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General

The Association Between Charcot-Marie-Tooth Disease and Developmental Dysplasia of the Hip: A Narrative Review

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Charcot-Marie-Tooth (CMT) disease, a condition comprising a variety of inherited peripheral neuropathies, is marked by progressive degeneration of motor and sensory nerves. The current review examines the link between CMT and developmental dysplasia of the hip (DDH), characterized by abnormal hip joint development. Although the frequency of DDH among CMT patients has been under-investigated, the musculoskeletal complications of CMT (including weakness and abnormal gait) may predispose individuals to hip dysplasia. This narrative review compiles data from 11 publications selected from a systematic search of PubMed and Web of Science. The criteria included established diagnosis of CMT and DDH in patients aged less than 14 years, English language, and availability of full text. This review examined patient characteristics, genetics, diagnosis, and treatment. Research demonstrates a high incidence of hip dysplasia among CMT patients, with some studies reporting 22% in clinical series. Patterns of diagnosis differ among subtypes, with earlier onset of Type-1 CMT. This link is influenced by genetic factors, such as duplication of the PMP22 gene, and family studies show vertical transmission. Diagnosis includes physical examination, X-rays and genetic analysis. Early intervention, including non-surgical and surgical treatment in severe cases, is crucial to management. The link between CMT and DDH highlights the importance of awareness and screening in at-risk groups. Appropriate screening and management algorithms can enhance quality of life. Additional epidemiological and genetic studies are needed to improve diagnostic and treatment strategies.

1. INTRODUCTION

Charcot-Marie-Tooth (CMT) disease is a heterogeneous group of inherited peripheral neuropathies resulting in progressive degeneration of motor and sensory nerves.[1] This degeneration results in substantial clinical symptoms such as weakness, wasting, and loss of sensation. There are more than 90 known genetic mutations that cause CMT, which can broadly be divided into two main subtypes: demyelinating (CMT type 1, CMT1) and axonal (CMT type 2, CMT2).[2] Each subtype displays unique clinical characteristics, inheritance patterns, and age of onset, highlighting the complexity and heterogeneity of this condition.[2] Continued research is crucial to comprehensively elucidate the effects of CMT on individuals, especially concerning its correlation with diverse musculoskeletal abnormalities, including developmental dysplasia of the hip (DDH). Hip dysplasia, especially DDH, is a condition that affects how the hip joint develops.[3] This malformation can result in dislocation, subluxation and complications if not addressed appropriately.[4] In the context of CMT, the occurrence and significance of hip dysplasia have not been well studied, although initial research has shown a significant prevalence of this condition in CMT patients. The distinctive musculoskeletal complications of CMT, such as muscle weakness, gait dysfunction and impaired proprioception, may predispose individuals to the development of hip dysplasia.[5] These complications can hinder diagnosis and treatment of affected individuals, as musculoskeletal changes in CMT can obscure or worsen underlying hip conditions.[6] Recent studies suggest that hip dysplasia in CMT patients may often be asymptomatic in early stages, resulting in misdiagnosis or delayed treatment.[2,7] Furthermore, there is emerging evidence that genetic factors may play a significant role in the development of hip dysplasia in this population, with familial clustering suggesting a potential hereditary basis.[8,9] Understanding the relationship between CMT and hip dysplasia is crucial for early detection, timely intervention, and improved patient outcomes.[9] Through a critical examination of existing literature and discussion of the potential mechanisms of this relationship, the review highlights the importance of awareness and research into orthopedic complications of CMT. This effort seeks to inform manage-

ment approaches for patients at risk of developing hip dysplasia, improving the outcomes of the disease in affected patients.

2. METHODS

This narrative review discusses the outcomes of studies on CMT and DDH. Our review was conducted between August and November of 2024 where we initially found 78 articles using a Boolean MeSH search query on the Web of Science and PubMed databases, and then screened the titles and removed duplicates via the Rayyan tool. 24 articles satisfied the inclusion and exclusion criteria, including the presence of CMT and DDH, age 14 years or younger, English language, and full text available. Exclusion criteria included articles that addressed other types of neuromuscular or skeletal diseases, previous hip surgery or abstracts. Following a comprehensive full-text review, 11 articles were included in this narrative review. The relevance of the studies was evaluated based on the research question, including patient characteristics, genetics, clinical and radiological presentation, physical and functional examination, orthopedic and neurological interventions, prevalence and epidemiology, and physical activity and lifestyle. The results were interpreted qualitatively, focusing on patterns and observations across studies rather than statistical results, as is the nature of a narrative review.

Boolean search terms: Charcot-Marie-Tooth disease, CMT, hereditary motor and sensory neuropathy, hip dysplasia, developmental dysplasia of the hip (DDH), Charcot-Marie-Tooth disease AND hip dysplasia, CMT AND hip dysplasia, Charcot-Marie-Tooth disease AND developmental dysplasia of the hip, hereditary motor and sensory neuropathy AND hip dysplasia, musculoskeletal complications of CMT, genetic neuropathies and hip disorders, neuromuscular disorders AND hip abnormalities, orthopedic manifestations of Charcot-Marie-Tooth disease, CMT AND joint dysplasia, CMT AND skeletal deformities, prevalence of hip dysplasia in CMT, CMT subtypes AND hip joint issues, gait abnormalities in CMT AND hip dysplasia, orthopedic interventions in CMT patients with hip dysplasia, neuropathy AND hip joint, genetic disorders AND hip dysplasia, peripheral neuropathies AND skeletal deformities.

ETHICAL CONSIDERATIONS

This study is a narrative review based on previously published literature retrieved from publicly available databases. It did not involve the collection of primary data, direct human or animal subjects, clinical interventions, or access to identifiable patient information. As such, ethical review and approval by an Institutional Review Board (IRB) or ethics committee were not required. No informed consent was necessary, as no participants were recruited and no personal data were collected or processed in the course of this review.

3. RESULTS

3.1. PREVALENCE AND EPIDEMIOLOGY

Research on the prevalence of hip dysplasia within the CMT population remains limited, although recent studies are shedding more light on the condition.[1] A cross-sectional study identified 5 of 30 CMT patients to have hip dysplasia in the sample population.[2] A clinical series has reported its occurrence in patients of various ages and both sexes, with 19 hips affected in 14 patients,[4] and that 22% of CMT patients had previous hip surgeries.[10] There seems to be an age-based pattern depending on the CMT sub-type, with type-1 typically being recognized in the first to second decade of life, while type-2 would emerge later on in the second decade or later.[11] A familial component is evident in CMT-associated hip dysplasia. Stover et al. (2013) reported hip dysplasia in three siblings within a single family,[12] while Bamford et al. (2009) reported a case of hip dysplasia in multiple generations of a family.[8] However, due to the asymptomatic nature of hip dysplasia in CMT patients, some cases remain undiagnosed until adolescence or adulthood.[1,7,13] These findings suggest that hip dysplasia in CMT patients might be more prevalent than previously thought, requiring larger epidemiological studies to accurately describe prevalence rates.

3.2. AGE OF ONSET AND DIAGNOSIS PATTERNS

Patterns of diagnosis for hip dysplasia in CMT patients differ, with Type-1 CMT patients frequently presenting with hip abnormalities during early adolescence, while Type-2 CMT presents later, after the second decade of life.[11] Standard diagnostic procedures include a physical examination, radiography, and a detailed family history.[11] Diagnosing hip dysplasia in CMT patients typically involves a comprehensive evaluation. Signs of weakness in the hip abductors, leading to a Trendelenburg gait, may be observed on physical examination, as described by Driscoll and Skinner (2008).[5] Radiographic imaging and genetic testing can also be used to confirm the diagnosis as described by Chan et al. (2006).[6] Restricted range of motion across various directions is typical, particularly in advanced cases.[13] Many diagnoses are made incidentally due to the asymptomatic nature of the condition, often when radiographs are taken for unrelated reasons.[13] Radiographic diagnosis typically requires criteria such as a neck-shaft angle over 147°, center-edge angles below 20°, and a Reimer's migration ratio of at least 20%.[11] In contrast, DDH presents with less severe pathologies than those seen with hip dysplasia in CMT, which would display greater degrees of subluxation and arthritic changes.[4] This progression is due to late diagnosis of symptoms and underlying neuromuscular pathology.[10] Finally, a comprehensive family history is crucial as the condition manifests across multiple generations within a single family,[1] emphasizing the importance of screening individuals with risk factors.

3.3. GENETIC FACTORS ASSOCIATED WITH CMT-RELATED HIP DYSPLASIA

CMT disease ranks among the most frequently inherited neurological disorders, with over 90 distinct forms differing by inheritance patterns, age of onset, symptoms, and progression.[1,2] It has been classified into two major subtypes: type-1 and type-2.[11] Even though autosomal recessive, X chromosome-linked, and sporadic transmission have been reported, both subtypes are primarily associated with an autosomal dominant pattern.[1,11] Type-1 CMT is the most common type and has a higher rate of incidence compared to type-2.[1] The most common genetic defect seen in it is caused by the duplication of the PMP22 gene, which is linked to the chromosome 17p11.2 and it is the most common cause of sporadic mutation and accounts for more than 50% of CMT cases for both subtypes.[1,11] In addition, familial studies have also highlighted the significance of genetic factors in documented cases of vertical transmission across multiple generations.[1,14] Bamford et al., for instance, documented a patient who shared that both her mother and grandmother had been diagnosed with CMT.[8] Furthermore, Stover et al. documented an example where three siblings (one male, two females) accounted for 5 out of 19 hip dysplasia cases in their surgical series.[15] These genetic patterns emphasize the importance of comprehensive family history assessment and genetic counseling in the management of CMT-associated hip dysplasia, particularly for family planning and early screening of at-risk individuals.[11]

3.4. DIAGNOSTIC METHODS AND RADIOGRAPHIC MEASUREMENTS

A standard evaluation for the diagnosis of a child with CMT begins with a thorough approach that includes the family history and a physical examination.[11] A significant family history and clinical signs lead to a high suspicion of the presence of the disease.[11] However, to confirm the diagnosis and help in distinguishing the subtypes, electro-physiologic testing, nerve conduction velocity, or DNA analysis can be done; nevertheless, a negative DNA analysis sample is not enough to rule out CMT due to the wide variety of genetic mutations seen.[11] In addition, although less favorable, and uncommon, a sural nerve biopsy could also be used to confirm the diagnosis of CMT.[11] In type-1, the electro-physiologic testing would show a slow conduction velocity of <38 m/s in comparison to type-2, which would be normal or slightly prolonged.[11] As soon as the diagnosis of CMT is established, a standing anteroposterior radiograph of the pelvis should be done.[11] Most of the critical and reliable measurements differed in the literature; however, the ones repeated the most were the lateral center edge angle (LCEA) of Wiberg, anterior center edge angle (ACEA) of Lequesene and de Seze, acetabular index, and Tönnis angle, which was specific to osteoarthritis grading.[1,3,10,11,13] Wiberg's LCEA, a vital indicator of dysplasia, suggests dysplastic changes when values are under 20°.[3,4,10] In addition to plain radiographs, CT or MRI could be considered in selected cases. A three-dimensional CT may be able to provide details about the femoral head in relation to the acetabulum to aid in surgical planning, while an MRI is used to evaluate the hip when an unossified femoral head is resistant to conservative treatment and is otherwise inadequate for other imaging modalities.[13] An ultrasound is helpful at birth if hip laxity is appreciated; however, this condition would be classified as DDH and should be treated as such.[11,13] On the other hand, if an infant is diagnosed with CMT type-1, a screening ultrasound is recommended, and if the infant is diagnosed with the other variant, type-2, a routine pelvic radiograph at least every 2 years is recommended.[7,13] Besides that, other imaging modalities have little to no role in the evaluation of hip disease in CMT.[11] If the hips display dysplasia, a thorough evaluation noting the amount of acetabular dysplasia, femoral-head coverage, femoral neck valgus and degree of migration or subluxation of the femoral anteversion, and degenerative changes must be done.[1,11,13] To accurately determine the true amount of femoral anteversion, a supine pelvic radiograph with the femur in 20 to 30 degrees of abduction and 20 to 30 degrees of internal rotation should be done.[11] Furthermore, CT imaging, particularly three-dimensional reconstruction, may be helpful in the quantitative analysis of acetabular dysplasia and identifying any rotational deformities of the femur.[11]

3.5. TREATMENT AND APPROACH OUTCOMES

Hip dysplasia in the CMT population requires treatment, even when asymptomatic.[11] Conservative management options include the use of assistive devices such as Ankle Foot Orthosis (AFO) and Knee Foot Orthosis (KFO) for stabilizing joints[7]; however, when conservative management fails to provide adequate relief, operative intervention may be necessary.[13] Correction of acetabular deficiency should be prioritized, as performing an initial femoral varus osteotomy may worsen Trendelenburg gait.[11] Once corrected, the femoral component, including the full range of motion and proper seating of the femoral head within the hip joint, should be tested in addition to assessing if there are any signs of impingement or subluxation.[11] Then, if the dysplasia and subluxation do not resolve, the deformity in the proximal femur should be addressed.[11] If it is due to anteversion, derotation should be performed; however, if it is due to increased neck valgus, a varus osteotomy such as periacetabular osteotomy, Chiari osteotomy, or Salter with a concomitant shelf procedure will need to be performed.[7, 11] The Bernese periacetabular osteotomy (PAO) has demonstrated success. Studies with 14 and 27 patients presenting with symptomatic hips showed significant radiographic improvements, with the mean LCEA increasing from -7.3° to 28°, the acetabular index decreasing from 30.3° to 6.2°, and the vertical center-edge angle (VCEA) rising from -18.6° to 22°.[3,10,15] Other surgical options include triple innominate osteotomy and proximal femoral varus osteotomy.[1, 11] For some patients, total hip replacement may be necessary, as reported in the case of a 21-year-old patient who progressed from shelf osteotomy to total hip replacement.[3, 11] Care must be taken when performing these varus osteotomies because they might result in the worsening of the Trendelenburg gait but reduce the chances of recur-

rent subluxation.[1] If excessive, they might weaken the fragile hip abductors or cause the development of compressive peroneal nerve palsy.[11] In addition, one case of femoral head avascular necrosis and five pubic ramus fractures were noted as complications.[15] Post-operative complications are usually classified according to the Dindo-Clavien complication scheme for the hip preservation surgeries in which complications are graded from one to five in severity, which are based on the long-term treatments and morbidity associated with the complications.[10] Grade 1 would require little to no change in post-operative care, grade 2 requires modifications in the outpatient care, grade 3 involves invasive intervention (surgical or radiological), grade 4 includes potential life-threatening complications that include hip arthroplasty or those with high long-term morbidity, and grade 5 complications would involve death.[10] It is also important to monitor post-operatively, as it has been found that 37% of cases had progression in Tönnis grading post-surgery, from which it is clear that this condition is difficult to treat.[3]

4. DISCUSSION

This review looks at the incidence, diagnosis, genetics, and treatment of hip dysplasia in patients with CMT disease, an inherited motor-sensory neuropathy. Although there is a lack of knowledge, recent research has shed light on the epidemiology, genetics and pathology of hip dysplasia in this unique group of patients. Our review found a high prevalence of hip dysplasia in CMT patients, with some reports of 16-22% having hip abnormalities.[10] Earlier studies, like that of Langlais et al., reported a lower prevalence of 6% of patients with CMT in their cohort having hip dysplasia.[4] The prevalence in our review indicates that CMT does predispose patients to hip abnormalities to a greater extent than previously believed, and suggests the need for early diagnosis and screening. Age of onset of hip dysplasia among CMT patients seems to be related to CMT type. Hip abnormalities occur in the first to second decades in type-1 CMT, but later in type-2. This is also seen in the review by Yagerman et al., which demonstrates that early onset of hip dysplasia is associated with greater severity of the disease.[10] It highlights the need for age-specific screening, especially for those with type-1 CMT.[7,8] CMT is known to be caused by the duplication of the PMP22 gene, one of the main causes of hip dysplasia. Research by Novais et al. and others show family members affected in multiple cases, and demonstrate the heritability of the disease.[1,4] These reports highlight the need for genetic counseling and consideration of family history when managing hip dysplasia in this population. Hip dysplasia in CMT patients can be difficult to diagnose as it is often asymptomatic in its early stages. Clinical examinations are not sufficient, and X-rays are required.[16,17] Markers such as the neck-shaft angle and center-edge angle have been used to diagnose hip dysplasia. Our study backs up previous work by Canavese and others on using these parameters to correctly diagnose hip dysplasia.[18] Early imaging can assist in early treatment, which can alleviate the results for CMT patients. CMT-related hip dysplasia is more severe than DDH. As Saran N et al. (2019) point out, the underlying deformities of CMT-related hip dysplasia often resulted in severe arthritic changes due to delays in diagnosis.[14] This difference from DDH implies that specific interventions are more suitable for CMT-related hip dysplasia, as it has special characteristics: progression and severity.[10] CMT patients commonly experience muscle weakness, mainly of the hip abductors, which may exacerbate hip instability. The presence of the Trendelenburg gait, observed in these patients, is indicative of hip stability weakness.[19] This is consistent with the literature review, which notes that neuromuscular weakness is a factor in progressive hip deformity in CMT patients.[10] This relationship suggests that therapeutic strategies that strengthen muscles may reduce the progression of hip dysplasia. Conservative management, including orthotics, is frequent, but surgery is often required for CMT patients with severe dysplasia. The Bernese periacetabular osteotomy (PAO) has been reported with good results, including improvements in center-edge angles and acetabular index. Stover et al. reported similar surgical outcomes but with greater complications in CMT dysplasia compared with DDH.[12] This indicates that despite the complexity of the surgery, PAO may be highly beneficial for certain patients. Surgery on CMT patients is not without its risks, including fractures of the pubic ramus and palsy of the peroneal nerve. This study confirms Saran's report, which shows higher complication rates in PAO for CMT patients than other patients.[14] Risks can be minimized through the consideration of the surgical approach and careful management of the patient in the postoperative period, but complications can still occur. Regular follow-up is essential as CMT patients are at risk of degeneration following surgery. Stover et al. reported that worsening of Tönnis grading is often seen in hip dysplasia patients following surgery.[15] Therefore, regular follow-up with routine periodic X-rays is advisable to address recurrent problems.

Some limitations are noted in this study. The sample size is not large enough to represent all CMT patients with hip dysplasia, as the condition's variability requires a larger number of studies. As early hip dysplasia in CMT patients can be asymptomatic, it is possible that some cases were overlooked in our study, particularly in cases where patients have not undergone regular imaging. The study is also dependent on the available literature, and may have been influenced by any biases they may have had, particularly those with small sample sizes or limited diagnostic capabilities. There needs to be more studies on larger multicenter studies of hip dysplasia in CMT patients to better understand its prevalence and progression. Studies with repeated imaging and follow-up would help diagnose early cases, and provide opportunities for early intervention. Genetic studies are also needed to identify how different subtypes of CMT predispose patients to developing hip dysplasia, and how this could be treated. Guidelines, specific to conservative and surgical treatment of hip dysplasia in CMT patients, would help clinicians and enhance patient care.

5. CONCLUSION

Hip dysplasia is a frequent musculoskeletal complication that is seen in CMT disease. Despite the underdiagnosis of CMT, hip dysplasia becomes more recognizable, especially in specific genetic subtypes and familial patterns. Our narrative review signifies the risks and the complexity of treating hip dysplasia in CMT patients, which can increase due to multiple factors such as a delay in diagnosis, patients who have multiple neuromuscular impairments in addition to CMT, and genetic predisposition. Meanwhile, improvement in radiological imaging and the advanced methods of genetic testing, which can be used for early diagnosis of CMT, help in specifying the abnormality and deciding the optimal management plan, which can range from conservative management with orthotic devices up to surgical procedures like periacetabular osteotomy or total hip replacement surgery. In addition, hip arthropathies, postoperative complications, and gaps in our current knowledge of the genetic and biochemical mechanisms are limitations that still exist despite the advancement. In conclusion, this review emphasizes the necessity of additional research in CMT, and more interdisciplinary care is needed to reach the maximum improvement in quality of life in patients with hip dysplasia who have CMT.

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AUTHORS' CONTRIBUTIONS

K.A.A. conceptualized the study, designed the review methodology, and drafted the manuscript. L.O.A. and R.A.A. conducted the systematic database search and article screening. R.O.A. and A.Y.A. performed the full-text review and data extraction. Z.T.G. and A.A.A. contributed to the synthesis and interpretation of findings. A.A.A.A. and M.S.A. contributed to writing and critically revising the manuscript. A.A. supervised the overall process and provided final approval of the version to be published. All authors have read and agreed to the published version of the manuscript.

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The authors declare no conflicts of interest.

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