inter-reader reliability and blinding to obesity status were rarely reported. Reporting bias was evident in the systematic omission of supplement use, caffeine/cola, vitamin D, and leptin across studies. Finally, most cohorts were cross-sectional, avoiding attrition bias but limiting causal inference. Future pediatric research should adopt prospective designs with repeated imaging, pre-specified confounders, and biochemical profiling to reduce bias and clarify temporal relationships in obesity-associated early joint degeneration.

4- DISCUSSIONS

This review brings attention to the nascent correlation between childhood obesity and early-onset joint degeneration, mainly of the knee joint, which bears most of the mechanical burden during growth. Even though OA was considered a disease of aging with a degenerative and non-inflammatory mechanism, new findings reinforce that childhood obesity starts a chain reaction of mechanical and inflammatory alterations that could lead to OA-like disease early in life (6,9,18).

The hallmarks of OA that are typically expected in adults, such as cartilage lesions, meniscal degeneration, and subchondral bone changes, are now seen in children through MRI (6,32). According to Widhalm et al.'s study, all of the obese

adolescents' knees that were examined had damaged cartilage (6). Some knees had grade II–III lesions, and many compartments were affected (6). These changes presented clinically with functional impairment, pain, and reduced quality of life (11,34). These results show that the joint degeneration process starts and shows symptoms in childhood.

In addition to mechanical causes, metabolic inflammation, caused by adipokines such as leptin, resistin, and visfatin, is a major reason for early joint deterioration. Particularly, leptin promotes cytokine release and matrix degradation through MMPs and ADAMTS enzymes (21,23). It is produced systemically and locally in the joints by chondrocytes and synovium (20). This causes a systemic inflammatory environment that affects both weight-bearing and non-weight-bearing joints (30).

The inflammation in the joints, specifically the infrapatellar fat pad (Hoffa's fat pad), is further depicted by ultrasound and elastography studies. Ultrasound showed increased echogenicity, Doppler flow, and thickness over 10 mm in the obese children group compared to the control group (26,27). These changes are correlated with inflammation through elevated IL-6 and TNF-α levels in the studied joints (27,29). Therefore, the stress-inflammation cycle is a critical mechanism triggering early joint damage in children living with obesity.

Clinically, physicians mislabel chronic anterior knee pain as growing pain and dismiss this pain in obese children. Although many studies have proved that this pain is correlated with structural joint damage, not normal joint development. [12,34] This early-life joint stress has a lasting impact. In other words, adults with previous childhood obesity are more likely to develop OA, even after controlling their adult weight (36).

Whether these changes are reversible or set a trajectory into adult OA is still a negotiable question. Some studies suggest that controlling weight with anti-inflammatory medications can decrease inflammation and inflammatory biomarkers. This indicates that early interventions can reverse some joint changes (27). However, other studies suggest that the leptin-driven degeneration and pain are not eliminated with normalization of the BMI (30). This implies that it is a process that cannot be reversed after crossing a threshold.

Although the reversibility of these changes is not yet identified, urgent interventions are needed. Such interventions must target both mechanical and inflammatory drivers of joint degeneration. Pediatric weight management regimens should include techniques to preserve the joints, such as physical therapy, gait correction, and even anti-inflammatory drugs. Moreover, screening the high-risk groups through imaging techniques could identify early changes and enable early monitoring.

Despite the rising evidence, there is no diagnostic criterion for early-onset OA in the pediatric population. The diagnostic criteria for adults do not take into consideration anatomical, biomechanical, and developmental differences in pediatric joints (8). While Juvenile Idiopathic Arthritis (JIA) has established diagnostic criteria, OA in children remains underdiagnosed and undertreated. It is important to differentiate these disorders, as they demand different management approaches. This is a major limitation that needs to be addressed since diagnosing the disease means treating it and taking early steps before joint function worsens.

In summary, this review presents various evidence from imaging and biomarkers to clinical studies that support the existence of early-onset OA in obese children. Neglecting the joint health of such children will worsen life quality. Thus, these

changes need to be recognized and treated to slow the disease progression and preserve joint function. Future research should focus on developing diagnostic criteria for children, studying the reversibility of the changes with disease progression, and identifying biomarkers.

5- CONCLUSTION

Childhood obesity is not just a superficial risk factor for knee pain; it leads to pathological changes that result in subclinical joint degeneration and early-onset OA. Current evidence suggests that adipose tissue creates a stress-inflammation cycle due to biomechanical stress on the joints leading to local and systemic inflammation established before adulthood. These changes are not only predictive of future disease, but they are also pathologic at the joint level, resembling adult OA. However, children with these joint alterations are not evaluated or suitably treated due to the lack of diagnostic criteria.

This review highlights the need to redefine OA as a disease that can begin in individuals under 50, even in children. Sensitive imaging modalities along MRI and ultrasound, combined with biomarker profiles and symptoms, can be used for early screening. Early screening leads to diagnosis and intervention that can limit progression. Moreover, preventing the cause is more vital than treating the disease; therefore, lifestyle modification, weight reduction, and educating parents can eliminate the lifelong burden of OA. Recognizing and treating early joint deterioration in obese children may be the best chance we have to break the cycle of pain, disability, and lower quality of life that these children experience as adults.

Ultimately, further research is needed to investigate the impact of early-onset osteoarthritis on the quality of life of children. In addition, the suitable authorities should be contacted to set up weight-loss programs or other interventions that address the issue at the community level. This rising disease not only affects children, but it also impacts the future adult generation, compromising their movement early in life and adding more burden to society.

6- DECLARATION

AI Use: During the preparation of this work, the author utilized ChatGPT to improve language quality and readability. After using this tool, the author carefully reviewed and edited the content as necessary and takes full responsibility for the publication.

7- CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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