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Primary Total Hip Arthroplasty for Charcot Arthropathy is Associated With High Complications but Improved Clinical Outcomes

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ABSTRACT

Background: Neuropathic (Charcot) arthropathy of the hip is rare but can lead to joint destruction, bone loss, and dysfunction. While total hip arthroplasty (THA) may be considered a treatment option, only very limited data in the form of case reports are available on the results of THA. The goal of this study was to analyze the outcomes of primary THA for Charcot arthropathy with emphasis on implant survivorship, complications, and clinical outcomes.

Methods: Eleven patients undergoing 12 primary THAs for Charcot arthropathy from 2007 to 2014 were retrospectively reviewed. All patients had a severe underlying neuropathy and clear radiographic evidence of Charcot arthropathy. Mean age was 54 years with 4 patients being female. Mean follow-up was 5 years.

Results: Survivorship free of any revision was 75% at both 2 and 5 years. Three THAs (3/12) were revised: 2 for recurrent instability and 1 for femoral component loosening. Survivorship free of any reoperation was 67% at both 2 and 5 years. One additional THA underwent open reduction and internal fixation of a Vancouver B₁ periprosthetic fracture. The overall complication rate (including revisions and reoperations) was high at 58% with 3 recurrent dislocations, 2 periprosthetic fractures, 1 femoral component loosening, and 1 delayed wound healing. Harris Hip Scores improved from a mean of 43 preoperatively to 81 postoperatively (P < .001).

Conclusion: In this study, the largest to date, we found that patients undergoing primary THA for Charcot arthropathy have a significant improvement in clinical outcomes but that there was a high risk of early complications and revisions, mostly related to recurrent instability. Specific precautions to avoid early complications, namely utilization of components that provide robust fixation and strategies that provide enhanced hip stability, should be considered. *Level of Evidence:* Level IV.

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9

Neuropathic arthropathy, colloquially referred to as Charcot arthropathy, is thought to result from a lack of periarticular sensory and nociceptive feedback, resulting in microtrauma [1-8]. This repetitive articular insult can lead to progressive joint destruction and deformity [4-8]. Classic radiographic findings include

progressive destructive changes with bone fragmentation, often resulting in bone loss and soft tissue attenuation [4-8]. While the condition is often less painful than would be expected from the radiographic appearance due to the underlying theorized pathology, Charcot arthropathy can result in notable functional impairment [4-8].

Charcot arthropathy has been more commonly described in the foot, ankle, and knee, but the hip may become involved as well [4,5,9]. While total knee arthroplasty (TKA) for end-stage knee Charcot neuropathy has been described with good results utilizing surgical techniques and implants typically reserved for revision procedures [4,5], there are only a few case reports describing the outcomes of total hip arthroplasty (THA) for end-stage Charcot

Investigation was performed at the Mayo Clinic, Rochester, MN.

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2

arthropathy in the hip [6–8]. In theory, patients with underlying hip Charcot arthropathy might be at higher risk of complications, specifically dislocation, secondary to their underlying neurologic condition. It has been well established that other underlying neurologic conditions, including Parkinson's disease, cerebral palsy, and poliomyelitis, increase dislocations after THA [10].

The goal of this study was to analyze the outcomes of THA for end-stage Charcot arthropathy at a single institution with specific focus on (1) survivorship free of revision or reoperation, (2) complications, (3) clinical outcomes, and (4) radiographic outcomes.

Patients and Methods

Eleven patients treated with 12 primary THAs for a diagnosis of Charcot arthropathy at single academic institution from January 1, 2007 to December 31, 2014 were identified through our institution's total joint registry, which captures Charcot and/or neuropathic arthropathy as a diagnosis. Thorough analysis of each patient's medical records and radiographs was performed to rule out other diagnoses and further substantiate the diagnosis of Charcot arthropathy. In all, 11 of the originally identified 28 patients met inclusion criteria; the other 16 patients met one of the following clinical or radiographic exclusions. While there is no definitive test to diagnose Charcot arthropathy, a specific set of criteria was evaluated in each patient for inclusion into this study. Patients had a clinical history of progressive functional loss in a short period of time, typically with pain levels disproportionately milder than expected base upon the severity of their radiographic disease. All patients had a severe underlying neurologic condition with an element of peripheral neuropathy as evaluated and diagnosed by a neurologist. Patients were excluded if they did not have classic radiographic findings of Charcot arthropathy, even in the presence of a severe neurologic condition such as Charcot-Marie-Tooth disease. Patients were also excluded if no underlying neurologic condition was present that could account for a Charcot radiographic appearance of the hip joint. Underlying neurologic conditions included Charcot-Marie-Tooth disease (3/12), severely uncontrolled diabetes mellitus in 2 patients (3/12), incomplete paraplegia secondary to prior spinal cord injury (2/12), lysosomal storage disease causing severe neuropathy (1/12), Guillain-Barre syndrome (1/12), severe prior head injury with neurologic sequelae (1/12), and severe cerebrovascular accident with neurologic sequelae (1/12).

On physical examination, all patients had a leg-length discrepancy and notable peripheral neuropathy. Mean preoperative hip flexion was 80 degrees, and abduction was 25 degrees; 3 patients had hip flexion contractures of at least 20 degrees; no patients had joint hypermobility. Radiographically, all patients had classic signs of Charcot arthropathy with progressive destructive joint space loss, bone loss, and bone fragmentation (Fig. 1). Several radiographs of patients included in this series (Figs. 2 and 3) were included to illustrate the severity of the hip pathology. While we do not have

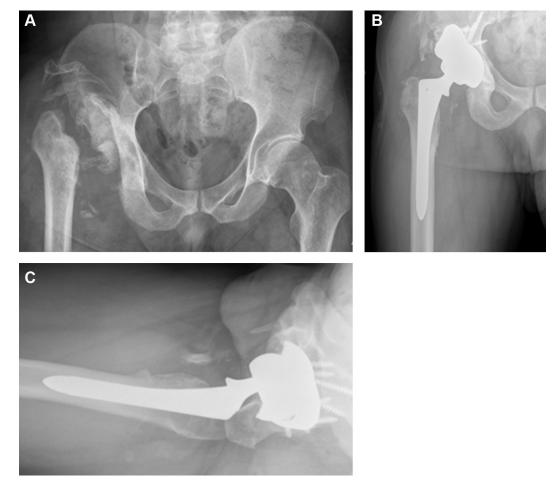


Fig. 1. An anteroposterior (AP) radiograph (A) of a 45-year-old male (patient 7) with incomplete paraplegia secondary to cauda equina syndrome with destructive changes in the hip joint consistent with Charcot arthropathy. He underwent a THA with a tantalum acetabular cup, a tantalum augment, and supplemental screw fixation for a Paprosky type 3A acetabular bone defect and a fully coated femoral stem as noted in the AP hip (B) and cross-table lateral (C) radiographs.

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