Prevalence of Nerve Injuries in Lower Limb following Total Joint Replacement Surgery, and Management

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Abstract

Nerve injuries during lower limb joint replacement are uncommon but serious complications. Review of this condition is sparse in literature. The present review aims to collate the current literature and provide an overview of the subject. Subclinical cases are quite common and preoperative counseling would be helpful. In case the nerve injury occurs, assurance and counselling helps. Since, the prognosis is not uniform and depends on multiple factors, it is best to try and avoid these iatrogenic injuries. A good surgeon knows how to manage his complications, but the best surgeon knows how to avoid them!

Keywords: Nerve injuries, knee arthroplasty, hip arthroplasty, iatrogenic

1. Introduction

Nerve palsy is a rare but devastating complication of Total Hip and Knee replacement. It causes permanent functional impairment and disability[1,2]. Common peroneal division of sciatic nerve is most commonly injured during Total Joint Replacement (TJR). Its prevalence during Total Hip Replacement (THR) and Total Knee Replacement (TKR) ranges from 0.3% to 4.0% [3-14]. Prevalence of nerve palsy after THR is documented in Table 1. Subclinical palsies are unrecognized and clinical diagnosis alone underestimates the presence of nerve injury. Prevalence of subclinical Common Peroneal Nerve (CPN) injury following THR is >70% on electromyography (EMG) studies [15,16]. Although occurrence of Sciatic Nerve palsies is less common, the increase in demand for THR and higher incidence following revision THR further enhances the problem[4,6,17-19]. Schmalzried et al reported that 81% patients with either Sciatic or Femoral nerve palsy had a persistent neurological deficit at two years[17].

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2.Etiology

Nerve palsy following TJR has multifactorial etiology viz. stretching during exposure, inappropriate retractor placement, compression by Gluteus Maximus sling, aberrant course of Sciatic Nerve with respect to Piriformis, haematoma and direct mechanical damage among many others. Tissues are stretched during exposure and lower limb is manipulated which places tension upon the nerves in TJR. Intraneural pressures in the Sciatic Nerve are affected by the position of the hip and knee during surgery. Also, there is increase in tensile strain of the sciatic nerve by 26% during flexion of hip which leads to a significant increase in intraneural pressure[20,21]. Further, inappropriately placed retractors around acetabulum may impinge upon the Femoral Nerve anteriorly and Sciatic Nerve posteriorly. Femoral elevator retractor used during femoral preparation may cause compressive force on the posterior soft tissues and cause of Sciatic Nerve palsy (SNP)[22]. Gluteus maximus femoral insertion (gluteal sling) can compress the sciatic nerve during femoral preparation i.e. 90° of internal rotation. In a series of patients with post-operative nerve palsy, MRI revealed edema within the sciatic nerve at the level of the gluteal sling [23]. In a cadaveric study, 20% of specimens had anatomical variants in which the sciatic nerve penetrated the Piriformis muscle. In a posterior approach to hip when the Piriformis tendon is released, it retracts and can pull the nerve [24]. Also, haematoma at the surgical site postoperatively can compress nerves, leading to neuropraxia [17,21,25,26]. Extruded methyl methacrylate can cause nerve palsy due to either thermal injury or mechanical injury[16,25]. Laceration from a screw used for fixation of acetabular component or delayed neuropathy due to impingement by screw can lead to SNP [21,27]. SNP is also reported due to suturing of sciatic nerve along with posterior capsule in THR [28]. Rare case of Internal Iliac artery aneurysm compressing on sciatic notch leading to SNP has been reported in a case report [29].

3. Risk factors for nerve palsy during TKR

CPN is the most commonly injured nerve during TKR. Others nerve palsies like Infrapatellar branch of Saphenous Nerve and Femoral Nerve (due to tourniquet palsy) are also reported. Valgus and flexion deformity correction during TKR has been considered most important risk factor.

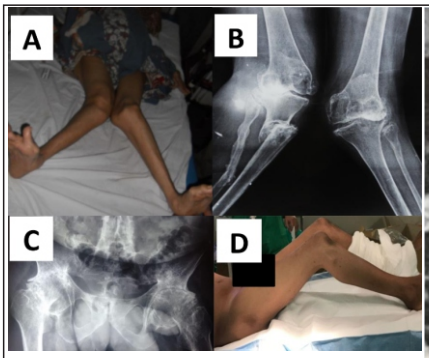

Figure 1: Showing severe deformity of knee (A, B) and hip (C, D). Careful manipulation in such casesis necessaryduring surgeryto avoid stretching of nerves.

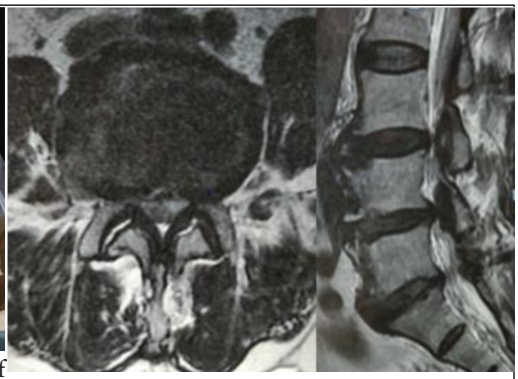

Figure 2: MRI image at L4-L5 level showing severe canal stenosis. Inserting epidural catheter into previously narrowed canal can further compromise nerve roots.

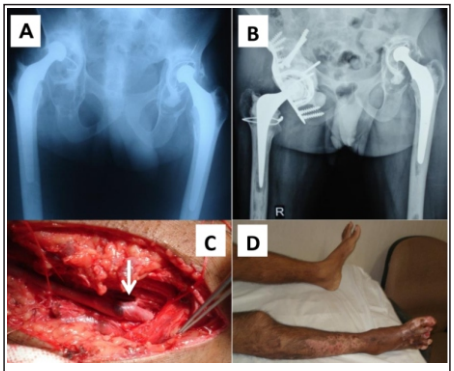

Figure 3: Revision THR with neurovascular complications due to excessive lengthening (A) Preoperative X-ray (B) Postoperative X-ray with Burch Schneider Cage and excessive lengthening (C) Thrombosis in Femoral Artery (white arrow) (D) Affected lower limb with pan-neuritis and ischemic changes.

3.1 Valgus knee

The mechanism for nerve injury is direct stretching of the nerve or perineural soft tissues, which occurs during the TKR of a severely deformed knee[30-32]. (Figure 1) A range from 15^0 or higher valgus angulations are defined as valgus by few studies. In these studies, the average preoperative valgus angulations in patients having Common Peroneal Nerve palsy (CPNP) ranged from 18^0 to 23.3^0 and the average flexion deformity ranged from 15.5^0 to 22^0. Few authors have reported that correction of severe valgus deformity and/or flexion contracture in TKR as a significant risk factor of CPNP[7-10,33,34]. Fixed/Rigid valgus are more prone to develop CPNP after correction than a correctible valgus. Idusuyi et al reported that only valgus deformity and not flexion contracture was significantly associated with Peroneal Nerve palsy[7]. In contrast, few studies did not find preoperative valgus deformity and/or flexion deformity as significant risk factors causing CPNP[13,35,36]. A cadaver study reported that CPN is at risk of direct injury while performing the inside-out release of the posterolateral capsule (danger zone) and not during pie crusting of the ITB (safe zone)[37].

3.2 Rheumatoid Arthritis (R.A)

CPNP after TKR is significantly associated with R.A. [13,38]. While retrospective studies have reported low incidence of 0.3% to 1.3%, Knutson et al reported incidence of 16.7% diagnosed via EMG. Schinsky et al reported R.A as a significant risk factor and not preoperative valgus/ flexion deformity while Rose et al did not find R.A as a significant risk factor[9,13].

3.3 Tourniquet

Tourniquet is useful in achieving haemostasis in TKR and minimizing operative time, but has its known complications. They are postoperative swelling, neurovascular injury, wound hematoma, cardiovascular complications and EMG-evident nerve injury[39-42]. Ischemia and mechanical deformation of nerve occurs in the area directly underneath the cuff [43-45]. EMG changes are seen in 71% to 75% patients. Horlocker et al reported that along with a preoperative valgus deformity of 10^0 or higher, preexisting neuropathy and postoperative bleeding, total tourniquet time more than 120 minutes is a significant risk factor for development of CPNP. The concept of safe duration of tourniquet time is controversial as values ranging from one to three hours are reported in few studies [44,46-48]. Tourniquet pressures of 240 mmHg for up to 80 minutes are safe with respect to the risk of nerve injury in patients undergoing TKR [49]. In contrast, few studies reported that pressure and duration of tourniquet were not significant risk factors [7,9,38]. Tourniquet use limited to two hours or less is considered safe and is followed until a randomized trial defines optimal time[43,46,50].

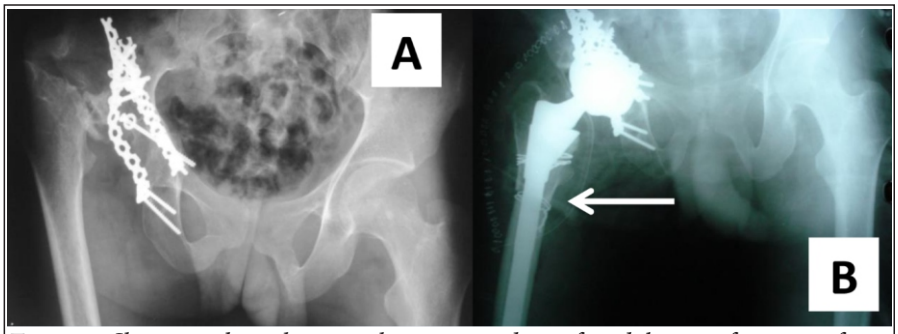

Figure 4: Showing sub trochanteric shortening and use of modular stem for a case of post acetabular fracture with superior migration of femur to avoid sciatic nerve palsy. White arrow showing site of osteotomy.

3.4 Constrictive dressing

Superficial anatomical location of CPN makes it vulnerable to compression injury. Application of constrictive dressing may lead to development of CPNP after TKR[9,11,12,36]. Formation of hematoma postoperatively at the wound site causing compression of nerve is a cause of the CPNP[7,8,38]. While most studies reported it minor factor, Horlocker et al reported it as significant risk factor for CPNP[10].

3.5 Preexisting Neuropathy

Centrally (i.e. lumbar canal stenosis, radiculopathy) (Figure 2) or peripherally,

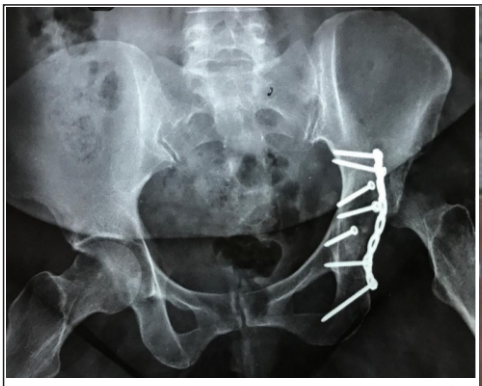

Figure 5: X-ray pelvis showing plating done for posterior column. Retraction needed for plating in supra acetabular region may lead to SGN palsy due to stretching of SGN. It is necessary to do preoperative EMG to rule out SGN palsy.

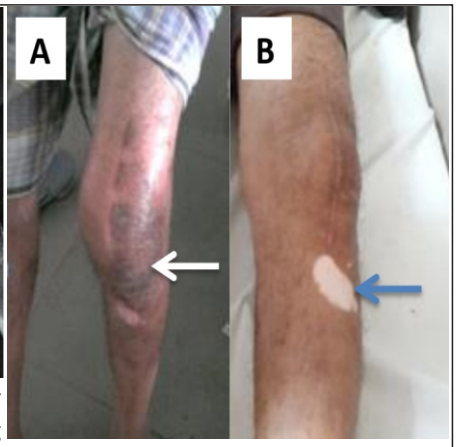

Figure 6: Showing neurodermatitis patch which is due to injury to infrapatellar branch of saphenous nerve.

occurring previous neuropathy has been significantly associated with the development of CPNP. The proximal lesion makes the CPNP more vulnerable to a distal injury by decreasing axoplasmic flow. This concept is known as double crush syndrome[51]. Diabetes mellitus is not a risk factor associated with CPNP after TKR although it is associated with a progressive peripheral neuropathy[7,8,10,13].

3.6 Postoperative Epidural Analgesia

CPNP is attributed to the resting of the fibular head against the bed rail leading to pressure on CPN. Patients are unaware because of epidural analgesia [26]. Patients tolerate excessive extensions on continuous passive motion or constrictive dressing which predisposes to CPNP. Epidural analgesia also prevents early diagnosis of CPNP [7,10]. CPNP after TKR is significantly associated with epidural analgesia. Only 1 of 32 palsies in a prospective study was discovered on the day of surgery[7]. Similarly, in another study most of palsies were diagnosed on day two[8]. In a case of lumbar canal stenosis even the epidural catheter itself may further compromise the canal space & give rise to nerve deficit post-operatively. In contrast, Horlocker et al in his study did not find the significant relationship between epidural analgesia and CPNP [10].

3.7 Miscellaneous

Young patients are at higher risk of nerve palsy[10,12,52]. Shetty et al reported no significant difference with age in a case control study[53]. Higher BMI (34.5 kg/m2) is reported as significant risk factor for CPNP. It leads to more difficult exposure thus stretching the nerve. Also obese patients tend to put their hip in more external rotation while lying down causing compression of CPN at the level of the fibular head[12]. Contrastingly, few authors reported that higher BMI is not significantly related to CPNP[10,53]. Women have higher risk of CPNP due to reduced muscle bulk, different vascular anatomy and shorter limbs [53].

Table 1 : Prevalence of nerve palsy following THR in multiple retrospective studies

Authors and year	THRs	Incidence of overall palsy (%)	Primary THR	Incidence of palsy in Primary THR (%)	Incidence of palsy in Revision THR (%)
Schmalzried et al (1991)	2355	1.7	1661	1.3 (DDH – 5.2)	3.2
Johanson et al (1983)	5667	0.6	-	-	-
Park et al (2013)	9570	0.32 (CPN)	-	-	-
Farrell et a (2005)	27004	0.17	27004	0.17	-
Navarro et al (1995)	1000	0.8 (posterior approach-0.6) (LTT approach - 1.0)	630	0.5	1.4

Table 2 : Recovery rate and mean follow up after nerve palsy in multiple studies.

Author	Recovery rate	Mean follow up
Idusuyi et al	50%	3.9 years
Asp and Rand et al	50%	5.1 years
Farrell et al	38%	21 months
Pekkarinen et al	33%	5 years
Zappe et al	25%	5 years
Edwards et al	16%	1 year

4. Risk factors for nerve palsy during THR

SNP is the most common neurological injury following THR accounting for more than 90%. Other nerves like Femoral Nerve, Superior Gluteal Nerve, Lateral Cutaneous Femoral Nerve (LCFN), Inferior Gluteal Nerve, Obturator nerve are also involved. The incidence for SNP ranges from 0.6 to 3.8 % [54]. In 50% cases the exact etiology of nerve damage is unknown[17,19]. Nerves have limited resistance to stretching and thus, limb lengthening is considered the most significant causative factor[6,20,21,54].

4.1 Lengthening

Intraoperative lengthening during THR is major cause for SNP. Farrell et al reported lengthening as a significant risk factor with average lengthening being 1.7 cm in patients who sustained SNP[3]. Edwards et al reported limb lengthening between 1.9-3.7 cm leads to overstretching of peroneal division and warned about lengthening greater than 4 cm [6]. If lengthening is 4.0 to 5.1 cm, the whole sciatic nerve is damaged and recovery is poor [18]. Absolute amount of limb lengthening which can be tolerated varies individually and potential for nerve recovery should be evaluated individually[55]. (Figure 3) Some surgical steps such as releasing femoral insertion of gluteus maximus, avoiding prolonged femoral internal rotation, feeling the tautness of nerve intraoperatively and Somatosensory Evoked Potential helps to avoid Sciatic Nerve injury due to lengthening. Sub trochanteric shortening can be considered if lengthening

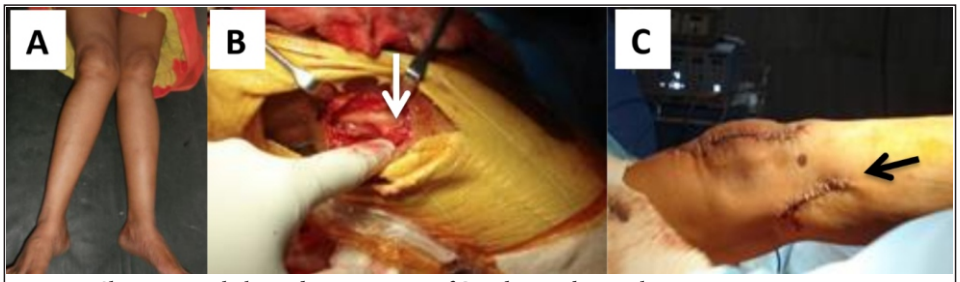

Figure 7: Showing prophylactic decompression of CPN by Krackow technique
(A) Valgus Knee planned for TKR (B) Prophylactic decompression of CPN done (White arrow – CPN)
(C) Oblique incision below the head of fibula (Black arrow)

is excessive[56]. (Figure 4) Use of a uncemented femoral implant results in axial impaction into the femoral canal leading to stretching of nerves repeatedly[3].

4.2 Developmental dysplasia of hip (DDH)

It is reported that preoperative diagnoses of DDH or post-traumatic arthritis are risk factors for nerve palsy after primary THR. Altered anatomy of dysplastic patients leads to aberrant course of the sciatic nerve, making it more susceptible to injury. Scar tissue or adhesions may tether the sciatic nerve in the post-traumatic conditions[3].

4.3 BMI and Sex

Higher incidence of SNP is reported in female patients. It is due to gynecoid pelvis, reduced hip offset and lower muscle mass to protect against nerve compression [6,16,17,19]. A meta-analysis reported that females had 1.5% risk of SNP compared to 0.77% for males[57]. Higher BMI is associated with a greater risk of nerve palsy.

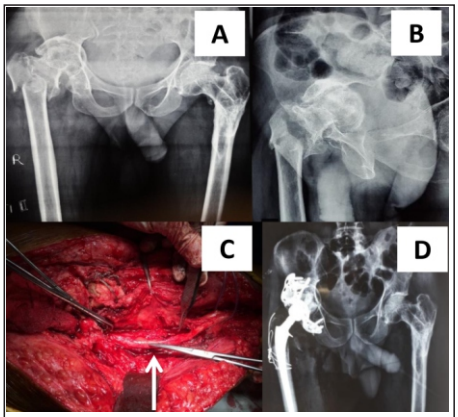

Figure 8: Image showing sciatic neurolysis after post-operative sciatic nerve palsy
(A) and (B) showing arthritic deformed right hip joint with protrusio acetabuli and intertrochanteric fracture
(C) Sciatic neurolysis done for postoperative SNP
(D) Postoperative X-ray showing excessive lengthening and hardware prominence.

One prospective and one retrospective study, both didn't find high BMI as a risk factor [3,12].

4.4 Miscellaneous

Fibrotic ankylosis after joint sepsis has highest risk of nerve injury, followed by DDH. Risk increases in revisions compared with primary THR. Anterior minimally invasive surgery (AIMS) for THR is significantly associated with nerve injury (Sciatic nerve, Femoral nerve, LCFN). It could be due to direct nerve injury by retractors, hyperextension, excessive external rotation of the leg and traction[58]. History of spine disease is reported as the strongest risk factor for nerve palsy. Medical comorbidities such as diabetes or obesity have no significant association with nerve palsy[59].

5. Clinical picture of specific nerve palsies

5.1 Sciatic nerve

Etiology of iatrogenic SNP is frequently unknown and is not identified in approximately 50% of the patients[2,6,12]. Traction, compression from hematoma and thermal burns from extraneous cement are the most commonly reported causes[54]. Common peroneal component is most commonly involved. It is easily predisposed to permanent neuronal damage than tibial component because it has few large funiculi with sparse connective tissue that restricts its elongation[8,10]. Also it is more lateral, more superficial and tethered at two sites (sciatic notch and fibular head). Being more lateral it is placed closer to the surgical field during the posterior approach [20,21,60]. Foot drop is the typical presentation. Patient is unable to clear toes during swing phase leading to fall. To prevent contractures of the Achilles tendon and to assist walking, ankle-foot-orthosis (AFO) can be used[54].

5.2 Femoral nerve

Femoral nerve palsy after THR is under-reported, with the loss of quadriceps function often overlooked[61]. It occurs most commonly due to compression by retractors placed against anterior acetabular wall. There is weakness in the quadriceps and iliacus component of iliopsoas with sensory loss over the anterior thigh. Patients have difficulty with walking and stair climbing[54]. Simmons et al reported 2.3% femoral neuropathy rate with all THRs done by anterolateral approach and most common fault was placement of acetabular retractors[62].

5.3 Superior Gluteal Nerve (SGN)

It arises from the L4–S1 nerve roots and exits through the sciatic notch to supply the Gluteus Medius, Gluteus Minimus and Tensor Fascia Lata (TFL). Khan et al reported 6.8% incidence of SGN injury with a direct lateral approach. It is most commonly injured during the anterolateral (Watson-Jones), lateral (Hardinge), or trans-trochanteric approach when the 3–5cm "safe area" proximal to the tip of greater trochanter is violated [52,63]. Wang et al reported distance between the superior acetabular rim to the SGN was 2.23 ± 0.28cm. SGN palsy leads to hip pain and limping following THR. The most common cause is inadvertent damage to the SGN intra-operatively. Abductor weakness after THR is higher in women and significantly higher in elderly women[64,65]. Retraction in the supra acetabular region can lead to over stretching of SGN, which can be avoided by performing trochanteric osteotomy. (Figure 5)

5.4 Lateral cutaneous femoral nerve (LCFN)

It has wide variation in its anatomy, which is considered as the cause of its common involvement with anterior approaches to hip. It is variable at the level of the Anterosuperior Iliac Spine and the Inguinal ligament[66,67]. As it comes through the interval between Sartorius and the TFL it is vulnerable distally[68]. Nerve can be injured through various mechanisms including stretching, compression,

laceration and involvement in scar formation[66,67]. Hip resurfacing is a risk factor for LFCN neuropraxia. Longer incision over branches of the LFCN and increased retraction needed for femoral head preparation are the causes. Contralateral LCFN may also get compressed due to lateral position given during THR leading to meralgia paresthetica. Goulding et al reported 88% patients having neuropraxia at first follow up after direct anterior approach for THR [69]. Modifications advised to avoid injury to LCFN include lateralizing the skin incision, sub fascial dissection, avoiding severe retraction of the Rectus Femoris and dissection of medial subcutaneous fat pad[66,68,70].

5.5 Inferior Gluteal Nerve (IGN)

IGN enters the deep surface of gluteus maximus inferiorly. To avoid injury to IGN, it is advised that five centimeters or less of muscle-splitting incision should be made from the tip of the Greater Trochanter across Gluteus Maximus. The lower part of the incision is extended distally creating a modified posterior flap thus, allowing protection of the IGN [71]. Damage to the branches given off to innervate the superior portion of Gluteus Maximus can occur during the classical posterior approach to the hip leading to weakness of hip extension. Minimally-invasive posterior approaches for THR should consider this surgical anatomy[72].

5.6 Miscellaneous

A prospective study reported 84% of the patients having signs of injury to the Infra Patellar branch of the Saphenous Nerve after TKR[73]. (Figure 6) Obturator nerve is at risk if violation of the floor of the acetabulum occurs and frequently undiagnosed. Its palsy leads to vague patterns of pain following THR. Extraneous cement should be excluded in such cases. Ulnar nerve may get compressed during the lateral position given for THR due to poor padding[74].

6. Management

6.1 Diagnosis

It depends on factors such as symptoms, surgical approach, degree of preoperative deformity and retractor placement. In certain cases like operative site haematoma, MRI is useful in identifying the source of compression. Preoperative EMG is used as screening tool for preexisting neuropathies and postoperative EMG for patients with neurologic symptoms[13]. Acute EMG should be avoided. EMG should be done if nerve injury in a patient shows no recovery after eight to twelve weeks. It helps in confirming the level of injury and discussing prognosis[3]. MRI spine is performed to rule out epidural or spinal haematoma if a neuraxial anesthetic was used[54]. Magnetic Resonance Neurography (MRN) allows visualization of the fascicular distribution of the nerve, which helps in determining potential for early surgical intervention. It is relatively insensitive to susceptibility artifacts (siMRN susceptibility insensitive MRN)[75]. Thus neurotmesis and neuromas in continuity are seen which helps to diagnose Sunderland fourth and fifth degree nerve injuries[76].

6.2 Treatment

An informed dialogue regarding the etiology and prognosis should take place between the surgeon and patient. The patient needs to be reassured about the prognosis. Limb should be positioned to decrease stretch on the affected nerve. In case of Sciatic Nerve involvement, hip should be extended and the knee flexed and in case of Femoral Nerve palsy, hip should be flexed. If CPNP is diagnosed in postoperative period, any constrictive dressing should be removed to permit 30 to 40 degrees of flexion of the knee. If the cause of nerve palsy is not known, supportive measures should be started. It includes physiotherapy to maintain range of movement, bracing during ambulation and splinting[54]. A study reported that conservative treatment for nerve palsy led to partial recovery in all cases, of which 68% showed complete recovery at 18 months[13]. Similarly, Idusuyi et al reported resolution of symptoms in 76% of patients with partial palsy and 20% patients with complete palsy at mean follow-up of 47 months[7]. Operative treatment is not advised for all cases of nerve palsy. With no evidence of a compressive hematoma, observation is advised as they resolve with time[9,77]. Lumbar canal stenosis must be ruled in old patients as a cause of foot drop after THR before exploration. A wide variability in recovery between patients with similar lesions is reported, and the disparity between function and neurological deficit is widest for Sciatic Nerve[78]. Decision to explore a nerve lesion in acute palsy is hard. Any mechanical damage of nerve demands prompt exploration. Even an evidence of post-operative neural haematoma warrants urgent decompression. Presence of pain in the area of distribution of the affected nerve is the most accurate diagnostic feature. It suggests that there is an ongoing insult to the nerve. Thus, all nerve palsies associated with pain should be explored. The advantage of exploration must be weighed against the risks of reoperation in an elderly patient and the risks of infection during the second procedure[61]. If the limb is lengthened excessively, then a shortening can be considered during repeat surgery[54]. Krackow's technique is surgical release of peroneal nerve at two specific sites, in which fascial band over the nerve and the intermuscular septum is sectioned. After this procedure, patients can resume physical therapy regimen[79]. (Figure 7) He reported that 80% patients had complete resolution of symptoms with CPNP after TKR. Also, if any patient is not showing clinical or EMG improvement after three months, exploration of the nerve is mandatory[80]. Similarly, Mont et al found subjective and functional improvement in 97% patients (mean follow up 36 months) after peroneal nerve release, helping them to discontinue use of an Ankle Foot Orthosis. Sensorimotor recovery deteriorates with time and thus, are advised early decompression as CPNP can result in severe functional disability[81]. Wilson et al reported peroneal nerve decompression at the fibular tunnel postoperatively in cases of SNP, leading to recovery of dorsiflexion strength in 65% of the patients[82]. Patients who underwent interfascicular neurolysis of the sciatic nerve, after having failed conservative management for a minimum of six months reported improved motor and sensory function as well as decreased pain with neurolysis. Hence, treatment should not be delayed for more than 12 months[83]. (Figure 8). Tibialis posterior tendon transfer to cuboid for dynamic foot drop and Lambrinudi arthrodesis for fixed equinocavo varus

deformity can be alternatives.
Spontaneous recovery after SGN Palsy in THR is reported in up to 95% of patients by 24 months. Hence, a 'wait and watch" approach is adopted for SGN palsy. Serial EMG is used for diagnosis and to assess recovery. There is severe abductor dysfunction and an increased risk of chronic instability. It can be managed using constrained liner for acetabular component. Hip stability can also be improved with the utilization of an Achilles allograft as augmentation for deficient abductors[84].

7. Prognosis

Prognosis for peripheral nerve injury depends on factors like nerve injured, type of nerve injury, age and sex of the patient, as well as factors such as diabetes, history of smoking and preexisting neuropathy[85]. Prognosis is not same for all nerves. It is poor for complete initial injury and fair for incomplete initial injury. There is strong association between initial retention of some motor function and normal or nearly normal recovery of function[12,17]. The results of exploration and neurolysis in nerve palsy due to traction are poor[78]. Park et al reported full recovery in 75% of patients with incomplete CPNP and only one patient with complete nerve palsy fully recovered. Recovery of CPNP is possible only if the cause is reversible. However, rate of recovery does not differ between patients with complete and incomplete palsy or between patients with and without motor nerve involvement[12]. In contrast, Fine et al reported that extent of neural damage didn't correlate with the chances of complete recovery[55]. In patients with CPNP, the residual disability caused by direct compression injury is less than that caused by stretch injury[6]. Body mass index (BMI) has more significant correlation than extent of motor nerve involvement, with the chance of full recovery[12].
Femoral Nerve palsy has a better outcome than SNP. It is believed to be due to the closer proximity to the spinal cord compared with the Sciatic Nerve. Hence, it requires less regeneration of nerve fibers for recovery[57].

8. Precautions

8.1 THR

(a) Complete awareness of the anatomy.
(b) Sciatic nerve should be identified during revision THR.
(c) Limb should be supported throughout the operation with care.
(d) Careful use of retractors, especially around the anterior wall of the acetabulum.
(e) Excessive traction should be avoided during dislocation of the hip and horizontal as well as vertical offset should be restored.
(f) Neuromonitoring of the sciatic nerve in high risk cases.

8.2 TKR

(a) Identifying high risk cases for nerve injury.
(b) Preoperative counseling of patients for nerve injury
(c) Selective use of tourniquet only for cementing and maintaining alertness with regards to tourniquet time and pressure.
(d) Prophylactic decompression of CPN in valgus and flexion deformity.

9. Medicolegal Aspects of Iatrogenic Nerve Injury

Surgeons must be aware of the potential medico legal consequences in addition to the clinical implications of iatrogenic nerve injuries. Medico legal cases regarding peripheral nerve injury have found surgical error, inadequate or absent informed consent, and failure to immediately diagnose the injury as factors prompting litigation. The literature is indeterminate whether intraoperative nerve monitoring is beneficial from a medico legal view[86,87]. A study was conducted among American Association of Hip and Knee Surgeons, which found that 78% surgeons were defendants of at least one lawsuit and nerve injury was the most common source of litigation[88].

10. Summary

Nerve palsy though rarely encountered in joint replacement surgeries, is a disabling complication. The rate of subclinical palsies remains high; hence, we recommend a high index of suspicion in identifying at risk cases. Of utmost importance is preoperative counseling for risk of nerve palsy and reassurance if it occurs. Careful manipulation of limb during surgery and post-operative neurological examination is essential. Decision regarding operative management should be based on pain in the distribution of nerve, evidence of haematoma and lack of signs of improvement after three months. Since, the prognosis is not uniform and depends on multiple factors, it is best to try and avoid these iatrogenic injuries. A good surgeon knows how to manage his complications, but the best surgeon knows how to avoid them!

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