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Original Article

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Assessment and Trèatment of Gait in Diabetic Peripheral Neuropathy: A Focused Review of Evidence

Kumar Senthil P.*, Adhikari Prabha, Jeganathan***, Misri Z.K.***, D'Souza Sydney C.******

Abstract

Background: Walking is an integrated function of neurophysiological and musculoskeletal systems which in turn depends upon cardiorespiratory and metabolic systems for energy cost and expenditure. **Objective:** To evaluate the abnormalities of gait in patients with diabetic peripheral neuropathy (DPN) by reviewing studies on assessment and treatment. **Methods:** A systematic review of PubMed was done using search terms of diabetic neuropathy and gait for articles in English with abstracts and independent blinded data extraction and synthesis was performed to identify studies on assessment and treatment. **Results:** Reduced gait speed, reduced double support time, reduced step length, reduced ankle range of motion, with increased ankle invertor-evertor moment; altered plantar pressures with increased load under midfoot compared to rearfoot; earlier muscle activity of soleus, tibialis anterior, vastus medialis and medial hamstrings with delayed muscle activity of vastus lateralis and lateral gastrocnemius; longer loading time with decreased mediolateral and longitudinal center of pressure excursions were reported in gait of individuals with DPN. Gait-related interventions in DPN population studied were physiotherapy including walking prescription, lower extremity strengthening and balance exercises, footwear and insoles, and visual feedback which were shown to improve balance, gait speed, muscle activity and plantar pressures in this population. **Conclusion:** There were alterations in temporal and spatial gait parameters, muscle activation patterns, and loading time responses which is essential for clinicians examining patients with DPN, and interventions such as physiotherapy, footwear and insoles and visual feedback were reported to be useful to improve gait in people with DPN.

Keywords: Gait; Human walking; Bipedal locomotion; Diabetic neuropathy; Functional mobility.

Introduction

Human walking or gait had evolved phylogenetically and ontogenetically from quadupedialism to bipedalism to provide locomotion with advanced adaptive functions to suit the needs of the person, the task and the environment.[1] Walking is an integrated function of neurophysiological and musculoskeletal systems which in turn depends upon cardiorespiratory and metabolic systems for energy cost and expenditure.[2]

The human gait has temporal and spatial parameters measured using distance and time variables respectively, which gets altered in pathological states that affect the sensorimotor function of gait. The dynamics of human gait is well understood for its complexity in its response to stress and evolution[3] and the importance of measuring human gait in different medical conditions cannot be overemphasized.[4]

The role of spine and pelvis,[5] hip and thigh,[6] and knee[7] in the evolution and natural history of human gait is recognized for their relative segmental alignment and their dynamic interactions as a closed kinetic chain.

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The role of visual, vestibular and somatosensory systems in static and dynamic balance as well as during gait is essential in controlling the direction or trajectory of center of gravity in a three-dimensional and multidirectional motion.[8]

Studies on gait had previously focused on disorder-specific deviations,[9] methods or techniques of gait analysis,[10] and direction-specific deviations.[11] Changes in gait parameters such as shorter stride length, reduced walking speed, and altered lower limb and trunk mobility were previously reported in persons with diabetes mellitus (DM)[12] which were influenced by cognition, mood, lower-extremity circulation and sensation, visual impairment, lower-extremity strength, physical activity, and body mass index (BMI).[13]

Although many of above gait deviations and influencing factors play a major role in diabetic patients to have gait abnormalities,[14] peripheral neuropathy might affect any or all of the above mentioned factors thus playing a major role in gait which is not yet clearly understood.[15] Thus there is a need to explore the role of peripheral neuropathy in gait of diabetic individuals and the objective of this study was based upon this need to evaluate the abnormalities of gait in diabetic peripheral neuropathy (DPN) by reviewing the published studies on assessment and treatment.

Methodology

A systematic review using the following search terms was done and entered into PubMed-(diabetes [Title] OR diabetic [Title]) AND (neuropathy [Title] OR neuropathic [Title]) AND (gait [Title] OR walking [Title]) with search filters activated for articles with abstracts and published in English language. The search was performed by two testers independently and mutual consensus method was adopted periodically. Two main themes were selected under assessment and treatment of gait.

Results

A total of 39 studies (2 excluded-1 abstract not available; 1 not on walking) were obtained in our initial search and after excluding inappropriate articles, a final list of 37 studies were included for data extraction and synthesis.

Assessment of gait in DPN

Gait parameters

Roman de Mettelinge *et al*[16] investigated the effect of peripheral neuropathy and cognition on gait performance in 101 older adults (56 diabetics, of which 28 with peripheral neuropathy and 28 without peripheral neuropathy; 45 matched controls). The study found that older adults with diabetes walked slower, took shorter strides during all walking conditions, and showed more gait variability especially during dual task conditions. Also older adults with diabetes showed that participants with impaired cognitive function walked slower, took shorter strides, and had shorter double support time and increased gait variability when compared to participants with intact cognitive function.

Lalli *et al* measured gait parameters in DM patients with and without diabetic peripheral neuropathy (DPN) during flat surface walking using a portable device (GaitMeterTM). DPN-P participants had greater variability of step length and step velocity, except for DM only participants.[17]

Gomes *et al* assessed kinematic and electromyographic data in 46 subjects (healthy and DN) who walked at two cadences (self-selected and 25% higher) and compared them with different phases of gait cycle. DN subjects showed a delayed peak in plantarflexor activity along the whole cycle (irrespective of cadence) compared with healthy subjects. However, during the imposed cadence, DN individuals showed reduced ankle range of motion along the entire cycle compared with the self-selected condition and healthy individuals walking at

both cadences.[18]

Paul *et al* compared temporal and spatial gait parameters of 15 older people with diabetes and no peripheral neuropathy (DM) and 15 people with diabetes and diabetic peripheral neuropathy (DPN) to investigate the effect of a secondary motor or cognitive task on their gait. Subjects underwent four walks: under normal walking conditions (single task); four times while simultaneously undertaking an additional motor task, carrying a tray with cups of water (dual task); and four times whilst undertaking a cognitive dual task, counting backwards in sevens. Subjects with DPN walked more slowly and with smaller steps compared with those with DM. In general, the secondary task had a significant and adverse effect on the gait parameters and this effect was greater for those with DPN in both absolute and relative terms.[19]

Katoulis *et al* investigated the effect of peripheral neuropathy on gait in diabetic patients by performing gait analysis in 20 normal healthy control subjects (NC), 20 non-neuropathic diabetic control subjects (DC), 20 neuropathic diabetic subjects (DN), and 20 neuropathic diabetic subjects with a history of foot ulceration (DNU). Walking speed was significantly slower in the DNU group compared with the two control groups. The maximum knee joint angle was smaller in the sagittal plane for the DNU group compared with the DC group values. The maximum value of the vertical component of ground reaction force (GRF) was found to be higher in the two control groups compared with the DNU group. The maximum value of the anteroposterior forces was also found to be higher in the DC group compared with the DNU group. The maximum frontal plane ankle joint moment was also higher in the DN compared with the NC group.[20]

Mueller *et al* compared (1) the gait characteristics, (2) the plantar-flexor peak torques, and (3) the ankle range of motion of 10 subjects with diabetes mellitus (DM) and peripheral neuropathy with those of 10 age-matched controls (NODM). The DM group subjects showed less ankle mobility, ankle moment, ankle power, velocity, and stride length during walking than the NODM group subjects. A significant decrease in ankle strength and mobility appeared to be the primary factor contributing to the altered walking patterns of the DM group.[21]

Plantar pressures during gait

Sacco *et al* investigated the ankle range of motion during neuropathic gait and its influence on plantar pressure distribution in two phases during stance: at heel-strike and at push-off in 15 DPN patients and 16 healthy adults and found that DPN patients walked using a smaller ankle range of motion in stance phase and smaller ankle flexion at heel-strike. Peak pressure and pressure-time integral values were higher in the diabetic group in the midfoot at push-off phase when compared to heel-strike phase.[22]

Bacarin *et al* investigated plantar pressure variables during gait and compared 20 healthy controls; 17 diabetic neuropathy patients without foot ulcers; and 10 diabetic neuropathy patients with at least one healed foot ulcer within the last year. The study findings showed that a previous history of foot ulcers in DPN subjects influenced plantar pressure distribution, resulting in an increased load under the midfoot and rearfoot and an increase in the variability of plantar pressure during barefoot gait.[23]

Maluf *et al* assessed the relationship between foot pressures measured during level walking and other types of ambulatory activity in 16 subjects with diabetes mellitus (DM) and peripheral neuropathy (PN), and found that peak pressure and PTI during level walking correlated highly with pressures during ramp climbing and turning at all regions examined and with pressures during stair climbing at 1st and 3rd metatarsals. Correlations between pressures during level walking and stair climbing were moderate at the great toe and poor at the heel. With few exceptions, pressures during ramp climbing, stair climbing, and turning were less than or equal

to pressures during level walking.[24]

Patil *et al* introduced new on-line foot pressure parameters, i.e. normalized peak pressure (NPP) and pressure contact ratio (PCR), which include effects of the weight of the subject, velocity of walking and duration of high pressures in any region of the foot, which were calculated on-line (using specially developed software) would help the clinician to quickly determine the heavily loaded foot areas that are potential sites of ulceration in insensitive feet and take the necessary action to prevent further damage to the foot sole.[25]

Muscle activity-kinetics

Akashi *et al* evaluated the EMG of the right vastus lateralis, lateral gastrocnemius and tibialis anterior were studied during the stance phase, and compared them between three groups: a control group (n=16), diabetic neuropathic group (n=19) and diabetic neuropathic group with previous history of plantar ulceration (n=10). The ulcerated group presented a delayed in the time of the lateral gastrocnemius and vastus lateralis peak occurrence in comparison to control's. The vastus lateralis and lateral gastrocnemius delay demonstrated that ulcerated diabetic neuropathic patients have a motor deficit that could compromise their ability to walk, which was partially confirmed by changes on ground reaction forces during the push-off phase.[26]

Kwon *et al* compared muscle activity and joint moments in the lower extremities during walking between subjects with diabetic neuropathy (DN) and control subjects. The study findings demonstrated that subjects with DN had less ankle mobility, slower walking speeds, longer stance phases, and lower peak ankle dorsiflexion, ankle plantar flexion, and knee extension moments than control subjects. Onset times with respect to heel-strike (HS) for the soleus, medial gastrocnemius, and medial hamstring muscles were significantly earlier during the gait cycle (GC) in subjects with DN than in control subjects. The cessation times of soleus, tibialis anterior, vastus medialis, and medial hamstring muscles were significantly prolonged in subjects with DN. Subjects with DN showed more co-contractions of agonist and antagonist muscles at the ankle and knee joints during stance phase compared with control subjects.[27]

Sacco and Amadio evaluated EMG variables during stance phase in self-cadence treadmill walking under biomechanical and somatosensorial considerations in 20 DPN and 20 healthy controls, and found that the somatosensorial responses and pain tolerance threshold in the diabetic neuropathic group were significantly higher and considered far from the normal patterns. The EMG responses of the thigh and leg muscles in the diabetic neuropathic group were delayed if compared to the normal recruitment pattern, especially the tibialis anterior and vastus lateralis.[28]

Plantar loading responses

Giacomozzi *et al* evaluated 21 healthy volunteers (C) and 61 diabetic patients (27 diabetic subjects without neuropathy (D), 19 with neuropathy (DN), and 15 with previous neuropathic ulcer (DPU)) and found that loading time was significantly longer in neuropathic patients than in control subjects. COP excursion along the medio-lateral axis of the foot clearly decreased from C to DPU groups as well as COP excursion along the longitudinal axis for the DPU group only. The decreased medio-lateral and longitudinal COP excursions and corresponding changes of loading times and patterns supported our hypothesis that a change in the walking strategy of diabetic patients with peripheral neuropathy does occur.[29]

Cavanagh *et al* measured the variability of plantar loading during gait and explored the differences between neuropathic and non-neuropathic patients by studying 39 patients (13 non-diabetics, 13 diabetic non-neuropathic, 13 diabetic neuropathic). The study showed that variability was not significantly influenced by the diagnostic group for any shoe condition or for any region of the foot which suggested

that reduced variability in plantar loading is not a factor in the development of plantar lesions in neuropathic patients.[30]

Methods of gait analysis

Meier *et al* investigated goal-oriented gait termination in 15 healthy elderly and 15 elderly type-2 diabetic subjects and found that the diabetic subjects approached the stopping line more slowly than the healthy elderly subjects. They also exhibited a weaker maximal braking force and a prolonged relative time to develop this force. Despite this slower motion, the centre of pressure overshoots were larger in the diabetic subjects than in the healthy elderly.[31]

Gait-related injuries

Cavanagh *et al* studied two groups of patients from the Pittsburgh Epidemiology of Diabetes Complications Study, matched for age and duration of Type 1 diabetes, but with significantly different vibratory sensation thresholds as determined by Vibratron II testing, and found that the neuropathic group had adjusted odds ratios for reported injuries during gait of 15.0 relative to the control group. The neuropathic group also reported significantly lower scores than the control group on perceived safety in unusual conditions.[32]

Relationship of gait parameters with other factors

Lower limb sensorimotor function and gait

Allet *et al* identified whether frontal plane lower limb sensorimotor functions predicted gait speed and efficiency (step-width-to-step-length ratio) on an uneven surface, in 33 subjects; 21 with diabetic distal symmetric peripheral neuropathy. Hip adduction RTD and ankle inversion RTD predicted 54% of gait speed, with the former predicting the majority (44%). Ankle inversion RTD was the only significant predictor of gait efficiency, which accounted for 46% of its variability.[33]

Menz *et al* evaluated acceleration patterns of the head and pelvis when walking to determine the effect of lower-limb sensory loss on walking stability in 30 older people with diabetic peripheral neuropathy (DPN) and in 30 age-matched controls. Participants with DPN had reduced walking speed, cadence, and step length, and less rhythmic acceleration patterns at the head and pelvis compared with controls. These differences were particularly evident when participants walked on the irregular surface.[34]

Dingwell *et al* quantified the sensitivity of the locomotor system to local perturbations that are manifested as natural gait kinematic variability in 14 patients with severe peripheral neuropathy and 12 matched non-diabetic controls, and found that neuropathic patients exhibited slower walking speeds and better local dynamic stability of upper body movements in the horizontal plane than did control subjects. The differences in local dynamic stability were significantly predicted by differences in walking speed, but not by differences in sensory status.[35]

Walker *et al* evaluated the ability of 30 diabetic and 20 non-diabetic individuals to learn to use a lower extremity sensory substitution device to cue gait pattern changes when they walked on a treadmill at three speeds (1, 2, and 2.5 mph) with auditory sensory feedback to cue ground contact greater than 80% duration of baseline, and found that persons in both groups were able to rapidly and significantly alter their gait patterns in response to signals from the sensory substitution device, by changing their gait cycles.[36]

Courtemanche *et al* examined whether a reduced peripheral sensibility caused by diabetic neuropathy increases the attentional demands necessary for controlling and regulating gait by comparing twelve diabetic patients with peripheral neuropathy and 7 control subjects who performed the walking task, auditory stimuli were randomly presented in the third, fourth, or fifth walking cycle on left foot toe off on left foot heel contact.

DPN patients had a smaller cycle amplitude, cycle speed, and percentage of time spent in the single support phase than control subjects. Also, reaction times while walking were higher for diabetic neuropathic patients than for control subjects.[37]

Brain volume and gait

Manor *et al* measured the relationship between walking outcomes (i.e., speed, stride duration vability, and double support time) and regional gray matter volumes in 29 older adults with DPN and compared it with 68 nonneuropathic diabetic patients and 89 nondiabetic control subjects. The authors found that DPN subjects walked more slowly with greater stride duration variability and longer double support as compared with DM and control subjects. DPN subjects with lower gray matter volume globally and regionally (i.e., cerebellum, right-hemisphere dorsolateral prefrontal cortex, basal ganglia) walked more slowly with greater stride duration variability and/or longer double support.[38]

Muscle activity and gait

Sawacha *et al* evaluated the role of altered muscle activity in gait alterations of 20 diabetic subjects with and 20 without neuropathy, and 10 healthy controls. At initial contact and loading response, an early activation of rectus femoris activity occurred in diabetic subjects with and without neuropathy. During midstance a delay of gastrocnemius activity was observed in diabetic non-neuropathic subjects. During terminal swing a delay of rectus femoris and gluteus medius activity was seen in diabetic non-neuropathic subjects'.[38]

Other comorbidities/complications and gait

van Sloten *et al* evaluated the associations of diabetic complications and underlying pathology with daily walking activity in 100 type 2 diabetic patients without manifesting mobility limitations. Neuropathy was associated with a reduction of 1967 steps/day, decreased muscle strength with 1782 steps/day, and an increase in BMI of 1 kg/m(2) with a decrease of 210 steps/day.[40]

Sacco *et al* investigated the influence of diabetic neuropathy and plantar ulcers on plantar sensitivity, symptoms, and plantar pressure distribution during gait with everyday shoes by comparing three groups: a control group (CG; n=15), diabetic patients with a history of neuropathic ulceration (DUG; n=8), and diabetic patients without a history of ulceration (DG; n=10). Diabetic neuropathic patients presented greater pressure-time integrals and relative loads over a larger midfoot area. Diabetic patients with ulceration presented an altered dynamic plantar pressure pattern characterized by overload even when wearing daily shoes.[41]

Kanade *et al* compared walking capacity between 23 subjects with diabetic neuropathy (DMPN), 23 patients with current diabetic foot ulcers, 16 patients with partial foot amputations and 22 patients with trans-tibial amputations. Total heart beat index (THBI) increased and gait velocity and daily stride count fell with progression of foot complications. The maximum peak pressures over the affected foot of patients with diabetic foot ulcers and partial foot amputations were higher than in the group with DMPN.[42]

Rao *et al* examined the relationship between ankle dorsiflexion (DF) range of motion (ROM) and stiffness measured at rest (passively) and plantar loading during gait in individuals with and without diabetes mellitus (DM) and sensory neuropathy, and found that subjects with DM have reduced passive ankle DF ROM and increased stiffness compared to non-diabetic control subjects, however, subjects with DM demonstrated ankle motion, stiffness and plantar pressures, similar to control subjects, while walking at the identical speed, 0.89 m/s (2 mph).[43]

Electrophysiological findings and gait

Yavuzer *et al* investigated the associations between electrophysiological findings and gait characteristics, in forty-six patients with DM (20 subjects with neuropathy, 26 subjects

without neuropathy) and 20 healthy control subjects. NDPN, but not DPN, group revealed slower gait, shorter steps, limited knee and ankle mobility, lower ankle plantar flexor moment and power than C group, and the difference was statistically significant.[44]

Sacco and Amadio studied the sensitive cronaxie in neuropathic and non-neuropathic diabetic patients as a measure of sensorial deficit and found that the pathological response of the sensitive cronaxie worsened progressively for neuropathic and diabetic patients, respectively. Longer double and single stance times, lower minimum vertical force and lower growth rates were seen in the neuropathic patients when compared to diabetic and non-diabetic subjects.[45]

Foot structure and gait

Mueller *et al* compared foot structure were taken from three-dimensional images constructed from spiral X-ray computed tomography and walking peak plantar pressures between twenty people with DM and PN and 20 people without DM. The study found that combinations of structural and walking variables accounted for 47-71% of the variance in the DM group and 52-83% of the variance of PPP during walking in the control group.[46]

Treatment of gait in DPN

Physiotherapy

Sartor *et al* designed a blinded randomised, controlled trial and studied the effect of a physiotherapy intervention on foot rollover during gait, range of motion, muscle strength and function of the foot and ankle, and balance confidence. The intervention was carried out for 12 weeks, twice a week, for 40-60 min each session as described in their study protocol.[47]

Kruse *et al* administered walking exercise as an intervention in combination with lower-extremity strengthening and balance exercises and studied its effects on balance, lower-extremity strength (force-generating capacity), and fall incidence in 79 DPN patients and found improvements in unipedal stance time 12-month post-treatment.[48]

Shoewear

Sacco *et al* investigated the effect of the participants own shoes on gait biomechanics in 24 diabetic neuropathic individuals compared to barefoot gait patterns and 21 non-diabetic healthy controls. The authors found that walking with shoes promoted an increase in the first peak vertical force and the peak horizontal propulsive force. They also demonstrated a higher peak horizontal braking force walking with shoes compared to barefoot. Diabetic participants also had a smaller second peak vertical force in shod gait and a delay in the vastus lateralis EMG activity in barefoot gait compared to controls. Walking with shoes did not attenuate vertical forces in either group.[49]

Insoles

Guldemond *et al* evaluated the effects of 12 different insole configurations on plantar pressures and on walking convenience in 20 patients with diabetic neuropathy.[50]

The configurations included different combinations of a metatarsal dome, varus and valgus wedges and arch supports with different heights were added on a fitted basic insole. For the central and medial regions, plantar pressure reductions (up to 36% and 39%, respectively) were found when using a dome, standard and extra supports. The largest reductions were achieved with combination of a dome and extra support. The basic insole and a standard support received the best ratings for walking convenience and gradually worsened by adding extra support, a varus wedge and a dome.

Visual feedback

York *et al* examined the role of visual feedback in the reduction of plantar pressures through teaching a "new" gait pattern to 29 olde-aged diabetic peripheral neuropathy

subjects. Subjects were randomized into feedback and no-feedback groups. Instruction to pull the leg forward from the hip to initiate swing rather than push off the ground with the foot while walking was given to all subjects. The feedback group received visual feedback regarding peak plantar pressures after each practice trial. The no-feedback group received no feedback. Peak plantar pressures were significantly reduced from baseline to retention 2 testing at the first metatarsal area in the feedback group. The feedback group walked slower at retention 1 and 1-week testing compared with baseline.[51]

Discussion

The study was aimed to evaluate the abnormalities of gait in diabetic peripheral neuropathy by reviewing studies on assessment and treatment, and there were more number of studies on assessment compared to that of treatment of gait in people with DPN.

Many assessment studies on gait in people with DPN had demonstrated altered gait parameters such as reduced gait speed, reduced double support time, reduced step length, reduced ankle range of motion, with increased ankle invertor-evertor moment; altered plantar pressures with increased load under midfoot compared to rearfoot; earlier muscle activity of soleus, tibialis anterior, vastus medialis and medial hamstrings with delayed muscle activity of vastus lateralis and lateral gastrocnemius; longer loading time with decreased mediolateral and longitudinal center of pressure excursions; with more likelihood for gait-related injuries. The gait deviations were correlated to brain volume, electrophysiological findings, lower limb sensorimotor function, foot structure, muscle activity, and other comorbidities and/or complications of diabetes such as ulcers and foot deformities.

There is a need to study inter-relationships between gait deviations and clinical examination findings,[52] clinical assessment scale scores,[53] neurodynamic examination findings[54] and/or quality of life[55] in people with DPN. The alterations in gait reported in the reviewed studies were much different from either diabetic individuals[56] or neuropathy[57] individuals considered alone thus reflecting the multifaceted multidimensional impact of peripheral neuropathy on gait in DPN population.

Gait-related interventions in DPN population were physiotherapy including walking prescription, lower extremity strengthening and balance exercises, footwear and insoles, and visual feedback which were shown to improve balance, gait speed, muscle activity and plantar pressures in this population. The evidence for intervention of gait and its deviations in people with DPN was limited and there is need for future high quality trials in this population-specific gait changes to medical,[58] surgical,[59] physiotherapeutic,[60] and neurodynamic[61] and/or acupuncture[62] treatment methods.

Future evidence-informed guidelines for DPN should thus incorporate assessment and treatment of gait from a multidisciplinary biopsychosocial perspective.[63]

Conclusion

Many assessment studies on gait in people with DPN had demonstrated altered gait parameters such as reduced gait speed, reduced double support time, reduced step length, reduced ankle range of motion, with increased ankle invertor-evertor moment; altered plantar pressures with increased load under midfoot compared to rearfoot; earlier muscle activity of soleus, tibialis anterior, vastus medialis and medial hamstrings with delayed muscle activity of vastus lateralis and lateral gastrocnemius; longer loading time with decreased mediolateral and longitudinal center of pressure excursions; with more likelihood for gait-related injuries. The gait deviations were correlated to brain volume, electrophysiological findings, lower limb sensorimotor function, foot structure, muscle activity, and other comorbidities and/or

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Gait-related interventions in DPN population studied were physiotherapy including walking prescription, lower extremity strengthening and balance exercises, footwear and insoles, and visual feedback which were shown to improve balance, gait speed, muscle activity and plantar pressures in this population.

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Review Article

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Brain Pacemaker

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Abstract

Brain implants, often referred to as neural implants, are technological devices that connect directly to a biological subject's brain - usually placed on the surface of the brain, or attached to the brain's cortex. "Brain pacemakers" are used to treat people who suffer from epilepsy, Parkinson's disease, major depression and other diseases. Pacemakers may also be implanted outside the brain, on or near the spinal cord (spinal cord stimulation), and around cranial nerves such as the vagus nerve (vagus nerve stimulation), and on or near peripheral nerves. The deep brain stimulation system consists of three components: the implanted pulse generator (IPG), the lead, and the extension. DBS leads are placed in the brain according to the type of symptoms to be addressed. Brain implants electrically stimulate, block or record signals from single neurons or groups of neurons networks in the brain. DBS reduces tremor, rigidity, bradykinesia, gait problems, dyskinesia, motor fluctuations, dystonia. The innovative technology may also come to the next generations that may replace the 1st generation Brain Pacemakers. There are very few cons for brain pacemakers that outweigh the potential benefits. In the short amount of time brain pacemakers have progressed so far. Given more time, brain pacemakers will be a really useful and a powerful technology.

Keywords: Brain pacemaker; Brain implants; Deep brain stimulation; Parkinson's disease; Alzheimer's disease.

Introduction

Neural-implants such as deep brain stimulation and Vagus nerve stimulation are increasingly becoming routine for patients with Parkinson's disease and clinical depression respectively, proving themselves as a boon for people with diseases which were previously regarded as incurable.

Brain implants, often referred to as neural implants, are technological devices that connect directly to a biological subject's brain - usually placed on the surface of the brain, or attached to the brain's cortex.[1] "Brain pacemakers" are used to treat people who suffer from epilepsy, Parkinson's disease, major depression and other diseases. The pacemaker is a medical device that is implanted into the brain to send electrical signals into the tissue. Depending on the area of the brain that is targeted, the treatment is called deep brain stimulation, or cortical stimulation. Brain stimulation may be used both in treatment and prevention. Pacemakers may also be implanted outside the brain, on or near the spinal cord (spinal cord stimulation), and around cranial nerves such as the vagus nerve (vagus nerve stimulation), and on or near peripheral nerves.[2]

Deep brain stimulation (DBS) was first used in the 1970s for the treatment of chronic pain.[3] A common purpose of modern brain implants is establishing a biomedical prosthesis circumventing areas in the brain that have become dysfunctional after a stroke or other head injuries, the sensory substitution, e.g., in vision, and even to record brain activity for scientific reasons. Some brain implants involve

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creating interfaces between neural systems and computer chips. This work is part of a wider research field called brain-computer interfaces.[1]

Components and Placement

The deep brain stimulation system consists of three components: the implanted pulse generator (IPG), the lead, and the extension. The IPG is a battery-powered neurostimulator encased in a titanium housing, which sends electrical pulses to the brain to interfere with neural activity at the target site. The lead (also called an electrode) is a thin (approximately 1.3 mm in diameter) coiled wire insulated in polyurethane with four platinum iridium electrodes which is inserted through a small opening in the skull and is placed in one of three areas of the brain.[3,4,5] The tip of the electrode is positioned within the targeted brain area. The lead is connected to the IPG by the extension, an insulated wire that runs from the head, down the side of the neck, behind the ear to the IPG, which is placed subcutaneously below the clavicle or in some cases, the abdomen.[4,5] The IPG can be calibrated by a neurologist, nurse or trained technician to optimize symptom suppression and control side effects.[3]

DBS leads are placed in the brain according to the type of symptoms to be addressed. All three components are surgically implanted inside the body. Lead and extension implantation may take place under local anesthesia or with the patient under general anesthesia ("asleep DBS"). A hole about 14 mm in diameter is drilled in the skull and the electrode is inserted. The installation of the IPG and lead occurs under general anesthesia. The right side of the brain is stimulated to address symptoms on the left side of the body and vice versa.[4]

Implantation of the DBS system is performed in 2 stages. During the first stage, the DBS lead is implanted stereotactically into the target nucleus. A combination of microelectrode recording (MER) and macroelectrode stimulation is used to refine the desired target physiologically. Magnetic resonance imaging (MRI) of the brain is performed immediately after the procedure to confirm proper electrode placement and to make sure that no hemorrhage has occurred. During the second stage, the DBS lead is connected subcutaneously to an implantable pulse generator (IPG), which is inserted into a pocket beneath the skin of the chest wall, like a pacemaker.[3]

Deep Brain Stimulation provides monopolar or bipolar electrical stimulation to the targeted brain area. Stimulation amplitude, frequency, and pulse width can be adjusted to control symptoms and eliminate adverse events. The patient can turn the stimulator on or off using an Access Review Therapy Controller or a handheld magnet. The usual stimulation parameters are an amplitude of 1-3 V, a frequency of 135-185 Hz, and a pulse width of 60-120 msec.[3]

Biochemistry

Brain implants electrically stimulate, block or record (or both record and stimulate simultaneously) signals from single neurons or groups of neurons (biological neural networks) in the brain. The blocking technique is called intra-abdominal vagal blocking. This can only be done where the functional associations of these neurons are approximately known. Because of the complexity of neural processing and the lack of access to action potential

Table 1: The following table summarizes the three different sites for DBS therapy

DBS Site	Effect of Therapy
Thalamus (Vim)	Reduces tremor but not the other symptoms of PD
Globus pallidus (Gpi)	Reduces tremor, rigidity, bradykinesia, gait problems, dyskinesia, motor fluctuations, dystonia
Subthalamic nucleus (STN)	Reduces tremor, rigidity, bradykinesia, gait problems, dyskinesia, motor fluctuations, dystonia.[6]

related signals using neuroimaging techniques, the application of brain implants has been seriously limited until recent advances in neurophysiology and computer processing power.[1]

It has been shown in thalamic slices from mice that DBS causes nearby astrocytes to release adenosine triphosphate (ATP), a precursor to adenosine (through a catabolic process). In turn, adenosine A1 receptor activation depresses excitatory transmission in the thalamus, thus causing an inhibitory effect that mimics ablation or "lesioning".[4]

Programming of the stimulator system is usually done on an outpatient basis, although in some DBS centers the system may be activated before discharge from the hospital. It may also be done in a rehabilitation center, where other therapies are being provided. Programming usually starts within a few weeks of the DBS surgery.[6]

Complications

Serious or permanent complications:

- Death is probably less than one percent.
- A 7.5% risk of stroke from bleeding in the brain during surgery.
- Hydrocephalus is a rare, but possible.

Temporary or reversible complications:

- Changes in mood, memory and thinking
- Seizures
- Infection
- Stroke
- Problems with movement and speech
- Stroke-like symptoms, such as weakness, numbness and slurred speech
- Worsening dyskinesia
- Headache, dizziness, tingling of the face or limbs, and an electrical jolting sensation

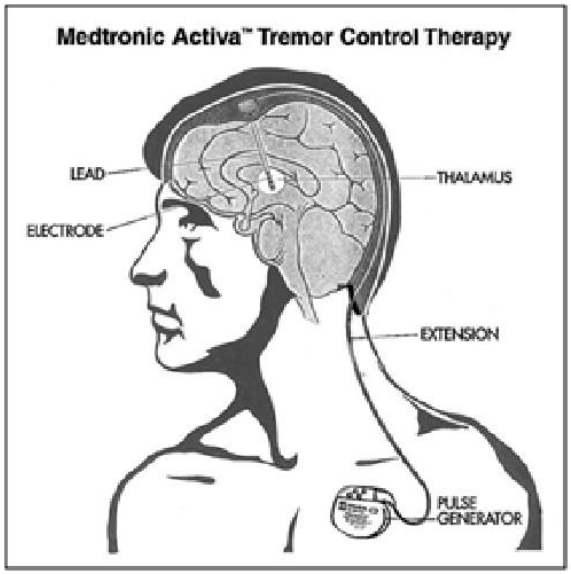
Malfunctioning DBS devices:

- Lead migration, in which the electrode has moved from the target site
- Fracture, disconnection or damage of the connecting wire
- Malfunction or injury to the neurostimulator, from direct physical contact
- Misplacement of the brain electrode.[6]

Education alerts and Warnings for the clients with brain pacemakers

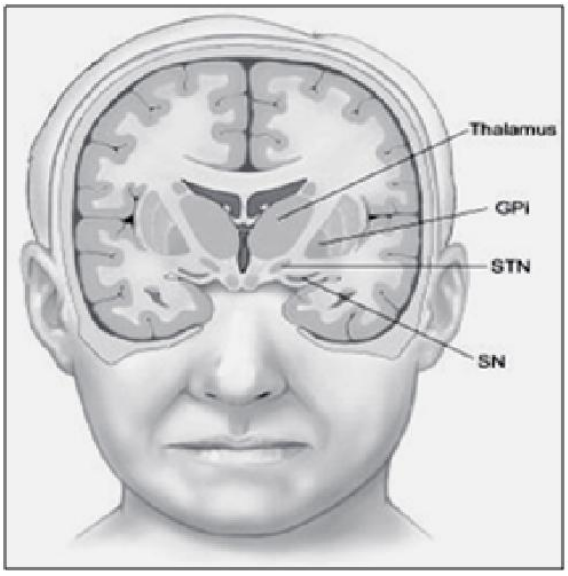
1. When entering stores with theft detection devices, walk in the middle of the door opening to minimize the likelihood of the DBS system being turned off.
2. Remove any unnecessary magnets in your home.
3. Stand away from the microwave when in use.
4. Avoid walking through metal detection devices if possible; ask security personnel to perform a manual body check at airports.
5. Carry a wallet-size medical card that describes the DBS system and warnings to show to security and store personnel.
6. Get a medical-alert bracelet that states that you have a DBS system and that you have a wallet card for special warnings and emergency contact phone numbers.
7. Do not allow any electrical or magnetic device to be placed near your neurostimulator, connecting wire or implant site on your scalp.
8. Carry your magnet or patient controller with you whenever possible.
9. Avoid hobbies or occupations that involve routine exposure to high voltage electrical and/or magnetic fields; in particular, avoid arc welding.[6]

Figure 1



Reference: Hauser RA. Deep Brain Stimulation for Parkinson Disease. Medscape reference: Drugs, diseases and procedures. 2012 Feb 1 [cited on 2013 Jul 24]. Available from URL: http://emedicine.medscape.com/article/1965354-overview#a1.

Figure 1



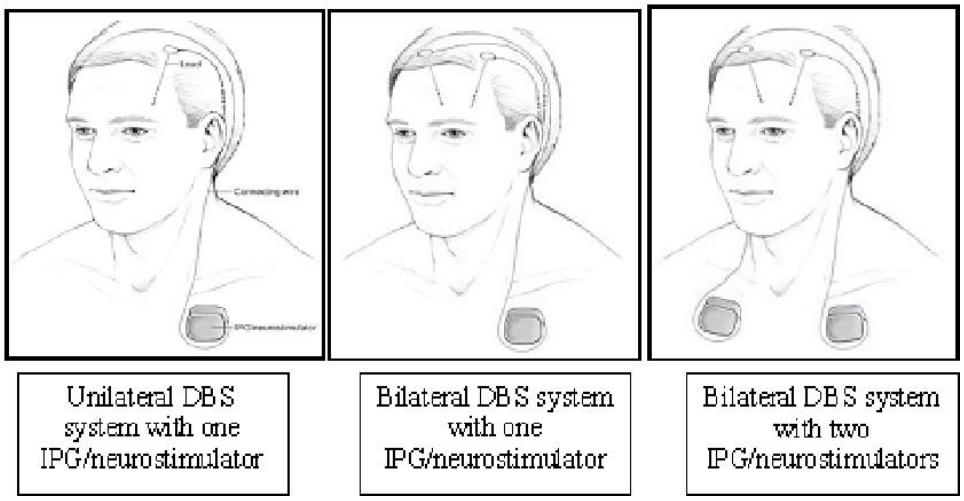
Reference: Lyons JM, Okun MS. Parkinson's disease: Guide to Deep Brain Stimulation Therapy. 2nd ed. USA, The National Parkinson Foundation Inc.– Medtronic; 2007. p 12.

Future inovative research on brain pacemakers

A thorough understanding of how the brain pacemaker works on brain cells and normalizes brain function is critical to the future success of this technology. Abnormal rhythmic brain cell firing are at the root of many movement disorders and other neurologic conditions. Therefore, a research is essential to know how therapeutic stimulation effects individual brain cells, and what improvements can be seen in patients with Brain Pacemakers.

The innovative technology may also come to the next generations that may replace the 1st generation Brain Pacemakers with leads, the generator and the electrodes which may have a small chip that can be directly implanted just beneath the brain cells where there is an essential stimulation or the

Figure 2



Reference: Lyons JM, Okun MS. Parkinson's disease: Guide to Deep Brain Stimulation Therapy. 2nd ed. USA, The National Parkinson Foundation Inc.– Medtronic; 2007. p 14,16.

depression of cells is required.

A second generation of Brain Pacemaker: a wireless and rechargeable system is the next technology a "smart" device, one that can sense abnormal brain firing and suppress abnormal activity only when required, in other words, work on demand that may benefit further for treating complex brain disorders.

The Brain Pacemaker has tremendous potential to treat many conditions result from disorganized brain firing, including dystonia, epilepsy, obsessive-compulsive disorder, refractory depression, chronic pain and perhaps even addiction, obesity and other eating disorders.

Further the innovation technology, which may protect the individuals with Brain pacemaker, through satellite tracked signals of impulses generated through the Medical Centers or Hospitals to control the exact frequency of Pulse generators required when the tremor attacks or malfunctions occur in the brain cells, may be developed.

Conclusion

The brain is a very complex organ, and it controls everything that the human body does. Therefore technology that can improve the health of the brain and allow those who have difficulty using their brain effectively due to medical conditions is extremely useful. Parkinson's, Alzheimer's and Depression are conditions that can cripple a person and burdena family emotionally and financially. There is tremendous promise for brain pacemakers because even though they're still in their earlier stages, they're already making a difference in people's lives. In the year 2050, the number of people with Alzheimer's is projected to triple and society needs an effective treatment for the condition. About a million people with Parkinson's disease have had brain pacemakers implanted and many of them are seeing the positive effects from the pacemaker such as fewer tremors, less spasms and overall more control of their body. Depression is such a subjective and relative condition that it is difficult to treat. Brain pacemakers can give a standardized treatment for the condition and increase self-esteem, and neural activity. Brain pacemakers scientifically have so much support at the current moment due to the recent success and the sound scientific thought process. There are very few cons for brain pacemakers that outweigh the potential benefits. In the short amount of time brain pacemakers have progressed so far. Drug therapies have been used for decades and have had limited and sporadic results that vary from person to person. Given more time, brain pacemakers will be a really useful and a powerful technology.

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Case Report

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Volume 6 Number 1, January - June 2014

Extracranial Extension of Anaplastic Ependymoma: Case Report and Literature Review

Fahad Alotaibi*, Mousa Alabbadi, Abdulrahman Sabbagh***, Maqsood Ahmad******

Abstract

16-years old male diagnosed six years ago as grade 2 ependymoma presented to our hospital with extracranial extension to the left side of face involving the left eye and left cheek.

Keywords: Glioma; Ependymoma; Extracranial extension.

Case report

16-years old male referred to our hospital king fahad medical city KFMC as case of recurrent brain tumor presented with decrease level of consciousness, visual disturbance, double vision and right side weakness for the last two months. He experienced only two cerebral convulsions the latest three years ago. For the last two months preadmission he experienced disturbance in his balance, oral intake had decreased significantly and often accompanied with vomiting. So far, he underwent six operations for excision of the tumor the latest was on six months for debulking of recurrent tumors from face, zygom and middle anterior cranial fossa and cranioplasty using titanium mesh, and muscle from the right upper leg. No pathology report from the the latest operation which was done in Germany. The previous operation were done in king faisal specialist hospital for debulking of the tumor and exploration of the orbital component and extended left frontotemporparital craniotomy. Drilling of the sphenoid wing was done because of tumor invasion to the bone. The orbit was explored superiorly through the orbital roof with excision of the orbital component, extra axial component excised with partial resection of the intra axial component due to infiltration of the cavernous sinus and extension of the tumor to the skull base foramen. The patient received full dose of radiation and chemotherapy after the 3rd operation on 2005.

On arrival he was disoriented to time, place and person. With proptosis of the left eye , swelling of left mid and lower face, with area of erythema adjacent to the left eye which was inflamed and red with evidence of retinal hemorrhage the in fundoscopy exam ,the right eye was completely normal (Figure 1). Gross

Figure1: Pictures showing the tumor invading the left side of the face including left eye

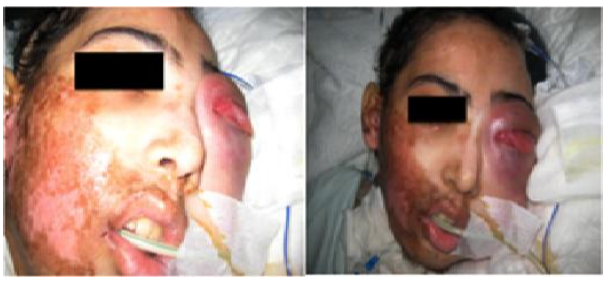

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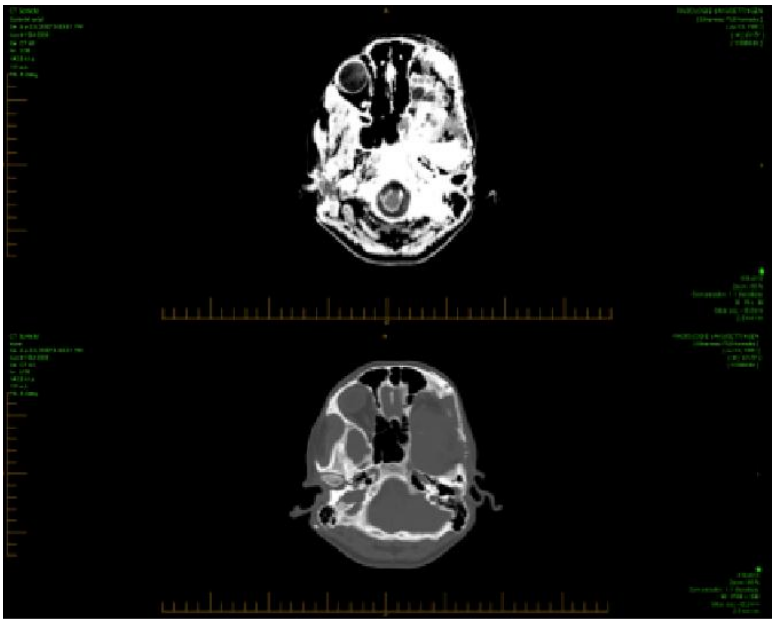
Figure 2: Axial CT scan showing direct invasion of the tumor to the skull base, lateral orbital wall with extension to the intraorbital compnant

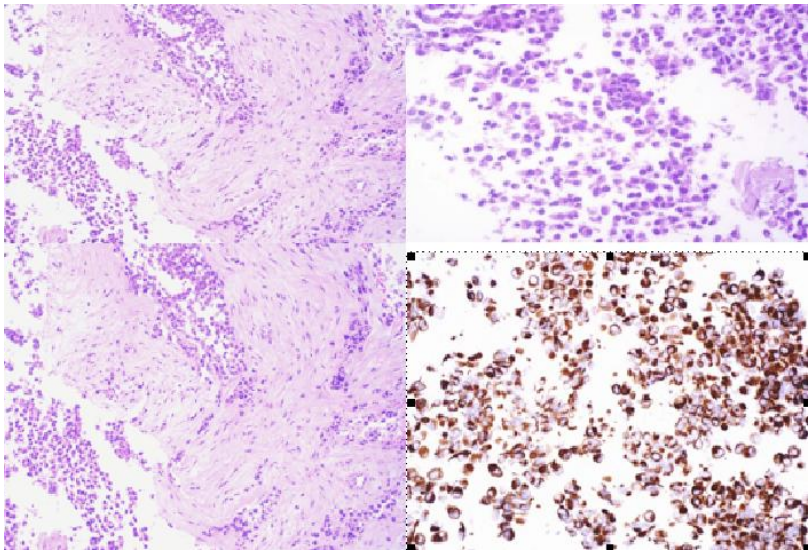


motor power preserved in the left side but significant right hemiparesis around 3/5 for both lower and upper limbs. Normal examination for the pharynex, ear drums and the abdomen.

Cerebrospinal fluid sent for cytology and the result was negative for malignant cells. Computerized tomography CT revealed the extensive bony defect of the skull base secondary to tumor invasion and previous surgeries with tumor remnants extending to the left maxillary, ethmoidal sinuses and left side of the nasal cavity.

Biopsy was taken from the left facial mass. The core biopsy at low power examination

Figure 3: Low power view of the tumor that infiltrates the stroma in single cells (Hematoxylin and Eosin 200X). Higher view showing the abundant eosinophilic cytoplasm and abnormal nucleoli (Hematoxylin and Eosin 400X). The tumor cells are strongly positive for the Glial Fibrillary Acidic Protein.



(Figure 4) showed myxoid stroma, which is infiltrated by tumor cells. The tumor cells exhibit slightly fibrillary background and infiltrated the stroma predominantly by single cells. At high power examination the cells contain abundant eosinophilic cytoplasm and occasional binucleated cells were identified (Figure 5). The nuclei were relatively small to medium size with significant nuclear pleomorphism and frequent mitosis. Well-controlled immunohistochemical stains revealed strong positivity for Glial Fibrillary Acidic Protein (Figure 6) and were concomitantly negative for lymphoid, epithelial and melanocytic markers confirming the glial nature of this tumor. Final diagnosis was compatible with anaplastic ependymoma WHO grade three.

Literature review

Most of the extracranial metastases was frequently found in medulloblastoma, glioblastoma multiforme, malignant meningioma and ependymoma.[1] Despite multiple cases reports in the literature showed extracranial metastases in patient with glioma most of it to the lung, bone, liver, lymph nodes, mediastinum, pleura and kidneys but no case very few cases reported direct invasion through the skull.[1,2,3] Although, there is only one study mentioned a direct invasion of the anaplastic cerebral glioma with metastases outside the neuraxis, which were seen among series of 1600 glioms. The series included 4 males and 4 females ranging in age from 5-58 years at the time of death. There were two children with anaplastic ependymomas , oligodendrogliomas, and five young or middle aged adults with astrocytomas grade III or IV. All patients had one or more craniotomies, and five had radiotherapy before the appearance of the remote tumor deposits. All the tumors showed invasion of meninges and/or ventricle walls, and in four cases they transgressed the dura and surrounding bone or soft tissue, and all the patient showed distance metastasizes to the bone liver and lung.[4]

Conclusion

This 16-years old male had recurrent anaplastic ependymoma tumor with direct extracranial extension to the left side of the face which is reported only in 4 cases before as a part of retrospective study of about 1600 glioms[4]. But in that study the patient were had distance metastases to the other organ, which is not the case in our patient.

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Case Report

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Multi-Corporal Abscess Formation due to Esophageal Perforation Post Anterior Cervical Discectomy and Fusion (ACDF)

Alturki A.*, Basamh M., Awwad W***, Jarzem P.******

Abstract

Anterior cervical discectomy and fusion (ACDF) is one of the most commonly performed spinal procedures in the United States. With an excellent outcome in most of them. The complications associated with this procedure are rare but can be troublesome and life threatening. We report the case of a patient that sustained a missed, late esophageal perforation after an anterior cervical discectomy and fusion, leading to multiple abscesses in the epiduaral, paraspinal, mediastinal, parapsoas, and pleural spaces, who survived after multiple procedures and was able to ambulate after a prolonged course of care. A proposed algorithm for treatment is included.

Keywords: ACDF; Multiple epidural abscess; Esophageal perforation.

Introduction

Anterior cervical discectomy and fusion (ACDF) is one of the most commonly performed spinal procedures in the United States.[1] More than 100,000 procedures are performed annually, with an excellent outcome in most of them.[1–8]

It is a well-established procedure for cervical myelopathy, cervical radiculopathy, neoplasms, cervical spondylitic diseases and cervical trauma.

The complications associated with this procedure are rare but can be troublesome.[10,11] These have been adequately described in the literature.[10-15]

Among them, one of the most serious is the extrusion of the implanted instrumentation, with various consequences each time.

We present a case of extrusion of implanted screws for anterior cervical plate insertion post ACDF procedure leading to esophageal perforation and extensive abscess formation. We also reviewed the literature on the incidence of this complication and its management.

Case report

A 62 years old male presented with septic shock one month after an ACDF of C5-C7, admitted to the intensive care unit in another hospital. He underwent resuscitation, Intubated and was eventually diagnosed with neck abscess, bilateral empyema and right psoas abscess. Underwent drainage for the neck and psoas abscesses and started on multi-Antibiotic treatment regimen. Due to the wide extent of his infections and the need for Tartiary care we accepted the patient in transfer and began a series of investigations and treatments.

62 days post op: Upon admission to our intensive care unite this Patient diagnosed with

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Figure 1: Screw disengagement from the plate disally

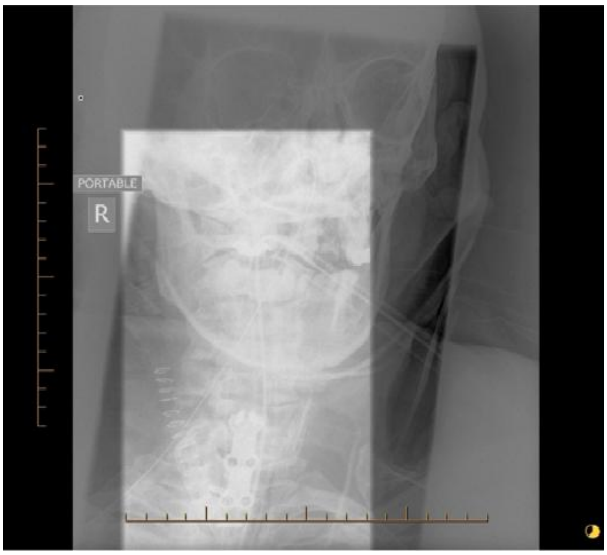


Figure 2: Sagital MRI STIR and T2 showing spondylodiscitis and epidural abcess

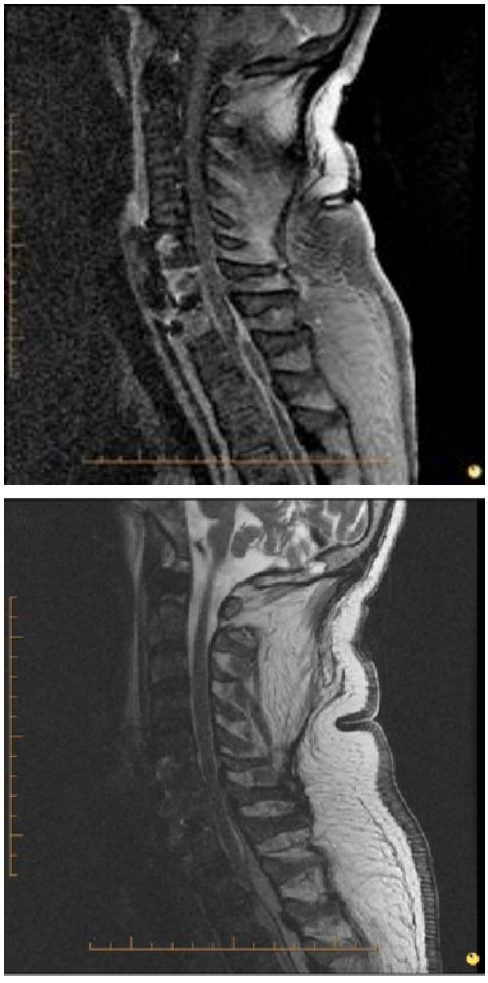


Figure 3: T2 thoracic and lumbar sagittal showing extensive epidural abcess

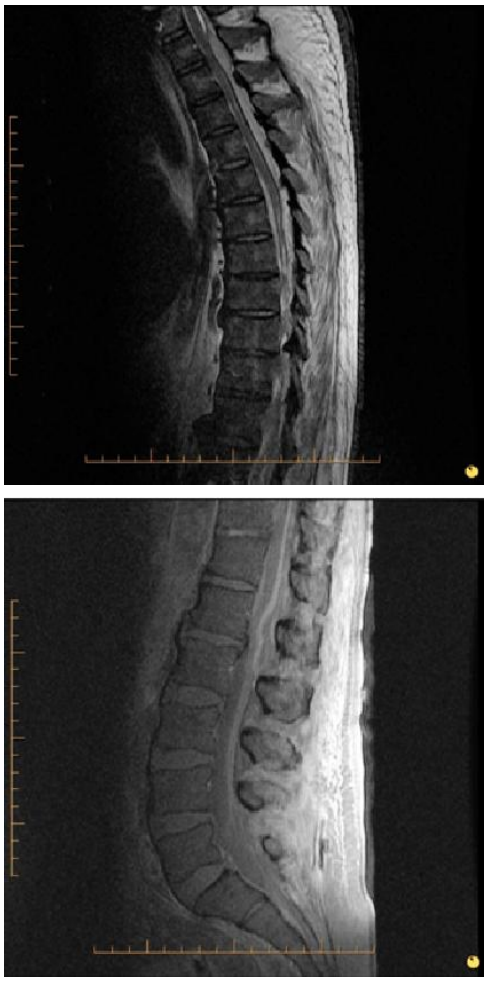


Figure 4: Axial CT stan showing T2 level decompression and bilateral chest tube

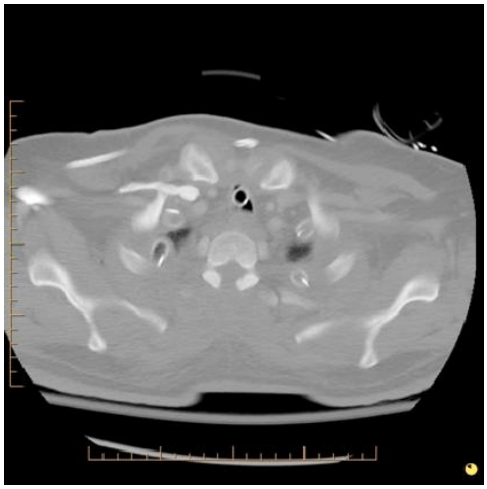


Figure 5: Axial CT scan at T7 level showing decompression and multiple chest tubes for decortication

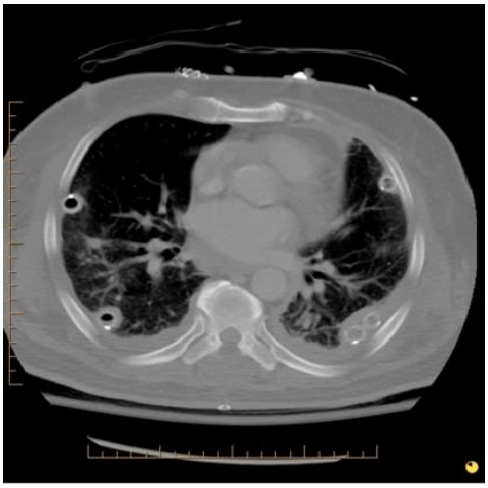


Figure 6: Axial CT at L2 level showing decompression

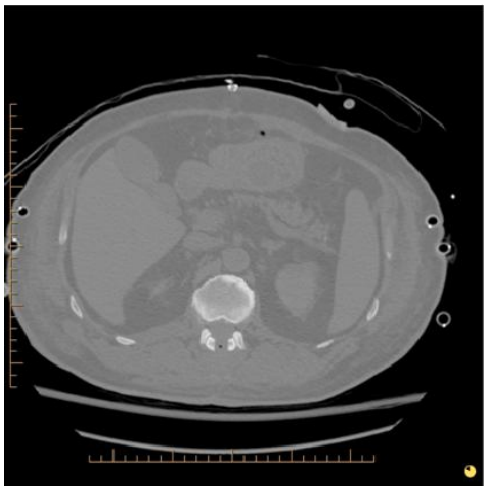


Figure 7: Axial CT at L5 level showing decompression

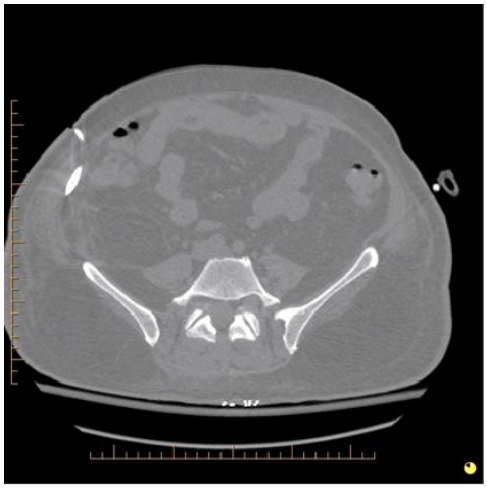


Figure 8: Fluro shot with Kelly holding the last loose screw for its removal

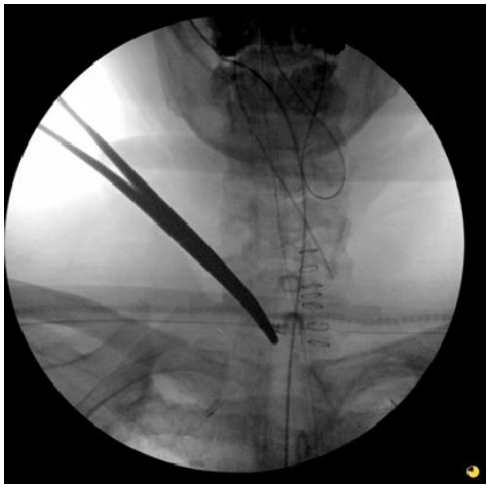


Figure 9: Sagital CT scan post removal of Hardware and T2 lamenectomy

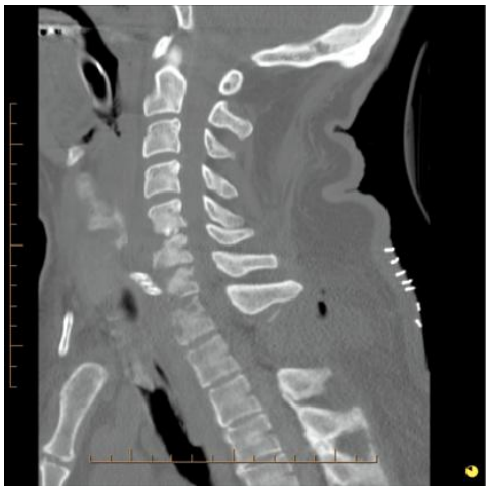


Figure 10: X-Ray post pectorals flap for esophageal repair

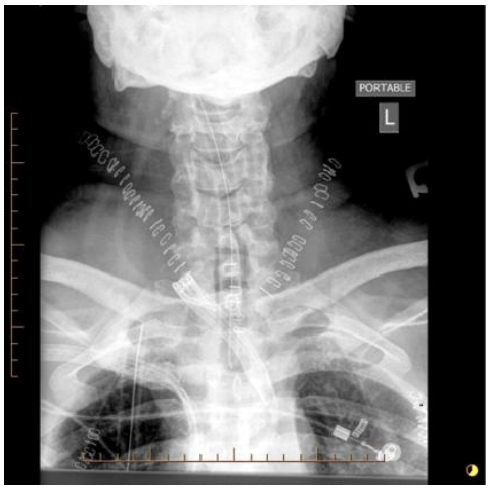


Figure 11: Sagital CT scan post corpectomy and lilac crest bone grafting

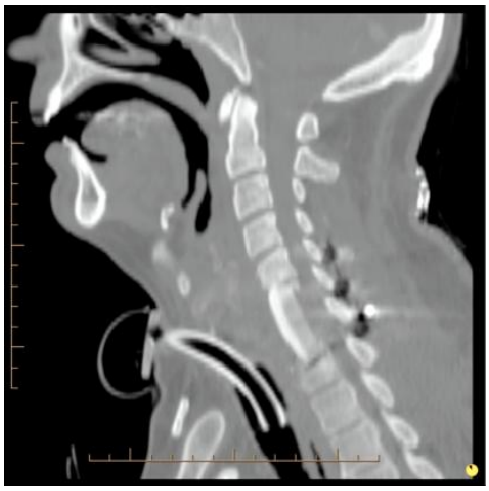


Figure 12: X-ray post posterior fusion

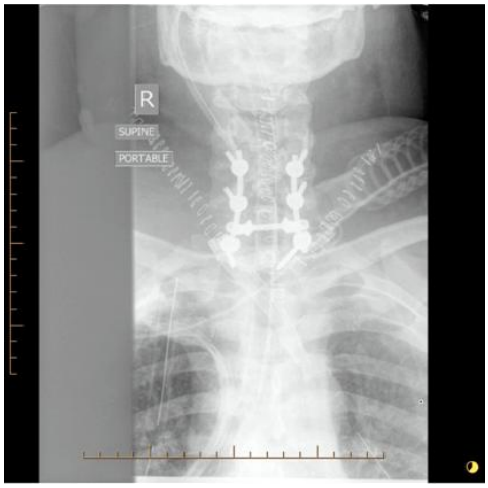


Figure 13: Sagital CT showing lateral mass screws for posterior fusion

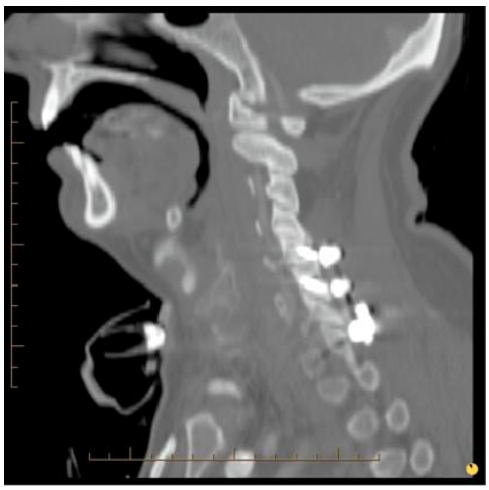
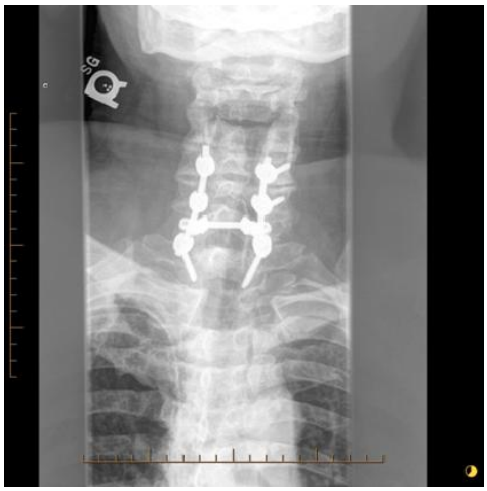


Figure 14: X-ray at Follow up post extubation



extensive epidural abscess, cervical spondylodiscitis, Psoas abscess and bilateral empyema. (Figure 1, 2 and 3). The esophageal perforation had not yet been diagnosed. In order to better diagnose the extent of the patient's cervical infection, whole spine MRI was obtained demonstrating extensive epidural and anterior and posterior paraspinal abscess formation.

63 days post op: Due to ventilation issues the decision was to start with left lung decortication and multiple chest tube insertion.

64 days post op: 4 level laminectomy (T2, T7, L2 and L5) and irrigation debridement. (Figure 4, 5, 6 and 7).

67 days postop: anterior irrigation and debridement and hardware removal. Many of the screws had disassociated from the plate (Figure 8 and 9)

69 days postop: feeding tube as well as Right chest tube insertion performed.

70 days postop: percutaneous drainage of retroperitoneal collection carried out.

74 days postop: Due to the persistent septicemia and abscess formation , patient underwent radiological studies which confirm the diagnosis for esophageal perforation.

75 days postop: One day after diagnosis, which was two and a half months after the index procedure, right lung decortication

carried at the same setting for esophageal repair by pectoralis flap. (Figure 10)

81 days postop: Anterior spine debridement, C6-C7 corpeectomy and anterior C5-T1 fusion with Illiac crest bone graft. (Figure11)

87 days postop: Posterior C5-T1 stabilization and repeat irrigation and debridement for thoracic epidural and paraspinal abscesses. (Figure 12, 13).

90 days postop: The patient is aseptic and awake but due to failure to extubation he receives a tracheostomy, and few days later transfer to the ward.

We saw the patient two months and four months post discharge at our clinic, his neurological exam at the last follow up was 4/5 for bilateral upper limb and 3/5 for hip flexor 4/5 knees extensors and 3-4/5 for ankles dorsiflexion and planterflexors, and was off antibiotics and wounds fully healed. (Figure 14)

Discussion

Although the use of anterior plating has remained controversial, it has become a common practice among spinal surgeons.[17-19] Anterior cervical plating is not without complications; among them, extrusion of the failed instrumentation is one of the most uncommon but serious complications.[21-25] Esophageal perforations is a dreaded but know complication of anterior plating, but these perforations are generally detected immediately after the surgery.[23,25] Delayed perforations like ours are unusual but are generally associated with anterior migration or dislocation (plates, screws, wires, bone grafts) of the fixation devices.[9,29,30]

Esophageal perforation related to anterior cervical surgery is rare and therefore may not be detected early. This complication may be life threatening.[26]

The incidence of esophageal injuries ranges between 0% and 3.4%, Early presentations are mostly caused by direct injury to the esophagus by sharp instruments or retractor blades .[9,26-30]

The mortality rate for all causes of esophageal perforation is about 20%, rising to 50% if treatment is delayed.[2,31]

Perforations occur more frequently after surgery for cervical spine fractures than for degenerative disease.[9,28,30]

Most perforations occur at the levels C5–C6 and C6–C7, in accordance with the prevalence of cervical spine pathology.[26]

Delayed perforations are mainly caused by anterior migration or dislocation (plates, screws, wires, bone grafts) of the fixation devices.[9,29,30]

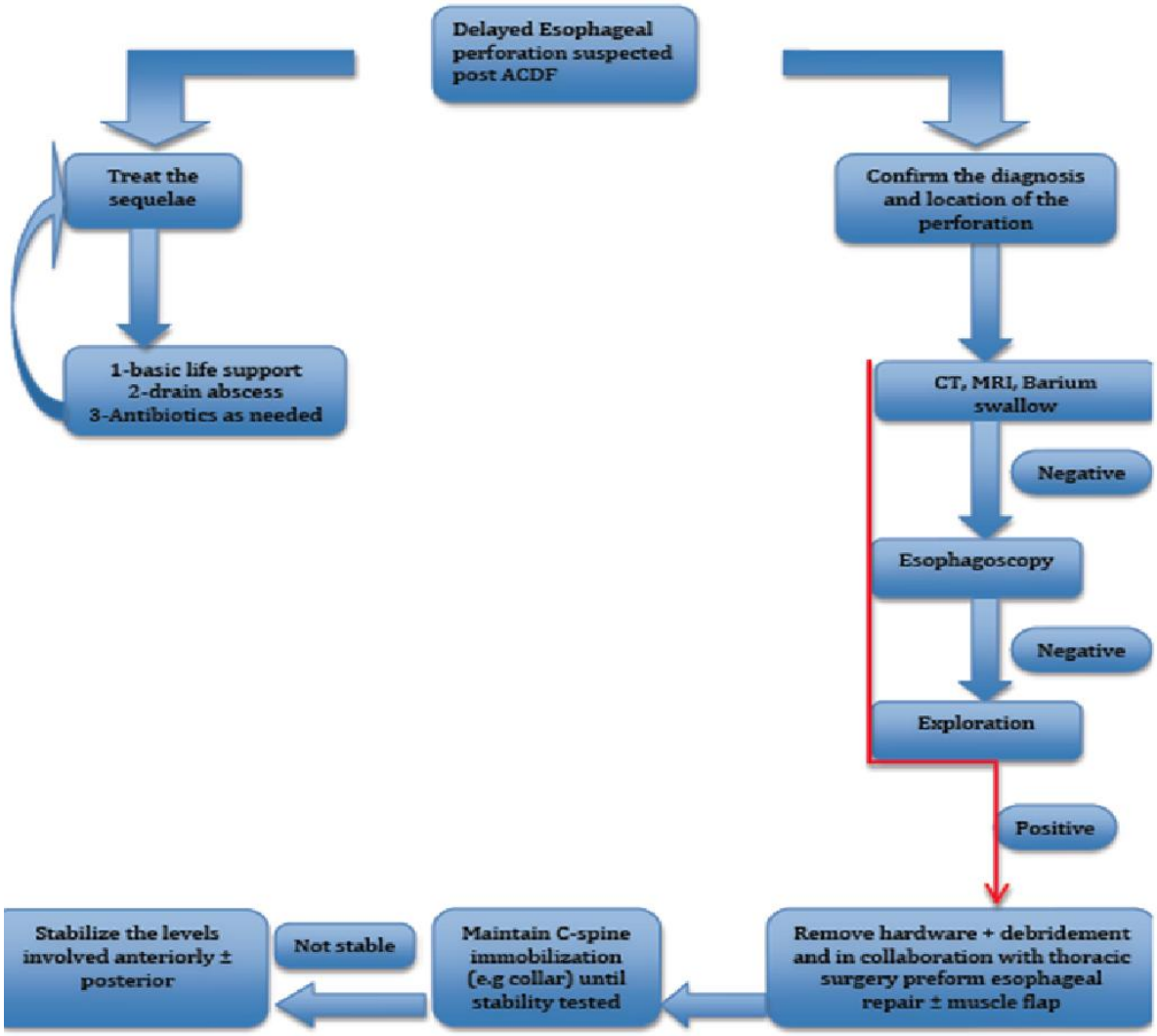
Hanci *et al* suggested that esophageal perforations were due to pressure sores caused by the metallic implant and its microtrauma effect as a mechanism of the observed esophageal perforation in three cases.[25]

Asymptomatic perforations have been described[14,32,33], but symptoms usually consist of dysphagia, local subcutaneous emphysema, fever and signs of infection. Other features are neck and throat pain, hoarseness, aspiration, unexplained tachycardia and blood in the nasogastric tube.[9,27-30,33] Symptoms of late perforation are usually discrete.

A high index of suspicion for esophageal perforations should include: (a) cervical spinal column or cord injury with previous anterior cervical spine surgery (especially when instrumentation is used); (b) systemic signs of fever, leucocytosis or an unexplained persistent tachycardia; (c) imaging evidence of air or fluid in the cervical fascial spaces or mediastinum.[9,28-29]

Diagnosis is made by imaging or endoscopic studies.[9,28-30], although these may give false-negative results. Therefore, clinical suspicion of the complication is most important. Plain X-rays may reveal subcutaneous emphysema, widening of the retropharyngeal space or loosening of hardware, but have a false-negative rate of 10–46%.[30] Contrast swallowing studies can aid

Figure 15



in the diagnosis and in determining the location of the perforation. CT scans can show graft displacement and abscess formation. However, in the series of 44 patients by Gaudinez *et al* imaging studies indicated an esophageal injury in only 72.7% of the affected patients.[9] Esophagoscopy can give false-negative results as well with a reported sensitivity ranging between 50% and 100%. [9,28,30] In cases of high clinical suspicion with inconclusive imaging studies, surgical exploration of the neck may be warranted. This was illustrated in the series by Gaudinez *et al* in which eight patients (18%) had to have the diagnosis of a perforation confirmed during a surgical exploration.[9]

The management of such a situation consists of surgery with removal of the hardware, drainage of abscesses and—if possible—primary closure of the perforation, parenteral nutrition and antibiotic therapy.[9,28-30]

If the perforation is diagnosed intra-operatively, suturing of the defect is sufficient. However, there is not much data on the role of antibiotic prophylaxis in this situation.

In early perforations, repair of the lesion may be possible with or without muscle flaps, Presence of an abscess requires surgical drainage. In case of a late diagnosis, surgical treatment should be restricted to removal of the hardware, drainage of abscesses and diversion of the salivary flow to the cervical skin.[9,30,34]

(Figure 15) summarize our management strategy.

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Use of Cortical Screws for Soft Tissue Fixation

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Abstract

Use of cortical screws for bony fixation is a well known phenomenon but use of same screws for softtissue fixation is a new concept. A case of necrotising fascitis of scalp was treated with repeated debridement and local flaps. In this case we had no difficulty in covering the wound as flaps were preoperatively planned but insetting tough oedematous flaps was difficult, hence we used cortical screws for fixation and they served the purpose well.

Keywords: Cortical screws; scalp reconstruction,rotation flaps; screws.

Introduction

Scalp reconstruction is challenging due to its tough layers and inelasticity of the skin.

How ever due to its high vascularity and definite axial vessels, planning a flap is not difficult.We had a situation where elevation of flap and planning was not difficult but insetting the flap was challenging. Hence we used cortical screw for inset of scalp flaps and they served the purpose very well.

Case

A 55yr old diabetic female visited the outpatient department for a swelling with redness over right temparo-parietal region. She was treated on out patient basis and patient did not visit the hospital for next 5 days. On 6th day patient was admitted under accident and emergency department with complaints of right half of face swelling, pain, fever. On examination there was necrotic patch over parietal region, tachycardia,tachyopnea and swelling in the right lateral cheek wall. Patient was diagnosed facial cellulitis in sepsis, probably due to parietal region abscess was done. Immediate debridement of parietal region was done to remove the dead and necrotic tissue and further incision given to expose the infected fascia (probably loose aerolar tissue and galea.(Fig. 1) Thorough wash was given. Incision over the scalp given in such way that elevated flaps covered the exposed bone. Incision extended to parotid region as well. After repeated debridement, a patch of 4 X 7cms skull bone was exposed. Reconstruction was easier, as flaps were planned preoperatively. Besides the exposed bone, the raw area granulated well and it was planned for cover with skin graft. The real problem was insetting the flaps, as there was no tissue to suture the flaps (Fig. 2) and also because of convexity of cranium and thick oedematous flaps to hold the flap in place was difficult.

Technique

As discussed thick flaps, convexity of the skull and friable surrounding tissue all made inset of flap challenging.2 mm X 8 mm titanium (Fig.3) screws were used to inset at the desired position over the exposed bone. As the flaps were thick and oedematous about 5 mm of screw spanned the flap and rest 3 mm spanned outer-table of skull bone. 3 screws were used to fix the flap. Postoperatively patient had no complications. Flaps stuck to the bone firmly and screws were removed after three weeks. (Fig.4)

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Discussion

The use of cortical screws for fixation of flat bones is well known. But the concept of screws for insetting scalp flaps is new and we could not find the literature for the same in our search.

Fig.1: Necrotising fasciitis of scalp after repeated debridement and skin flaps elevated

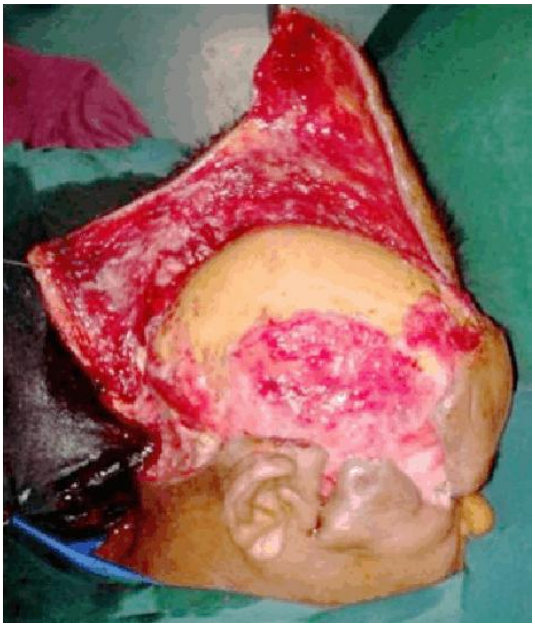


Fig. 3: Flaps inset using 2X8mm cortical screws

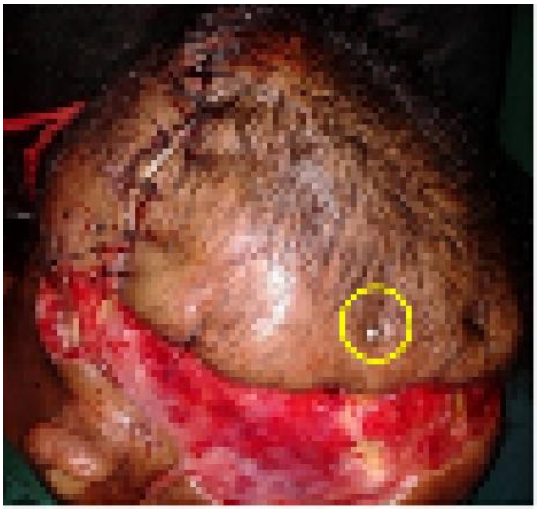


Fig. 2: Wound ready for cover with exposed skull bone and friable granulation

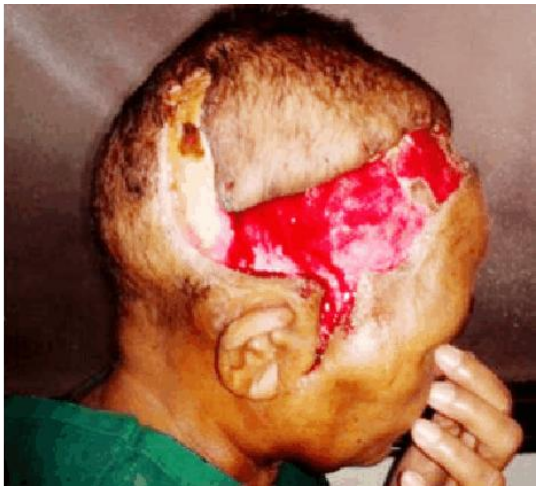
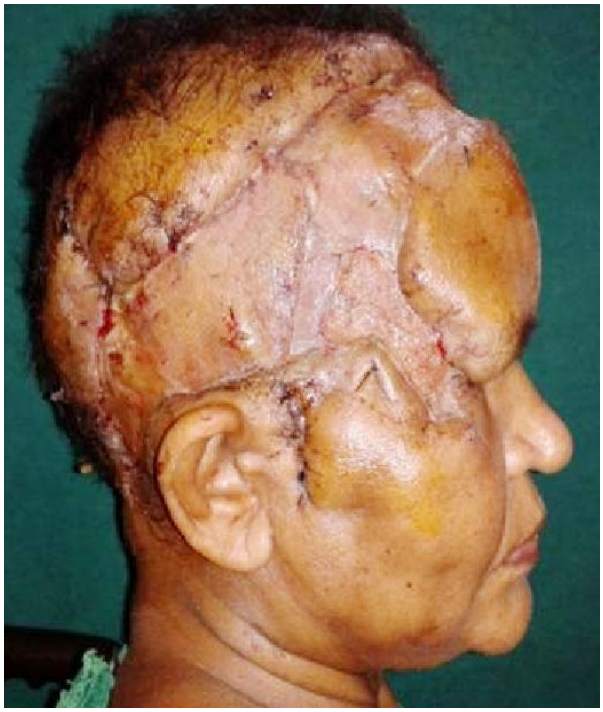


Fig. 4: Post op picture after wound healing



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