

ORIGINS AND PREVENTIVE MEASURES OF HIP OSTEOARTHRITIS (COXARTHROSIS): A COMPREHENSIVE REVIEW

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Abstract: Hip osteoarthritis, commonly referred to as coxarthrosis, is an insidious degenerative disease of the hip joint characterised by progressive cartilage degradation, sub-chondral bone changes, and joint dysfunction. Although it often presents in older adults, its origins span congenital, developmental, mechanical, metabolic and inflammatory pathways. This article examines the multifactorial etiology of coxarthrosis and explores evidence-based strategies for prevention. We begin by reviewing the principal causative factors—including joint dysplasia, traumatic injuries, femoral head necrosis, and chronic mechanical overload—and discuss how such factors compromise hip biomechanics, initiate cartilage damage and lead to subsequent joint degeneration. Risk factors such as ageing, obesity, joint malalignment, and muscle weakness are also highlighted. Following a literature review of more than two decades of hip osteoarthritis research, we focus on prevention: weight management, physical activity and muscle strengthening, early detection and correction of hip morphology abnormalities, injury prevention, and lifestyle modification aimed at preserving joint homeostasis. In addition, we outline a hypothetical research methodology to evaluate the effectiveness of a combined exercise and education programme in a high-risk population. Following the findings of recent cohort and retrospective studies, the article presents results indicating that early intervention and targeted preventive measures can significantly delay the onset or slow the progression of coxarthrosis. Finally, the conclusion synthesises these findings into a call for integrated, multidisciplinary prevention programmes emphasising risk factor modification and early hip health monitoring. Recognising the prevention of hip osteoarthritis as an achievable goal—not merely the management of advanced disease—could reduce disability, improve quality of life and reduce the burden on orthopaedic services.

Keywords: Hip osteoarthritis, coxarthrosis, etiology, prevention, hip dysplasia, mechanical overload, weight management, muscle strengthening, trauma, joint biomechanics.

Introduction

Hip osteoarthritis (coxarthrosis) represents a significant clinical and socio-economic burden worldwide. It impairs mobility, reduces quality of life, and frequently culminates in surgical intervention such as total hip arthroplasty. While much attention is given to treatment of established disease, far less emphasis has historically been placed upon its origins and prevention. Understanding the causative pathways of coxarthrosis—and intervening before irreversible joint damage occurs—offers an important opportunity to maintain hip health and delay or avoid end-stage disease.

Anatomically, the hip joint is a ball-and-socket articulation between the femoral head and the acetabulum. Healthy articular cartilage, synovial fluid lubrication, balanced load distribution and supporting musculature allow for smooth, pain-free movement. In coxarthrosis, this harmonious system is disrupted: cartilage thins and fissures, sub-chondral bone becomes sclerotic, osteophytes emerge, joint space narrows, and mobility becomes limited.

Etiologically, coxarthrosis may be classified as primary (idiopathic) or secondary (arising from known underlying conditions). Secondary causes include developmental dysplasia of the hip (DDH), slipped capital femoral epiphysis (SCFE), Legg-Calvé-Perthes disease, trauma (fracture or dislocation), avascular necrosis of the femoral head, inflammatory arthropathies, and metabolic bone conditions. From a mechanistic standpoint, these disturbances alter load distribution, impair joint congruity, lead to abnormal joint stresses, and accelerate cartilage wear.

Given these multiple entry points into disease progression, preventive strategies must be equally multi-faceted. Lifestyle factors—such as obesity, physical inactivity, repetitive joint loading, muscle weakness and poor biomechanics—exert modifiable influences on joint health. Early identification and correction of hip morphologic abnormalities or alignment issues may also forestall progression.

This article is structured to provide a comprehensive review of the origins (etiology) of hip osteoarthritis, followed by evidence-based preventive measures. We conduct a literature review, propose a hypothetical research methodology for prevention-focused intervention, present expected results based on current evidence, and conclude with recommendations for clinicians, public health professionals and patients. In doing so, we hope to emphasise that coxarthrosis is not merely an inevitable consequence of ageing, but rather a preventable condition amenable to timely intervention.

Literature Review

The literature on hip osteoarthritis underscores the multi-factorial nature of its etiology. One of the larger series analysed 965 hips from 886 patients undergoing total hip replacement and found that developmental dysplasia of the hip (DDH) was the most frequent etiologic factor (37.1 %) among the studied population. In the same study, the rates of primary (idiopathic) coxarthrosis were lower (24.4 % in men, 36.2 % in women), with other contributors including avascular necrosis, rheumatoid disease and post-traumatic conditions. Another key review analysing pathogenesis identified inherited hip diseases (e.g., dysplasia), developmental growth disorders (Perthes, SCFE), trauma and inflammatory/metabolic disorders as principal causative agents. This reinforces the concept that cartilage degeneration in the hip is often secondary to altered joint morphology or mechanics rather than purely idiopathic ageing.

Prevention literature emphasises early rehabilitation and kinesitherapy. For example, a study of 27 patients with coxarthrosis underscored the role of kinesitherapy in alleviating pain, protecting joint mechanics and enabling walking rehabilitation. Another investigation into rehabilitation at early stages of hip osteoarthritis following arthroscopy in 95 patients found that tailored rehabilitation protocols allowed effective recovery of function. These works support preventive measures once early joint changes occur.

Despite this, there remains a paucity of large randomized controlled trials explicitly addressing preventive interventions in coxarthrosis. Many studies are retrospective or focus on treatment rather than prevention. Moreover, quality-of-life assessments show that etiology (traumatic vs

non-traumatic) influences functional outcomes in hip arthroplasty patients. In short, while causative factors are increasingly well-defined, the preventive evidence base is still emerging.

Main Body

Etiology and Mechanisms of Coxarthrosis

Coxarthrosis (hip osteoarthritis) evolves when articular cartilage and associated joint structures fail to maintain homeostasis under mechanical, biological or metabolic stress. The pathogenesis begins with increased focal loading, cartilage fissuring, loss of proteoglycans and water content, structural failure of the cartilage matrix, and eventual exposure of sub-chondral bone.

Congenital & Developmental Abnormalities

A major etiologic category includes congenital developmental conditions such as DDH. In the study of nearly 900 patients, DDH was identified as the leading factor among those requiring total hip replacement. Hip dysplasia leads to insufficient acetabular coverage of the femoral head, altered biomechanics, increased contact pressures and premature cartilage breakdown. Growth disorders like SCFE or Perthes disease similarly alter hip morphology and predispose to early degeneration.

Trauma and Post-traumatic Conditions

Fractures of the femoral neck, acetabulum, or dislocations disturb joint congruity, lead to secondary osteoarthritis (secondary coxarthrosis) and accelerate degeneration. Post-traumatic hip joints incur altered load paths, micro-instability and increased wear.

Avascular Necrosis & Inflammatory/Metabolic Conditions

Avascular necrosis (AVN) of the femoral head compromises bone integrity and alters joint loading, promoting osteoarthritis. In the referenced population, AVN accounted for around 10 % of cases in women and 21 % in men. Inflammatory arthropathies or metabolic bone diseases (e.g., osteoporosis, gout, pseudogout) also contribute by altering cartilage nutrition or provoking joint stress.

Mechanical Overload and Age-Related Factors

Even without obvious congenital or traumatic origins, the hip joint is susceptible to degeneration through cumulative mechanical loading, obesity and muscle weakness. Ageing per se reduces cartilage resilience, and overweight increases hip joint load, accelerating wear. Additionally, misalignment (leg length discrepancy, varus/valgus hip alignment) magnifies stress concentration, leading to focal cartilage damage.

Preventive Measures

Given the multifactorial etiologies, preventive strategies must likewise be multidisciplinary.

Weight and Load Management

Maintaining a healthy body mass index reduces load on the hip joint. A reduction in load slows cartilage wear, enhances joint longevity and delays onset of coxarthrosis.

Physical Activity and Muscle Strengthening

Regular targeted exercise strengthens the hip abductors, gluteal muscles and core stabilisers, improving joint stability, distributing load more evenly and reducing focal stress. Rehabilitation studies show improvement in pain and function when kinesiotherapy is applied. Non-weight-bearing therapies (swimming, cycling) may also reduce hip joint stress while maintaining mobility.

Early Detection and Correction of Hip Abnormalities

Screening for hip dysplasia or other morphological abnormalities in younger individuals allows for corrective interventions (osteotomies, proper physiotherapy) before major joint degeneration

occurs. This proactive approach could shift hip osteoarthritis from an inevitable outcome to a preventable event.

Injury Prevention and Safe Biomechanics

Educating individuals (especially athletes or those in physically demanding occupations) about hip joint loading, safe movement patterns, and avoidance of repeated high-impact activities can reduce the risk of early cartilage injury. Professional gymnasts, for example, have been shown to be at risk for hip changes leading to osteonecrosis and early coxarthrosis.

Lifestyle, Nutrition and Joint Health

Adequate nutrition (vitamin D, calcium, and overall bone health), good posture, avoidance of smoking (which impairs bone healing) and managing comorbidities (diabetes, hypertension) contribute to joint resilience.

Integrative Preventive Framework

Combining the above, an integrative preventive framework might include:

1. Identification of individuals at high risk (family history of hip dysplasia, obesity, previous hip injury).
2. Weight management programmes and physical activity prescription.
3. Targeted physiotherapy to strengthen hip-related musculature.
4. Screening imaging to detect hip morphological abnormalities in adolescence or early adulthood.
5. Education around lifestyle, safe biomechanics and hip-preserving strategies.
6. Regular follow-up to monitor hip health and intervene early when changes are detected.

Such a structured preventive pathway holds the promise of reducing the incidence of advanced hip osteoarthritis, decreasing the surgical burden, and improving long-term mobility and quality of life.

Research Methodology

To evaluate the effectiveness of preventive strategies for coxarthrosis, a prospective cohort design is proposed. The target population would comprise individuals aged 30–50 years who have one or more risk factors (e.g., BMI > 30, history of hip injury, diagnosed hip dysplasia, sedentary lifestyle). The cohort would be divided into an intervention group and a control group matched by age, sex and baseline risk profile.

The intervention group would receive a structured preventive programme for 24 months consisting of:

- Nutritional counselling and weight-management support.
- A supervised physiotherapy regime aimed at strengthening hip musculature, improving joint biomechanics, and promoting low-impact aerobic activity (swimming, cycling).
- Screening imaging (hip ultrasound or radiograph) at baseline and 24 months to detect morphological changes.
- Educational modules on hip loading, safe movement patterns and injury prevention.

The control group would receive usual care (general advice on healthy lifestyle) but no structured programme. Outcome measures would include:

- Primary outcome: incidence of radiographic hip osteoarthritis at 24 months (measured by joint space narrowing, osteophyte formation).

- Secondary outcomes: hip pain (measured via visual analogue scale), hip function (via Hip disability and Osteoarthritis Outcome Score—HOOS), hip muscle strength (measured by dynamometry), and quality of life (SF-36 questionnaire). Data collection at baseline, 12 months and 24 months would allow for temporal analysis. Statistical analysis would utilise intention-to-treat principles, comparing the cumulative incidence of radiographic osteoarthritis and changes in functional/pain scores between groups (Chi-square for categorical outcomes, t-test/linear mixed models for continuous outcomes). Confounding factors (age, sex, baseline BMI, comorbidities) would be adjusted. This methodology enables assessment of whether a preventive programme can delay structural hip degeneration and improve joint-related outcomes.

Results

Based on analogous studies and plausible effect sizes, it is anticipated that the intervention group will show:

- A lower incidence of new or progressive radiographic signs of hip osteoarthritis (e.g., joint space narrowing) compared to the control group over 24 months.
- Significant improvements in hip muscle strength in the intervention group, leading to better functional scores (HOOS) and reduced hip pain (VAS).
- Enhanced quality of life (SF-36 scores) in the intervention group due to improved mobility and decreased pain.
- Secondary benefits such as weight reduction, improved metabolic parameters (if tracked), and fewer hip joint injuries over the study period.

For example, previous rehabilitation studies in early coxarthrosis showed that kinesitherapy alleviated pain and improved walking function. Similarly, patients with early hip joint pathology undergoing structured rehabilitation achieved full functional recovery. Although direct preventive trials in hip osteoarthritis are limited, these findings support the hypothesis that early intervention can favourably modify the disease trajectory. The expected results would suggest that a multifaceted preventive approach can not only improve joint-related symptoms but also decelerate structural hip degeneration, reducing the need for surgical interventions in the long term.

Conclusion

Hip osteoarthritis—or coxarthrosis—is often framed as an inevitable consequence of ageing, but a careful review of the literature reveals that its origins are complex and, crucially, modifiable. Developmental hip dysplasia, trauma, avascular necrosis, mechanical overload and metabolic insults are among the principal causative factors. Structural hip abnormalities, repeated joint stress and muscle weakness set the stage for progressive cartilage degeneration and eventual joint dysfunction.

This article has shown that prevention of hip osteoarthritis is a realistic objective. It requires shifting focus from late-stage management to early detection, lifestyle modification, biomechanics optimisation, weight control and targeted muscle strengthening. While the evidence for prevention in hip OA is less robust than that for knee OA, emerging studies support the premise that early rehabilitation and risk-factor control can improve outcomes and delay disease progression.

Our proposed research methodology underscores the value of long-term, structured preventive programmes in high-risk populations. Anticipated results suggest that such programmes can reduce radiographic progression, alleviate symptoms and enhance quality of life—thereby potentially lowering the rate of hip replacements and related healthcare burden.

For clinicians, the implication is clear: identify individuals at risk (those with hip dysplasia, prior hip injury, obesity or sedentary lifestyle) and initiate preventive strategies proactively. For public health, the call is to integrate hip-joint health into musculoskeletal prevention campaigns, emphasising the importance of weight management, physical activity and safe hip mechanics from an early age.

In sum, the key message is this: do not wait until hip osteoarthritis is advanced—start upstream. By intervening early and comprehensively, we may preserve hip joint health, delay or avoid the need for invasive surgical treatments, and enhance the mobility and wellbeing of individuals across the lifespan. Future research should prioritise high-quality preventive trials, long-term follow-up, and cost-effectiveness analyses to determine how best to implement hip-preserving strategies in diverse populations.

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