

Arthroplasty of a Charcot knee

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Abstract

The Charcot knee - or neuropathic arthropathy - presents a considerable challenge to the orthopaedic surgeon. Caused by a combination of sensory, motor and autonomic neuropathy, it was originally described as an arthritic sequelae of neurosyphilis. In today's western orthopaedics it is more often caused by diabetes. A Charcot knee is often symptomatically painful and unstable. Traditional management has usually been conservative or arthrodesis, with limited success. Arthroplasty of a Charcot joint has commonly been avoided at all costs. However, in the right patient, using the right technique, arthroplasty can significantly improve the symptoms of a Charcot joint. This article explores the evidence surrounding the role of arthroplasty in the management of a Charcot knee. Arthroplasty is compared to other forms of treatment and specific patient demographics and surgical techniques are explored in an attempt to define the role of arthroplasty in the management of a Charcot knee.

Introduction

The Charcot joint or neuropathic arthropathy was first described by Charcot as an arthritic sequelae of neurosyphilis. It results in significant joint destruction and instability. Albeit rare, a Charcot knee presents a considerable challenge to the orthopaedic surgeon. Literature surrounding the Charcot knee and especially the role of arthroplasty in its management is limited and in some respects contradictory. This article will attempt to summarize the evidence concerning arthroplasty and the Charcot knee.

Pathophysiology

The pathophysiology of a Charcot joint is still not fully understood. Two theories currently exist; the French and German theories. The first supports the idea that the osteopenia associated with a Charcot joint results from autonomic dysfunction. This dysfunction leads

to increased blood flow to the joint caused by decreased sympathetic tone from damage to the trophic centres.^{1,4} This theory is well supported by perfusion and temperature studies of neuropathic joints.⁵ A five-fold increased in perfusion with diabetic neuropathy has been noted. This increased perfusion is only minimally affected with sympathetic arousal stimuli in those with non-painful neuropathy. This is in contrast to those with painful neuropathy, where sympathetic arousal stimuli are effective vasoconstrictors.⁵

The German theory implicates trauma as the underlying cause of joint disease.⁶ The patient's decreased nociceptive response due to neuropathy, coupled with ongoing activity and micro-trauma, results in joint and ligament damage. This trauma ignites the body's inflammatory response causing further deformity through bony resorption.⁷

A combination of the two theories provides a viable explanation for the pathogenesis of neuropathic joint disease. The Charcot joint is likely to be a result of co-concomitant sensory, motor and autonomic neuropathy. The sensory neuropathy results in decreased sensation, proprioception and protective reflexes to the joint, leading to damage from undetected trauma. The motor neuropathy results in decreased support for the joint, exacerbating instability. The autonomic neuropathy increases blood flow to the joint, causing resorption and subsequent osteopenia of the bone.^{8,11}

Demographics

Charcot's description of the disease implicated tertiary syphilis as the underlying cause. Many other diagnoses resulting in a neuropathic arthropathy have thereafter been identified. These include leprosy, alcoholism, syringomyelia, lacunar infarct,¹² hereditary cerebral and cerebellar atrophy¹³ and, more commonly in modern times, diabetes mellitus (DM).¹⁴

The distribution of the neuroarthropathy is partly determined by its underlying cause. Tertiary syphilis often affects large weight bearing joints such as the knee. Syringomyelia more commonly afflicts the joints of the upper extremity such as shoulder and elbow.⁷ Diabetes mellitus is more likely to affect the foot and ankle.⁷

With increasing use of antibiotics, syphilis as a cause of neuroarthropathy is declining. In the modern age, diabetes has taken over as the major cause of the Charcot joint.^{12,15} A patient suffering from DM has a 7.5% chance of suffering from neuroarthropathy. This number increases almost 4-fold in patients with co-concomitant peripheral neuropathy.¹⁶

Due to the epidemiological change of underlying cause from syphilis to DM, the ankle and feet are now more commonly affected than the knee.¹⁴ In a series of patients suffering from neuroarthropathy as a result of DM, only 6% of the affected joints were knees. The majority

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(64%) of affected joints were feet.¹⁴ Hence, in effect, 0.45% of all diabetic patients are likely to suffer from neuroarthropathy of the knee. This should be kept in mind when a diabetic patient presents with a diagnosis of osteoarthritis seeking joint replacement. With the ever increasing number of diabetic patients in the western world and the limited literature regarding the treatment of a Charcot knee, the neuroarthritic knee may once again present as common surgical problem in the near future.

Signs and symptoms

Neuropathic arthropathy has classically been described as a painless arthropathy. However pain and instability are the two major presenting complaints of the Charcot knee sufferer. Mild to moderate pain is usually felt early in the course of the disease, during the destructive process.¹⁷ This pain is usually worse on walking and rare during rest.¹⁸ The degree of pain does not correspond with the level of joint destruction. This results in neuropathic arthropathy's classic description as relatively painless.⁷ The instability is attributed to the decreased proprioception leading to extremes of range of motion, ligamentous laxity, effusion and bony destruction.^{7,12} Loss of deep tendon reflexes is common in this population, especially when syphilis is the underlying cause.¹⁹ Oedema and joint temperature are indicative of the stage of the disease. A Charcot joint has been reported to undergo three phases, largely determined by the radiographic findings (*see below*). The first phase (developmental phase) is clinically characterized by strong pulses and a red, hot, oedematous joint. In the next two phases (coalescent and reconstruction phases) the joint tempera-

ture drops to be similar to the contralateral side, and the oedema begins to resolve.⁴

Investigations

An x-ray is a useful tool in diagnosing and determining the extent of joint disease. Findings of subluxation, fragmentation, bony destruction and the presence of periaricular bone formation are all common in a Charcot joint¹² (Figures 1 and 2). Acute destruction and periaricular fragmentation of bone is seen in the developmental phase. The bony debris is seen to be absorbed and sclerosis evident in the coalescent phase. This progresses to radiological signs of fusion in the reconstructive phase.⁴ It is important to recognize the phase of disease as it has bearings on management.

Further imaging using a CT or MRI does not add much clinical significance. An MRI can be useful in excluding other pathology. A Charcot joint usually exhibits low signal intensity on both T1 and T2 weighted images. High signal intensity in T2-weighted imaging can indicate the presence of osteomyelitis, tumours, trauma or osteonecrosis.²⁰

The diagnostic gold standard for a Charcot joint still remains the histological appearance of fragments of bone and cartilage embedded within the synovium.^{10,21,22}

Non-arthroplasty management

Non-operative

Conservative management is preferred in this subset of patients, as surgical management can be difficult and demanding.

The role of conservative management is to prevent further injury to the joint.⁷ The mainstay of treatment is to instigate a non-weight-bearing regime as soon as possible, especially

in the early destructive phase.^{4,7,9,14} Non-weight-bearing can be augmented with elimination of movement at the joint either via a brace, plaster or total contact cast.^{4,7,9,14,23} A non-weight-bearing period of 3 months has been recommended by some.⁷ Early conservative management may alter the progression of certain neuropathic disorders.^{4,22}

Adjuncts to conservative management include the use of bisphosphonates and low intensity ultrasound. Bisphosphonates have been reported to help stop osseous destruction by preventing bony resorption. In one study, six patients with neuropathic arthropathy were treated with bisphosphonates and monitored using temperature sensors. This resulted in a reduced temperature reading in all 6 patients within 2 weeks.²⁴ This was verified with a randomized control trial of 39 patients comparing bisphosphonates with placebo. The active group was found to have significantly reduced bone turn-over, disease activity and consequently symptoms.²⁵ However, more research is needed to substantiate the effectiveness of bisphosphonates in neuropathic arthropathy. Low intensity ultrasound is thought to be beneficial in the management of a Charcot foot; hence its use may also be applicable to the knee.¹⁷ Again, more research is required to determine efficacy. Failure of conservative measures is common due to the difficulty in minimizing severe instability and shearing stress.¹⁹

Arthrodesis

The severe instability, soft-tissue laxity and bony destruction often renders conservative management as futile and arthroplasty as high risk.²³ Hence, the surgical treatment of choice has classically been arthrodesis.^{14,19,22} Once the destructive phase has radiologically ceased and bone reconstruction has started, then surgical intervention can be considered.¹⁴

Arthrodesis is commonly achieved using an

intramedullary nail.^{19,23} The patella, along with the entire synovium is usually excised. Any remaining synovium may fibrose and act as a vascular barrier, resulting in shunting of blood around the fusion site and hence preventing fusion. Careful removal of cartilage and sclerotic bone follows. A rod is then inserted with the bony edges apposed at 10-20 degrees. The limb is immobilized using a long leg cast in neutral rotation. Post operative orders includes non-weight bearing status until the knee is clinically and radiologically fused.¹⁹ A study of 10 patients treated with fusion resulted in successful fusion in the 9 patients who were followed up. Three patients developed an infection, which cleared by time passing. No nail was reported as broken during the follow-up period ranging from one to 23 years.¹⁹

Arthroplasty of the Charcot knee

Population

Arthroplasty of a Charcot knee is challenging at best, and some consider the diagnosis as an absolute contraindication to arthroplasty.²⁶⁻²⁸ If arthroplasty is decided as the management pathway of choice, then some patient considerations must be made prior to surgery.

As with any joint, the degree of joint destruction determines the difficulty of the operation and the success chance.²³ Those with milder deformities have generally fared much better with fewer complications.^{22,29}

The phase of the disease is also an important factor to consider. The disease should have completely passed its destructive phase before surgical intervention can be considered. This can be determined radiologically using Eichenholtz's stages of disease⁹ with the cessa-



Figure 1. Charcot knee anteroposterior view x-ray.



Figure 2. Charcot knee lateral view x-ray.

tion of destruction indicated by the final radiological stage of reconstruction.²⁹ Arthroplasties performed before this stage risk complications of dislocation of prosthesis and implant fractures.³⁰ Arthroplasty should only be considered in patients showing radiological evidence of bony reconstruction.^{14,18}

The final consideration is the underlying cause of the disease. Traditionally, the population most affected with the Charcot knee were sufferers of syphilis. This subset of patient has the risk of deteriorating and developing ataxia. This would have a detrimental effect on any performed arthroplasty, due to uneven and abnormal stress exerted on the joint.^{30,31} With the modern patient demographic suffering predominantly from diabetes mellitus, it is difficult to say whether they will respond in the same way as those suffering from syphilis.³²

If all the considerations mentioned above are taken into account, then arthroplasty can be considered a suitable option for symptomatic neuropathic knee.^{12,13,22}

Although a few case series do report good results from arthroplasty, most case series, unfortunately, do not indicate which patients had been excluded from joint arthroplasty. It is therefore difficult to ascertain the risk of offering knee arthroplasty to all patients (Table 1).¹²

Benefits

The benefits of arthroplasty for the sufferer of a neuropathic knee are many.

Arthroplasty has been very successful in the treatment of pain relief. In one study, arthroplasty resulted in 92% of patients having no or only mild pain, compared to 15% pre-operatively. The number of those with severe pain dropped from 10 knees to 1 post-operatively.¹²

In terms of functional outcome, all reports of functional scores have significantly improved with arthroplasty.^{12,18,22} These include average post-operative score gains of 42,²² 39.5,¹⁸ and 40¹³ points. The change in range of motion was not consistent amongst case series. In some it increased,¹² in others it decreased.¹⁸ But with both series, pre-operative range of motion was not noted as a significant problem. In a few patients (three knees), hyperextension of the knee was noted post-operatively, but no recurratum was evident at final follow-up.¹²

Alignment is an import factor for increased longevity of prosthesis and prevention of instability³³ (Figure 3). Arthroplasty is capable of improving alignment in neuropathic knees. On average alignment improved in all case series. The preoperative alignment averaged at 9.18 degrees varus in one series, and this improved to 6.18 degrees valgus post arthroplasty.¹² In other series, the alignment improved to 7.4 degrees of valgus.¹⁸ In most cases the patella was located centrally.^{12,18} Other benefits of arthroplasty compared with different forms of management include decreased need for rest and non-weightbearing orders, decreased bone loss and ability to convert to arthrodesis if

arthroplasty fails, given an uncemented component is used.¹³ In functional terms, the above translates to multiple cases whereby patients, who had previously been disabled to such an extent that they were unable to walk, were then able to do so post-operatively, although still requiring support.¹⁸

Complications

Arthroplasty performed on a neuropathic knee is at greater risk of complications.^{12,14} This increased risk is a combination of a higher incidence of the common complications of knee arthroplasty, with an added subset of complications that are specific to this group.

A review of the available literature reveals that the total complication rate is approximately 50% (35 complications on 72 knees).^{12,14,18,22,23} This is remarkably higher than that of arthroplasty performed for OA.

The list of complications included 5 knees (7%) suffering dislocations. Patellar dislocations were treated with a lateral release and those suffering tibiofemoral dislocations (3 knees) were treated successfully with six months of bracing.¹⁸ Five knees suffered periprosthetic fractures (7%), requiring open reduction and internal fixation. Four knees were showing signs of loosening and instability. The rate of deep venous thrombosis (DVT) was also remarkably high with 6 knees (8%) suffering DVTs. Other rarer but serious complications included disruption of the quadriceps

Table 1. Neuropathic arthropathy of the knee treated with arthroplasty.

Study	Year	No. of knees	Underlying cause	Management	Prosthesis	Complications	Outcome
Vince ²³	2005	1	Syphilis	Arthroplasty	Constrained	Dislocation, infection (MRSA)	Prosthesis removed
Parvizi ¹²	2003	40	Familial sensorimotor deficit (16), diabetes (DM) (7), syphilis (4), lacunar infarct (1), syringomyelia (1), idiopathic (11)	Arthroplasty	Longstem (27), Rotating hinge (5), Cruciate condylar (8)	Avulsion of tibial tubercle, patellar tendon rupture, loosening (1), 6 revisions (aseptic loosening x2, (3 and 7 years post), instability x1 (1 year post), infection x1, arthrodesis for periprosthetic fracture x1, periprosthetic fracture x another 1), MCL avulsion (x2), symptomatic instability (x3), haematoma x2, superficial infection x1, DVT x1	Thirty-four (85%) Free of revision at 8 years, Thirty-three (82.5%) free of mechanical failure
Kim ¹⁸	2002	19	Syphilis	Arthroplasty	Hinged (1), semiconstrained (1), condylar, constrained (17)	Loosening (1), Dislocation (4), Peri-prosthetic fracture (3) Rupture of the quadriceps tendon (1)	Only 53% were satisfactory at a mean follow-up of 5 years. Two awaiting arthrodesis
Fullerton ¹⁴	1997	2	Diabetes mellitus	Arthrodesis (1), Arthroplasty (1)	Highly constrained rotating hinge	Haematoma (1)	Satisfactory
Chong ¹³	1995	1	Hereditary cerebral and cerebellar atrophy	Arthroplasty	Unknown	Nil	Satisfactory at 2.5 years
Soudry ²²	1986	9		Arthroplasty	PS condylar (2), Custom prosthesis (7)	DVT (5)	Satisfactory at 3 years

mechanism in 3 knees, and deep infection in 2 knees. Disruption of the quadriceps mechanism could be attributed to the difficulty in exposure of these severely deformed knees and was treated with intraoperative repair, required immobilization post-operatively.¹²

The above mentioned complications resulted in 11 (15%) knees requiring subsequent surgery. It is a widely held belief that neuropathic arthropathy secondary to syphilis will result in a much higher complication rate.^{12,23} Apart from intraoperative and short-term complications, patients with syphilis may deteriorate and develop ataxia, which could adversely affect the replaced joint.¹² A comparison of the two largest studies of neuropathic arthropathy treated with arthroplasty reveals that the syphilis cohort did seem to suffer a larger proportion of complications. This increase was moderately large but not catastrophically so. In Kim's¹⁸ cohort of 19 neuropathic knees with syphilis as the underlying cause, 8 serious complications were noted; while in Soudry's²² series of 40 patients, of which only 4 suffered from syphilis, 12 serious complications were noted.^{12,18} In terms of satisfactory long-term outcome, 82.5% of Soudry's cohort was free from mechanical failure at 8 years, while only 53% of Kim's syphilis afflicted knees had a satisfactory outcome at 5 years.

Technique

To minimize complications for this high-risk group of patients, certain fundamental techniques have been mentioned in the literature.

Before arthroplasty is even considered in a patient with neuropathic arthropathy, it is important to council the patient first. The patient must be aware of the risks involved in such a procedure and the high possibility of failure and its implications. The patient should understand that arthrodesis and even amputation are possible outcomes if arthroplasty is attempted.¹³ Only then should arthroplasty be considered. Prior to the operation, it is recommended antibiotics and anti-thromboembolics to be used routinely in these patients.¹²

A few key intra-operative steps may increase the likelihood of long-term success. Charcot knees are characterized by their severe deformity, bone loss and consequent ligamentous instability. Apart from the routine steps of arthroplasty, realigning the knee, fixing the bony defects and meticulous ligament balancing are essential. Bony defects and the extensive bone fragmentation associated with a Charcot knee are present in approximately 90% of cases.¹² In approximately half of the patients with neuropathic arthropathy, such defects were greater than 10 mm in the lateral tibial plateau post medial plateau resection.¹⁸ These bony defects need to be filled and/or reinforced. This can be achieved through bone



Figure 3. Long leg standing x-rays showing significant malalignment.

grafting, either autologous or synthetic^{12-14,18} or through custom augments.^{12,14,22} Autologous bone graft can be acquired from the excised femoral condyle or tibial plateau.¹⁸

Realignment of the joint will require ligament balancing and probable lateral release. In some cases the requirements of balancing have been so great that neurolysis of the popliteal nerve could not be avoided. Resection of the iliotibial band, lateral ligament, lateral gastrocnemius and biceps tendon may also be needed.¹³ The quadriceps mechanism is almost always malaligned and requires a lateral release to correct.^{12-14,18} Occasionally, tibial tubercle transfer is warranted.¹² Quadriceps mechanism rupture is a noted complication of this patient cohort due to difficulty in exposure.¹² Finally a complete synovectomy is recommended.^{13,14,18} The synovium is hypothesized to interfere with normal bone metabolism and



Figure 4. Long-stem prosthesis anteroposterior view x-ray.



Figure 5. Long-stem prosthesis lateral view x-ray.

increase the likelihood of surgical intervention.²² Post-operative orders are dependent on intraoperative findings. The majority of knees will require bracing and restricted weightbearing for a period of time after the operation.¹² This is to prevent any damage to the bony repair and ligament balancing. Post-operative ligament laxity may likewise require bracing.^{12,13} Like all knee replacements, range of movement exercises should begin early.¹²

Prosthesis

The choice of prosthesis can be difficult in arthroplasty of the neuropathic knee. Like all arthroplasty, the choice of prosthesis is usually dictated by the level of joint deformity. With the neuropathic joint, this level of deformity can be substantial.

Most would agree that a condylar type prosthesis is the most appropriate choice for most knees.^{12,18,22} Examination of previous studies shows that condylar type prostheses have fewer complications compared with other prostheses.^{12,34} However, bony destruction can be extensive in a neuropathic knee; the surgeon should therefore always consider whether a constrained or hinged prosthesis is to be used. Parvizi¹² warns that a less constrained device may lead to symptomatic instability, requiring revision surgery. This author cites 2 cases in his series where the patient suffered instability and required a revision to a more constrained prosthesis. He also recommends that surgeons have a low tolerance for using long-stem prosthesis (Figures 4 and 5).

On the other hand, Kim¹⁸ warns against using highly constrained prostheses. He argues that the increased stress to the cement–bone interface will eventuate in aseptic loosening. Furthermore, the considerable bony excision required for constrained prostheses makes a revision difficult. One patient treated with a hinged prosthesis in his series is reported as suffering a peri-prosthetic fracture and subsequent aseptic loosening. He recommends that hinged prostheses should be avoided if possible.

Custom augmentation of prostheses is also common;^{14,22} it is usually required to repair severe bony defects. The patella is also commonly replaced.^{12,13}

Conclusions

In conclusion, although technically demanding and high risk, arthroplasty on a symptomatic Charcot knee can be beneficial for the patient. With the growing number of diabetic patients in the population, the presentation of symptomatic neuropathic arthropathy to the orthopaedic surgeon is likely to become more frequent. Conservative management should be trialled

before surgery is considered. The surgeon must be careful that the destructive phase of the disease has ceased before surgery is attempted, and the patient must fully be aware of the risks of failure. Meticulous attention should be directed at alignment and ligament balancing. A Charcot joint should not be considered an absolute contraindication to arthroplasty.

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