Chapter

Infections in Neurosurgery and Their Management

Seyed Arad Senaobar Tahaei, Seyyed Ashkan Senobar Tahaei, Zoltan Mencser and Pal Barzo

Abstract

Surgical site and postoperative infections are common problems in surgical wards and treating them can be challenging and very complicated. It is important to understand different types of postoperative infections and their best management. In this chapter we try to emphasis on infections which are occurring in neurosurgical units and how to approach them. Foreign body infection is another challenge that happens in neurosurgical units, and it is vital to recognize these infections in time and start the treatment as soon as possible. Atypical infections occurrence is low therefore this problem is not addressed often in textbooks or in the literature, therefore atypical infections will be discussed in this chapter too. By discussing the most common postoperative complications and their best management profile, the authors here will try to widen the perspective of readers on infections in neurosurgical units in order to understand this problem better. Untreated infections or poorly treated infections can lead to sepsis and catastrophic results.

Keywords: post-operative infection, neurosurgical infection, CNS infections, septic cerebral embolization, subdural empyema, spondylodiscitis, epidural abscess, meningitis, atypical CNS infections, spinal infections

1. Introduction

1.1 General aspects

Central nervous system (CNS) infections are challenging in terms of correct and on time diagnosis, therefore treating them are quite complicated and require correct understanding of its origin, type of infection and severity of it. Here in this chapter, we will be discussing the most common CNS infections within the field of neurosurgery as well as their best treatment and patient management.

As a known fact, CNS infections can cause very mild to severe signs and symptoms as well as neurological deficits if left untreated or not treated properly. Identification of the primary source of infections are very important, as quite often seen, the source can be infected foreign bodies, or an infection originating somewhere else in the body. In surgical units, wound contamination and deficiencies in correct post-operative patient care are other common sources of infection.

In this chapter we will be discussing infectious diseases which are commonly seen in neurosurgical wards or infections which require neurosurgical

interventions, therefore a vast majority of CNS infections which do not require surgical management and-or not occurring in neurosurgical wards are not discussed here.

Various risk factors contribute to the occurrence of infections, general risk factors such as existing co-morbidities (Diabetes mellitus (DM), Anemia, AIDS etc.), the use of immunosuppressant drugs, chronic existing infectious diseases and abnormalities of the immune system are among most common risk factors. Factors such as specific patient skin bacterial composition and local bacterial resistance patterns play an important role in post-operative surgical site infections. Indeed, the extend of aseptic practice and use of proper prophylactic antibiotics before/during surgery is another factor contributing to the occurrence/prevention of post-operative surgical site infection. However, there are studies where indicate that prophylactic use of antibiotics before surgery increase the rate of post-operative infections.

To minimize the risk for post-operative infections, it is recommended that a strict patient screening to be done in order to minimize risk factors which contribute to infections. Patients with higher blood glucose levels are monitored and their glucose profile and therapy are modified if needed, to achieve an acceptable blood glucose level. Patients on immunosuppressant drugs need greater attention pre-operatively and immunosuppressant drugs should be discontinued temporarily (if no contraindications) to minimize the risk of post-operative infections. Anemia should be corrected if present at the time of admission and screening for multi resistant bacteria should be done for every patient which is planned to undergo an elective surgery. All patients, regardless of their immune system and microbiological status should be given a proper Betadine bath the morning before the surgery, and proper prophylactic antibiotics should be administered intravenously, about 20 minutes before the skin incision is made. For longer procedures it is recommended to administer the prophylactic antibiotic once more while the surgery is going on. Proper wound closure, maintaining a good blood flow to the surgical site and proper fixation of existing wound drains with respect to the aseptic technique drastically can decrease the post-operative surgical site infection rate.

1.2 Pathogenesis

Understanding the pathomechanism of CNS infections and infection routes are necessary for proper diagnosis and proper treatment therefore in this section the basics to understand these infections are discussed. Since viral infections are not common in neurosurgical wards, and post-operative infections are mainly bacterial infections, we will be discussing the bacterial pathogenesis of the most common CNS infections here.

In general, bacterial infections reach the CNS by means of the hematogenous spread, but usually the organisms which are colonizing on the surface of the mucosal membranes of the nasopharyngeal cavities are responsible for most of the bacterial pre-operative infections of the CNS. These organisms can find their way to the nasal sinuses and then via the sinuses to the meninges and intracranial cavity. The blood brain barrier (BBB) is a very effective to repel and reduce the penetration of unwanted substances and organisms to the intracranial cavity, but sustained bacteremia and inflammatory and cytotoxic mediators of these organisms cause damage to the BBB and this in turn causes increase permeability of the BBB which is in fact the reason why these pathogenic organisms progress further and end up in the subdural and subarachnoid spaces. In most of cases the intracranial infections are originating from the adjacent ongoing inflammatory processes such as mastoiditis, otitis media, sinusitis and rhinitis [1].

The mucosal membranes in the oral and nasal cavities are lined with different types of bacteria in high quantities which are the normal flora of these mucus membranes, these organisms usually do not cause any CNS infections unless immunodeficiencies are present or these organisms find their way into the intracerebral space and start colonizing there. Skull base fractures are usually causing cerebrospinal fluid (CSF) leakage and this in return is facilitating the migration of bacteria from these adjacent places into the intracranial space causing various problems such as meningitis, cerebritis, intracerebral abscess, fistulas and subdural empyema formation. It is of great importance to mention that infection and inflammation of distant or adjacent organs can also be causing septic cerebral embolization which can be very challenging and hard to identify and differentiate from other lesions such as metastatic lesions or tuberculomas. Endocarditis and cardiac vegetations are among the common cause of septic cerebral embolization, but in general any distant or adjacent infection which spreads through the hematogenous pathway and causes septicemia can be causative of septic cerebral embolization.

The mechanism behind spinal infections is somewhat the same and the blood spine barrier which is the equivalent of BBB is having the same role in protection and isolation of the spinal cord. Spinal epidural abscess or empyema formations are mainly caused by hematogenous spread of bacteria, but besides this paravertebral injections, acupuncture and epidural catheters can be direct causes of bacterial colonization and formation of spinal epidural abscess and empyema. If the inflammation reaches the bony structures of the spinal column, then the term spondylitis is used to describe the latter. There are different forms of spondylitis, the most common type being spondylodiscitis which involves the inflammation of the vertebral body and the adjacent disco-ligamental system. Spondylodiscitis are usually complications of spine surgery but spontaneous forms of it in diabetic patients or patients who are on steroid or immunosuppressant therapy drugs can be found. The pathogenesis of these infections is also via the hematogenous spread and even simple infections such as urinary tract infections if left untreated can be a cause for septicemia and advancement of the infection to the spinal column.

1.3 Diagnosis

Diagnosing CNS infections require multiple tests and imaging, performing the right type of modality at the right time plays a crucial role in early and correct diagnosis. At early stages, infections of CNS can lack absolute neurological signs and only general inflammatory signs and symptoms are present, with progression of the infection, milder complains such as headaches and malaise appear and as the infection progresses further neurological signs and symptoms begin to appear as well. Most of the patients complain of headaches which are partly responsive to NSIADs, fatigue, nausea and vomiting, photosensitivity and with progression of the infection epileptic seizures and paresis can occur. In patients with spinal epidural abscess or severe spondylodiscitis, paraparesis with back pain which radiates to lower extremities can be the very first signs and symptoms of patients.

Depending on the patient's history, physical examination, signs and symptoms and laboratory findings, if a CNS infection is suspected then immediate action to confirm diagnosis should be done by performing supplementary tests such as imaging and lumbar puncture if necessary. CSF cell count as well as CSF biomarkers (such as IFN-γ, TNF-α, IL-2, IL-6, CD8, MIF, NfH-SM135, GFAP-SM126, S100B) analysis are necessary to confirm the presence of inflammatory process in CNS [2], but since these CSF findings in most of the inflammatory states are similar, a differential diagnosis becomes challenging based on the CSF cell analysis therefore further steps such as image acquisition is needed to be able to proceed further. The

physical findings such as meningeal irritation signs (Brudzinski's and Kernig's sign) and photosensitivity are not enough for the establishment of any type of CNS infection; even in cases where beside the above mentioned signs and symptoms, pyrexia and increased inflammatory markers are present, a definite diagnosis of CNS infection cannot be made, because conditions like subarachnoid hemorrhage (SAH) can have the very same clinical findings, and the origin of pyrexia or elevated inflammatory factors can be something rather than the CNS. For establishing a diagnosis of CNS infection imaging techniques are necessary since without it other specific conditions such as SAH cannot be surely ruled out (despite positive CSF cell count for red blood cells (RBCs) as lumbar puncture (LP) can be traumatic) and abnormalities such as subdural empyema, cerebral abscess or fistulas cannot be detected.

MRI scans are the gold standard when it come to the imaging of the CNS, since the resolution and clarity of the MRI scans for soft tissue detection are much better and higher than CT scans, their use are more prominent in detection of CNS infections. CT scans are also capable of detecting abnormalities such as subdural empyema or cerebral abscess, but since the radiologic features of these conditions on CT scans are not very specific and very similar with other pathologies, once the suspicion of subdural empyema or cerebral abscess is raised, a contrast enhanced MRI should be done to confirm or rule out the diagnosis. In case of the spinal column infections and epidural abscess or empyema in the spine, MRI is the preferred choice due to its capability of detecting diffusion restriction both in bony structures and soft tissues as well as having a higher sensitivity and specificity for detecting lesions in the spinal canal.

2. Meningitis and its treatment

Meningitis is the term used to describe the inflammation of the meninges anywhere in the neural axis. The inflammation can be caused by viral, bacterial or even fungal pathogens, sterile meningitis is a form of inflammation of the leptomeninges where the patient is clinically having symptoms of meningitis, but leukocytosis is not seen in CSF analysis and no pathologic pathogen can be cultured from the CSF. The most common route of infection is usually the spread of pathogens via the hematogenous pathway after upper respiratory tract infections as well as transmission of bacteria from adjacent ongoing infections via the emissary veins from the nearby structures to the meninges (e.g. sinusitis, mastoiditis, odontogenic infections, otitis media), intra or post-operative contamination of the intracranial structures (e.g. after neurosurgical interventions) is another common way of transmission for these pathogens, skull base fractures are also a source for bacterial inoculation and can cause meningitis. In the following section we will be discussing the most common bacterial and viral pathogens causing meningitis.

2.1 Bacterial meningitis

Bacterial meningitis is a serious CNS infection which can be life threatening if left untreated or not treated properly. Various pathogens can cause meningitis in various age groups, in this section for the sake of simplicity we will be describing the most common adult pathogens and their treatments in details.

The common pathogens causing meningitis are, *Streptococcus pneumoniae*, Neisseria meningitidis, group B Streptococcus spp., *Listeria monocytogenes*, and Haemophilus influenzae. *Streptococcus pneumoniae* is usually causing meningitis in adults and children, neonates and infants are generally not infected by this

microorganism. Patients who suffer from skull base fractures and have CSF leaks often develop meningitis due to S. pneumonia inoculation [3]. This so-called community acquired bacterial meningitis (CABM) is considered as a rare pathology with high mortality and morbidity rates. The prevalence of this disorder has decreased significantly in the past decades due to administration of conjugate vaccines and more efficient diagnostic methods and treatments. The most comm pathogens causing CABM are *S. pneumoniae*, N. meningitidis, L monocytogenes according to a study published by Tubiana et al. in 2020 [4].

The diagnosis of CABM is based on clinical findings, CSF analysis and complete blood count (CBC) findings, MRI scans can be useful for obtaining more information about the extent and severity of meningitis but not necessary for establishing the diagnosis. **Figure 1** shows alterations on brain MRI scans in a patient with severe meningococcal meningitis.

Once the diagnosis is confirmed, empirical broad-spectrum antibiotics based on local bacterial resistance should be started and afterwards modified based on antibiograms. For initial treatment third generation cephalosporins [4] alongside amoxicillin or ampicillin to cover treatment for L monocytogenes are recommended and once antibiotic susceptibility results are available then targeted therapy should be started with agents having higher CSF penetration; Steroids and C5 antibodies are also recommended, in case the ongoing infection is not responding well to systemic antibody treatment [5].

2.2 Post-operative

Post-operative meningitis or post neurosurgical meningitis (PNM) is a complication of cranial surgery, and its rate of occurrence is depending on multiple factors. We will be discussing the most common factors contributing to the occurrence of postoperative meningitis and its treatment. Surgeries are done in aseptic environment, careful disinfection of the surgical site and keeping the aseptic environment while performing the surgery is the most important factor in preventing postoperative meningitis. Studies have shown that shaving hair within the surgical site has little to no effect in decreasing postoperative infections [6], but regardless of this fact it is highly recommended to have surgical site free of hair in order to

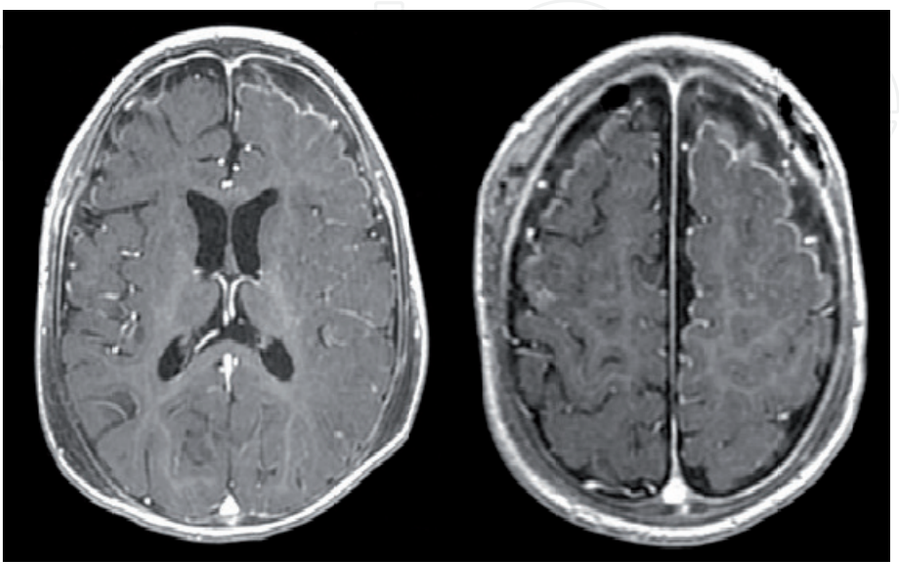

Figure 1.
Diffuse contrast enhancement of the arachnoid membrane and dura bilaterally in the fronto-temporal region is seen in a patient with meningococcal meningitis. The Arachnoiditis is a complication and sequel of untreated or very severe aggressive meningitis.

achieve better and easier access to the site. Prophylactic antibiotic administration is also an important key in preventing postoperative infections, however its use can be controversial as there are studies indicating that prophylactic antibiotic administration before surgery has little to no effect on preventing post-operative infections. The use of administration of antibiotics are usually depending on the existing local bacterial resistance patterns, individual skin flora, type and location of the surgery.

Usage of broad-spectrum antibiotics for cranial surgeries are not recommended simply because their use has not shown any advantage over other antibiotics; Cefazolin has been used for many years now for prophylactic purposes in cranial surgery. If multi resistant *Staphylococcus aureus* (MRSA) is present in the institute where the procedure is carried out or if the skin floral composition of the patient includes MRSA, then administration of Vancomycin is recommended supplementary to Cefazolin [7]. Since implanting foreign bodies both in cranial and spinal surgeries are quite often, it is important to handle the instruments and implants with care to maintain a fully aseptic environment.

One of the important factors contributing to prevention of the postoperative infections is wound closure and surgical wound management; An efficient watertight wound closure ensures proper anatomical closure of layers, minimizing the risk of infections in deeper layers and by proper management of the surgical wound the risk of superficial wound infection can be reduced drastically. It should be noted that patients scheduled for elective surgery should be screened for the presence of multi resistant bacteria in their flora. Patients with multi resistant bacterial composition should be isolated before and after surgery in order to decrease the transmission of the multi resistant bacteria to other patients and the medical staff. Disposable protective clothing should be used by medical staff in order to minimize the risk of transmission.

If signs and symptoms of surgical site wound infection are seen, a CBC and superficial and deep wound sampling for microbiological culturing are necessary. Empirical antibiotic treatment should be started and then modified based on the antibiogram results of culturing. After cranial surgery if signs and symptoms of meningitis are present, then LP should be performed for analysis. Slight elevations of WBC in CSF (up to 50 cells mm3) usually do not require prompt antibiotic treatment, symptomatic treatment for headache, nausea and vomiting and hydration are recommended, leukocytosis should be controlled and if a rise in WBCs count is seen then antibiotic treatment should be started. For values above 50 cells per mm3 empirical antibiotic should be considered and then modified based on culture results if needed. If severe leukocytosis in CSF is seen (above 1000 WBCs per mm3) or if systematic treatment is not achieving desired results, intrathecal antibiotic administration should be considered. Empirical treatment for PNM should include Vancomycin for the coverage of staphylococci and P. acnes, as well as a cephalosporin or carbapenem to cover gram negative bacteria and in particularly pseudomonas [8].

Postoperative meningitis after cranial surgeries can cause local osteomyelitis at the level of surgery and/or subdural empyema or cerebral abscess formation. These complications will be discussed in the upcoming sections.

2.3 Viral

Viral meningitis is not a common finding in neurosurgical wards, and its route of infection and treatment is usually different from PNM and bacterial routes, therefore this topic is not relevant to our discussion, and it will not be discussed here.

2.4 Sterile

Sterile meningitis is defined by irritation of the meninges without the presence of pathological organism. In sterile meningitis the cause of meningeal irritation is usually SAH and circulating red blood cells in the subarachnoid space which can damage the arachnoid granulations and cause meningeal irritation. Clinically signs and symptoms of sterile meningitis are similar to bacterial meningitis in terms of neck stiffness, photophobia, nausea, vomiting and headaches, but leukocytosis or pyrexia are not accompanying the above-mentioned signs. Damage of the arachnoid granulations by the RBCs can cause non-resorptive hydrocephalus following a SAH or a traumatic brain injury with intracranial hemorrhage.

If sterile meningitis is suspected clinically then a LP is performed for CSF analysis. CSF analysis generally reveals elevated number of RBCs with normal or slightly elevated WBCs and normal protein and glucose content. It is important to mention that the protein and glucose content of the CSF can be affected by other factors but in general CSF analysis in sterile meningitis reveals elevated RBCs count. Treatment of sterile meningitis in mild cases is just symptomatic treatment for headaches, nausea and vomiting whereas in moderate to severe cases, CSF drainage is necessary to wash out the RBCs in the subarachnoid space and clear out the CSF. Lumbar drains for continuous CSF drainage are used if there are no contraindications. External ventricular drains (EVD) can be used to clear out the CSF if a lumbar drain is contraindicated or cannot be used for any reason.

If Sterile meningitis is not treated, non-resorptive hydrocephalus occurs due to the fact that RBCs damage the Pacchionian granulations and CSF reabsorption becomes impaired. In these cases, hydrocephalus is treated by placing a ventriculo-peritoneal (V-P) shunt to divert the extra amount of CSF; if the abdominal cavity cannot be canulated due to ongoing inflammatory process or previous abdominal surgery and severe adhesions, then ventriculo-atrial or ventriculo-pleural shunts can be a substitution for the V-P shunt system.

3. Subdural empyema, cerebral abscess and septic cerebral emboli

Subdural empyema is the accumulation of pus in the subdural space which is usually a complication of cranial surgery and untreated postoperative meningitis. Purulent meningitis is another cause of subdural empyema, since primary purulent meningitis infection's rate has decreased drastically due to vaccinations and early diagnosis as well as antibiotic treatments, we will be focusing on postoperative meningitis and subsequently subdural empyema. Other pathologies such as chronic sinusitis, otitis media and mastoiditis, if left untreated contribute to formation of subdural empyema and mostly cerebral abscess formation. Odontogenic sources are also important to be mentioned, as often poor dental hygiene or invasive dental procedures are the origin of subdural empyema or cerebral abscess formation. Immunocompromised patients have a higher susceptibility for postoperative and primary infections, therefore they must be treated with care and normalization of their immune system prior to surgeries is required to prevent postoperative complications, for instance in cases where patients are on prolonged immunosuppressant drugs such as steroids due to chronic diseases, or other immunosuppressive drugs for treating autoimmune diseases, reduction of dose and even a complete halt of treatment for a temporary time should be considered if doing so does not interfere with the course of surgery or patient's primary treatment, reducing the dose of immunosuppressant drugs can be very effective in decreasing the PNM and in general postoperative infections.

Accumulation of pus in the subdural spaces irritates the meninges as well as the arachnoid membrane causing arachnoiditis, this in return can cause irritation of the cerebrum and cause cerebritis. If this process is not disrupted properly and intime, then cerebral abscess formation is occurring. With time as the abscess is maturing, it's wall thickens, and it gets bigger and bigger. The symptom presentation of patients can be very different and vary in a vast range, patients can have mild to severe meningitis signs or severe epileptic seizures and signs of increase intracranial pressure. In cases where an ongoing meningitis is not healed completely and it becomes chronic, the patient's signs and symptoms improve temporarily, but meantime the ongoing chronic cerebritis is leading to abscess formation. Depending on the site of abscess formation severe symptoms such as seizures, hemiparesis, agitation, aggressiveness and even loss of consciousness can occur (in accordance with lesion localization neurological deficits are present). **Figure 2** demonstrates a right sided temporal lesion with rim enhancement, perifocal edema and central diffusion restriction. The patient developed a sudden left sided hemiparesis due to the perifocal edema, and acute drainage and abscess excision was done to minimize neurological damage and complications.

Treatment of subdural empyema and cerebral abscess can be surgical or conservative, depending on their size and symptoms of the patient. Smaller abscesses or subdural empyemas can be treated with systematic antibiotics if they are not causing severe neurological deficits which are acute emergencies, such as hemiparesis, loss of consciousness, decreased arousal state and uncontrollable seizures.

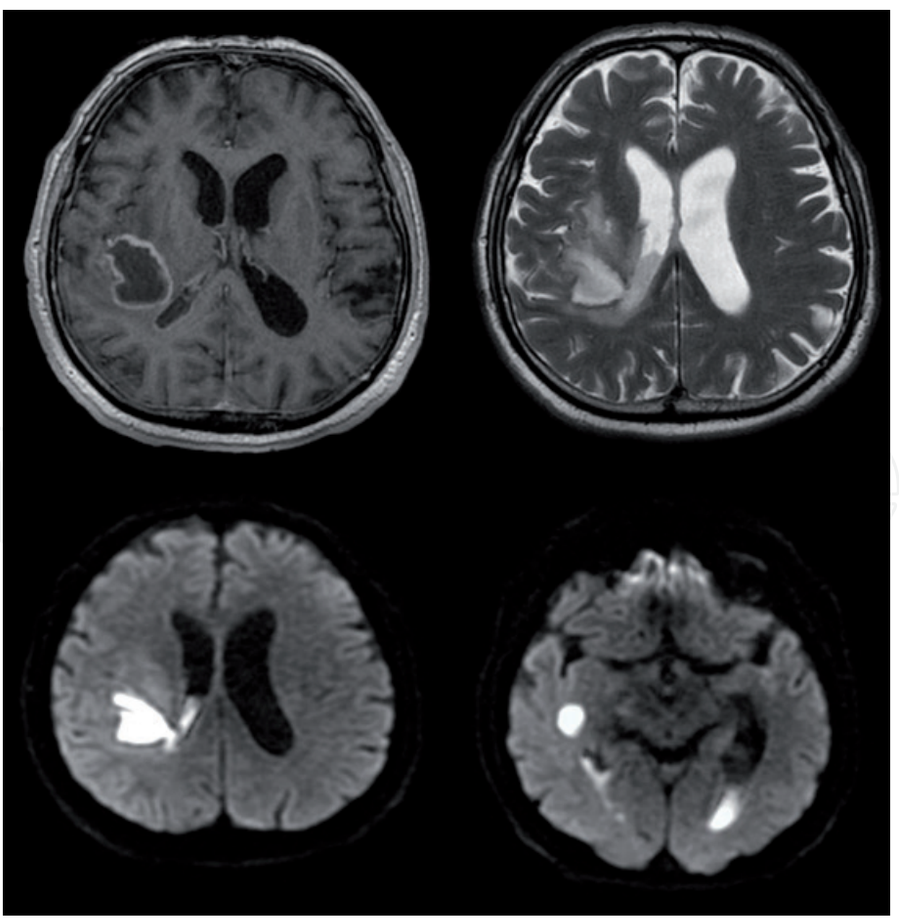

Figure 2.
Rim enhancement on T1 imaging (upper left) and perifocal edema on T2 (upper right) scans in this case are indicative for brain abscess. DWI sequences (lower images) show diffusion restriction in the areas as well as in the right lateral ventricle. It is to be noticed that the occipital horns of the lateral ventricles are also filled with pus and diffusion restriction can be seen there as well (lower right image).

If the above-mentioned symptoms are present, regardless of the size, surgical drainage and systemic antibiotic therapy should be started as soon as possible. In other scenarios where the mentioned symptoms are absent and the size of the abscess is not causing mass effect and midline shift, then proper antibiotic treatment can be the first line treatment, and if it fails or patient deterioration occurs, then surgical removal and drainage should be considered.

Figure 3 demonstrates a severe case of odontogenic subdural empyema which required immediate surgical drainage due to mass effect of the empyema and the neurological status of the patient. Multiple surgeries had to be carried out to achieve an acceptable level of drainage. In **Figure 4** post-operative scans show a significant reduction in contrast enhancement and no diffusion restriction can be seen on the DWI sequence. Surgical treatment was followed by intensive intravenous antibiotic treatment (intravenous Vancomycin, Ceftriaxone and Metronidazole).

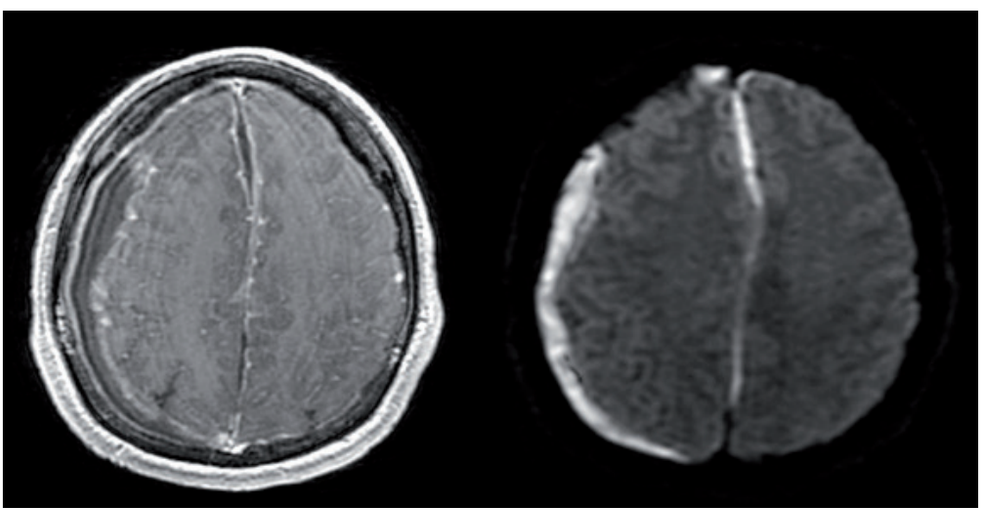

Figure 3.
Contrast enhanced T1 MRI scan (left side) shows contrast enhancement on the arachnoid membrane and dura diffusely on the right hemisphere. Next to the falx cerebri similar alteration are seen and cavitation between falx and dura is noticeable. On the right sided image, we can see diffusion restriction in the hypointense areas seen on T1 sequences. These alterations show accumulation of pus in the subdural space.

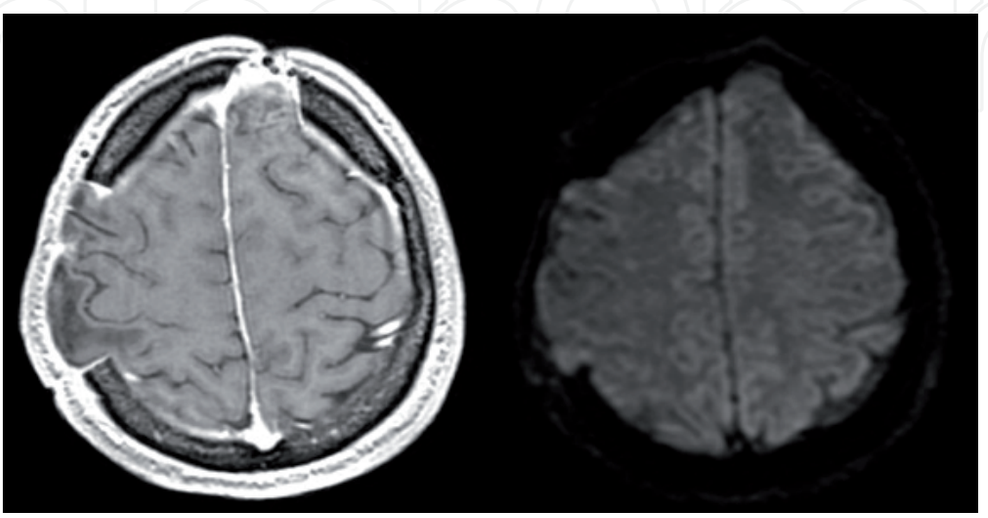

Figure 4.
Post-operative contrast enhanced T1 scan (left image) showing a significant decrease in contrast enhancement; Craniectomy site on the right fronto-temporal region and a burr hole on the left frontal region can be seen. DWI scans show no diffusion restriction.

Septic cerebral emboli are not common findings, and their diagnosis is quite challenging as they often resemble tumor like masses. Septic emboli usually originate from a distant organ via hematogenous spread and after seeding in the CNS they cause abscess formation. Cardiac vegetations are the most common cause of septic cerebral embolization and patients who have gone under valve replacements are at a greater risk of developing cardiac vegetations and consequently septic cerebral embolization. Differential diagnosis is the key point of a proper treatment here and the course of disease development plays a crucial role in differentiating septic cerebral emboli from other pathologies such as tumor masses or granulomas. A sudden onset of signs and symptoms with intracranial lesions should alert the physician for possibility of septic cerebral embolization and if ongoing inflammatory or infectious diseases or comorbidities such as DM, autoimmune disorders, existing mechanical heart valves are present then the probability of septic embolization significantly increases.

4. Foreign body infections

Implantation of foreign bodies in neurosurgery is quite often and so is their complications, but it should be taken into consideration that not all the foreign body implantations come with high infection rates. Aneurysm clips, coils and bone cement implantation have a much lower infection rate in comparison with shunt implantation, screws and rods, EVDs, cranioplasty flaps and deep brain stimulation (DBS) stimulators and electrodes. In both spinal and cranial surgery, there are a variety of pathologies which require implantation of foreign bodies for treatment, in this chapter we shall discuss the most common types of foreign body implantation which can result in a higher infection rate.

4.1 Cranioplasty flaps

Cranioplasty is defined by replacing a missing bone flap, either by means of 3D printed material, autologous bone graft, bone cement or titanium mesh. Regardless of indications for performing craniectomy followed by cranioplasty, the infection rates for this neurosurgical procedure compared to the other procedures is higher. In a broad-based study in 2019 by Ying Chen et al., it was revealed that after craniotomies (6.58% Infection rate), cranioplasties had the highest infection rate (5.89%) in neurosurgical procedures [9]. Infections in cranioplasty procedures can occur early or late in the post-operative period. Early onset of infection usually appears with symptoms of meningitis and quite often wound oozing and surgical site infection is seen as well. PNM is a common complication after neurosurgical procedures, and this is also true for cranioplasty procedures too, if the symptoms cannot be treated by antibiotics, and the bone flap is the source of infection with or without wound oozing, then the flap should be omitted as soon as possible to prevent subdural empyema or abscess formation. Once the ongoing inflammatory process is treated after removal of the bone flap, a minimum of 2 months is recommended before a newer skull reconstruction surgery is performed.

4.2 EVD

External ventricular drains may be the most frequently used implant in neurosurgery. There are different types of ventricular drains with different impregnations which offer a lower risk for EVD related meningitis and ventriculitis. Antibiotic coated or ionized silver particles impregnated EVDs lower the risk of EVD related

infections similarly to the administration of intraventricular antibiotics via the EVD, however there are multiple factors contributing to the infection of EVDs, such as existing infection, lack of proper tunneling of EVD, multiple CSF sampling, leakage and improper isolation of the surgical site by the nursing staff [10]. EVDs quite often are inserted at bedside by neurosurgeons in intensive care units (ICU), where the environmental bacterial composition is quite different and more resistant and severe in terms of contagiosity compared to other sections of a hospital, therefore there is a higher chance of EVD infection if it is placed at the bed side and in ICU and this can be prevented by performing this procedure in an operation room where sterility is maximized. If an EVD becomes infected or colonized, then it should be removed, and a newer drain should be inserted in a different location if needed.

4.3 Shunts

Shunting CSF is one of the common procedures done routinely in neurosurgical wards, different variants of shunting are used for divergence of the CSF and they all include implantation of foreign bodies, therefore having similar high infection rates postoperatively. As V-P shunts are the most common used variant of the CSF divergence, we will be discussing only the complications of V-P shunts. The V-P shunt infection occurs at a rate of 5–10%, *Staphylococcus epidermidis* accounts for the majority of infections (60%) as well as *Staphylococcus aureus* (30%). The shunt

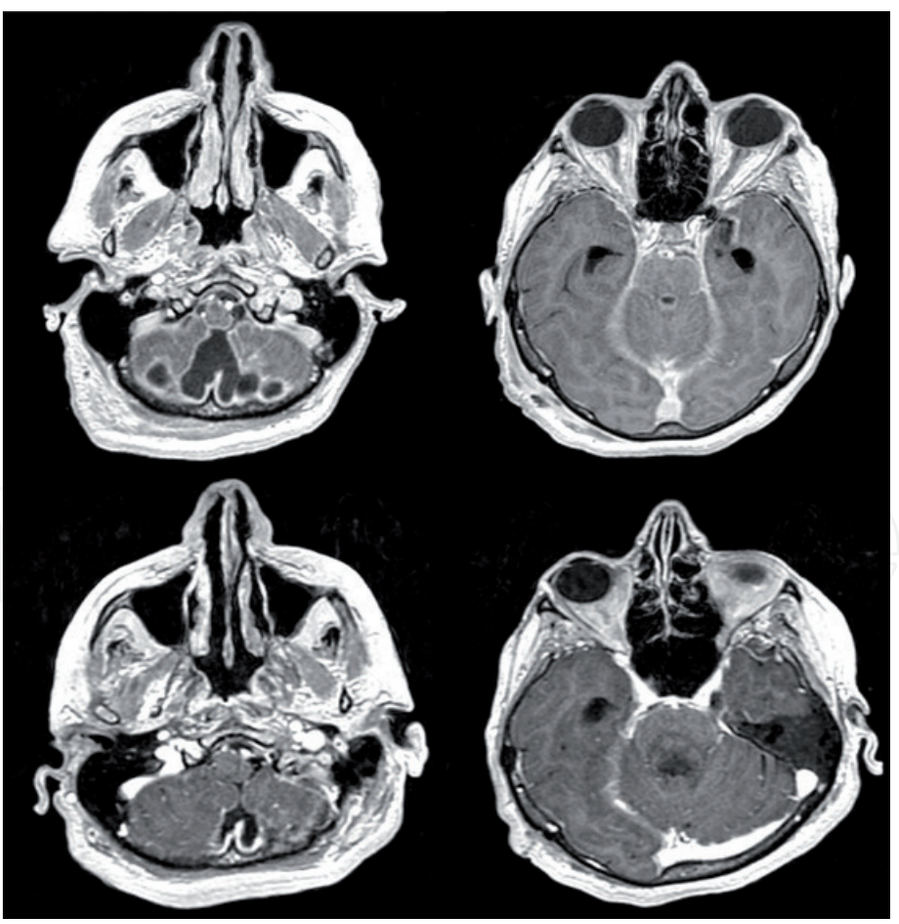

Figure 5.
Contrast enhancement on T1 scans (upper images) can be seen, rim enhancement and hypointense central compartment in the cerebellar region reveals multiplex abscess formation (upper left image). Contrast enhancement and dilation of aqueduct is seen, and this is indicative of intraventricular involvement of inflammation (upper right image). Lower pictures show regression of the inflammatory lesions after aggressive antibiotic treatment and removal of the V-P shunt system. Minimal contrast enhancement is seen between the two cerebellar hemispheres (lower left image).

infections usually occur within the first 6 months of implantation, the first month having the highest probability [11]. Factors such as contamination of shunt with skin flora during implantation, leakage, improper wound closure and ongoing intracerebral or abdominal inflammation at the time of surgery are the common causes of V-P shunt infections within the first month of implantation, long term complications of shunts are usually due to secondary infections. Peritonitis can be a severe complication of V-P shunt infection as well as a source for V-P shunt infection. Odontogenic infections often cause intracerebral inflammation and with the presence of V-P shunts, this can cause shunt failure and serious complications. **Figure 5** shows a case of V-P shunt infection after 3 years of implantation due to an ongoing untreated dental infection which had caused severe ventriculitis and multiple abdominal infected granulomas.

4.4 Deep brain stimulation and spinal cord stimulation

DBS surgeries are often used for treating movement disorders such as Parkinson's disease, dystonia, and essential tremor as well as psychiatric disorders such as obsessive–compulsive disorder. In the other hand as cases of chronic lumboischialgia and failed back surgery syndrome are rising, spinal cord stimulation surgeries are getting more attention. Both procedures involve implanting electrodes and neuromodulators in different sites, therefore these surgeries are carrying a high risk of infection. Infection rates are different in different areas and hospitals, in the literature the infection rate for DBS surgeries can be up to25%. As mentioned before *S. aureus* is the most common cause of neurosurgical infections and DBS and SCS surgeries are not an exclusion of this fact [12].

Intraoperative topical use of vancomycin has shown no advantage in preventing postoperative infections and its use intraoperatively remains controversial, in fact Bernstein et all in 2019 concluded that intraoperative topical use of vancomycin increases the risk of postoperative infection after electrode implantation [12].

Infection of brain electrodes can lead to abscess formation and spinal electrode infection can cause epidural abscess formation. Neuromodulators can also get infected and be a source for septic reactions.

4.5 Screws, rods and cages

The most common implants used in spine surgery are screws, rods and intervertebral cages. Different materials such as polypropylene-polyester, titanium, and polyetheretherketone (PEEK) are used in spinal surgeries and a study in 2019 revealed that the above-mentioned materials among all other materials used in neurosurgical procedures have the highest rate for infection [9]. Fusion surgeries require the removal of the intervertebral disk and implantation of an intervertebral cage, this in turn can cause spondylodiscitis which in turn can cause colonization and infection of the implanted screws and cages. The ongoing inflammation causes loosening of implanted screws, and this will lead to spine instability, therefore in such cases patients need to be immobilized, treated with antibiotics and revision surgery should be done when suitable. In addition, using thoracolumbar spinal orthosis (TLSO) braces can add some degrees of spine stability and fine patient mobilization can be allowed when wearing TLSO. Diagnosing is based on elevated inflammatory parameters on blood tests as well as contrast enhanced MRI scans to visualize the spinal canal and assess the extent of ongoing inflammation.

4.6 Treatment

Foreign body infection requires immediate attention and treatment; if left untreated it can cause severe intracranial or epidural inflammation and severe neurological deficits as well as sepsis and multi organ failure consequently. Diagnosing foreign body infection requires precise imaging, multiple repeated blood tests, culturing and patient examination. Once the diagnosis of foreign body colonization or infection has been made, broad spectrum empirical systemic antibiotic treatment should be started immediately and later on modified based on antibiograms. If the infection is caught in early phases and only mild symptoms are present and imaging modalities rule out presence of abscess or empyema and colonization of foreign bodies are not suspected then systematic or intrathecal antibiotic treatment might be enough to treat the infection, but in cases were abscess or empyema formation is already present on scans or CRP-PCT levels are not normalizing with antibiotic therapy, then removal or revision of the implanted foreign bodies are required.

Infected V-P shunts need to be removed and CSF divergence with EVD is preferred at a different site rather than the primary surgical site. In cases where deep brain electrodes need to be removed, patient management can become very challenging; for example, patients who suffer from Parkinson's disease and are non-responders to oral medication, will have severe disabilities if their neuro pacemaker was turned off suddenly and there was no pacing in the subthalamic nuclei. When infections are properly treated, then permanent electrodes, shunts or any other foreign body should be reimplanted if the patient's status and treatment require so.

5. Fungal and parasitic infections

Fungal and parasitic infections are not as common as bacterial infections in postoperative patients, therefore there is no solid information about fungal infection in postoperative patients in the field of neurosurgery. As a known fact patients who are immunocompromised are having a greater risk for developing any kind of infection, and fungal infections can be seen in these group of patients more often. Indeed, a prolonged antibiotic treatment increases the susceptibility of patients for developing fungal infections and this should be taken into consideration. Mycosis in the CNS can be hard to diagnose as often they resemble tumor masses on MRI or CT scans and their differentiation is quite challenging if the patient does not have any background for infection or does not present signs and symptoms of infection physically or on laboratory findings. A complication of ongoing fungal infection can be mycotic aneurysms which can rupture and be life threatening.

As in any other infectious disease, mycosis of the CNS should be treated immediately based on culture results. Surgical resection of fungal abscesses or granulomas might be necessary in severe cases where antifungal therapy is not yielding positive results or due to the mass effect and midline shift caused by these lesions in the brain.

6. Atypical infections

Atypical infections are quite rare and therefore very misleading, their diagnosis is very challenging and if not diagnosed correctly, the course of treatment can be very different and ineffective. The term atypical applies when the infection is caused by an organism which is not known to cause CNS infection or has not been

reported yet, or infections which happen without any background or any obvious reason. Recently a case of septic cerebral embolus caused by Corynebacterium mucifaciens was described in a diabetic patient, C. mucifaciens is a normal flora of the skin and it can also be found in sterile body fluids [13]. Immunocompetent patients usually have a lower risk for atypical infections, but patients with defective immune system tend to have superinfections and even infections caused by organisms which normally do not cause any pathology. Course of disease development plays a crucial role in diagnosing these atypical infections, for example patients on prolonged antibiotic treatment, steroid treatment or immunosuppressants and in general immunocompromised patients should be considered for atypical strains of bacterial infection. If atypical strains are cultured or isolated in the abovementioned patient categories, they should not be precepted as contamination or false positive results but rather considered as atypical pathogens and they should be further investigated in order to confirm diagnosis.

The other scenario would be when a healthy immunocompetent individual suffers an atypical bacterial infection, this too should not be considered as false positive results or contamination, but rather it should be alarming as most immunocompromised patients are unaware of their condition are considered immunocompetent until such infections come along. This in turn does not mean that if a healthy individual is infected by atypical strains, then a defective immune system is the cause; this simply has to be investigated further to rule out any defects of the immune system and find the origin and primary cause of atypical infection. Healthy immunocompetent individuals can also be infected by atypical bacterial strains without any background or comorbidities playing along.

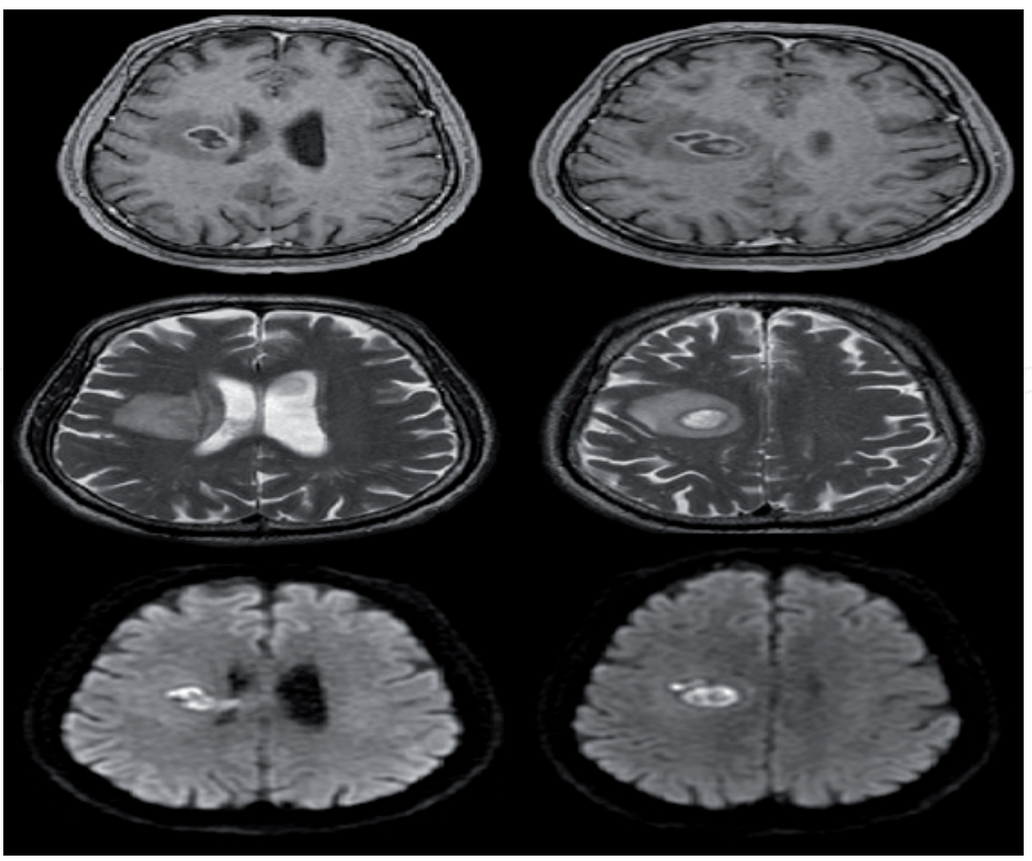

Figure 6.
T1 contrast enhanced images (upper images) show a cystic like lesion with perifocal edema and rim enhancement in the right temporal lobe, at the level of the internal capsule. T2 scans (middle images) reveal the extent of perifocal edema and the fluid content of the lesion. Diffusion restriction can be seen on DWI sequences (lower images) which is a typical finding for cerebral abscess.

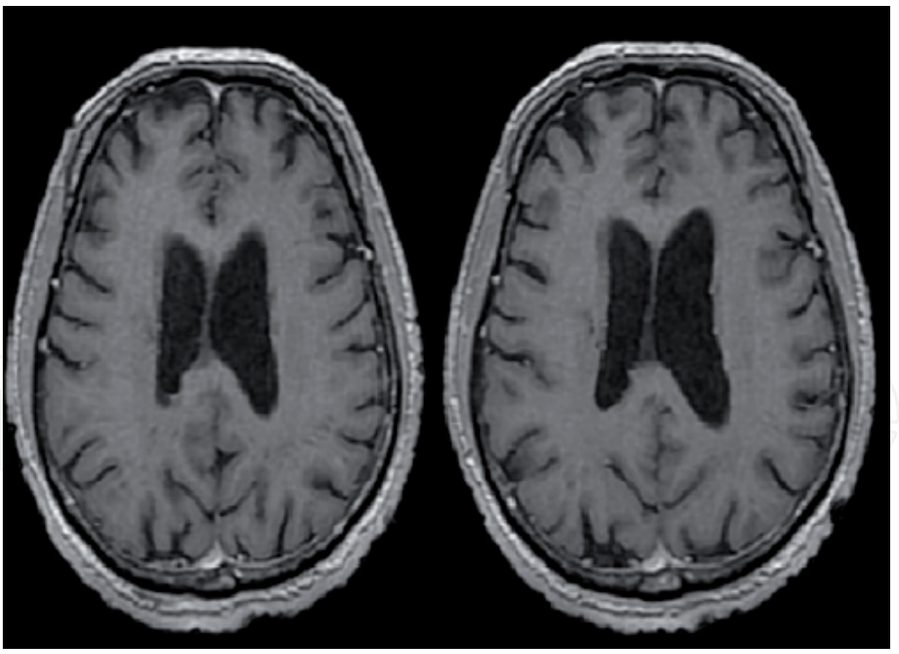

Figure 7.
Complete resolution of the lesion is seen after proper antibiotic treatment. The post gadolinium MRI scan was done for control purposes after 1 year.

Atypical bacterial infections should be reported so that, medical society all over the world can recognize the possibility of infection by these strains in immunocompetent or immunosuppressed individuals. Treatment of atypical strains remains the same as typical strains, except for if standard antibiotic treatment fails to control the infection, a more aggressive antibiotic treatment profile should be chosen.

Figure 6 shows the first ever reported case of cerebral embolus caused by C. mucifaciens by Tahaei et al. The radiographic findings are very typical for cerebral abscess or metastatic tumor lesions, and simply because the relevance of metastatic tumors and brain abscesses are much higher than septic emboli, the possibility of a cerebral septic embolus is often ignored, and they can be misdiagnosed and mistaken for tumors initially. A biopsy confirmed the diagnosis of septic cerebral embolus and proper antibiotic treatment based on antibiogram results were started after empiric treatment. In **Figure 7** the complete resolution of the septic embolus is seen after proper antibiotic treatment.

7. Spinal infections

They represent about 4% of all cases of osteomyelitis and 2–7% of all musculoskeletal infections. The incidence is between 1:20000 and 1:100000 and it has been increasing in the last decades [14–17].

Spinal infections can be extremely destructive and can cause instability and progressive neurological symptoms. Diagnosis of spinal infection is very challenging due to the fact that they mimic other noninfectious degenerative disorders [17].

7.1 Pathogenesis

Spinal infection can develop in three different ways:

- hematogenous spread

- direct inoculation
- spread of infection from an adjacent site

The sources of hematogenous infections are usually the skin, respiratory tract, genitourinary tract, gastrointestinal tract or the oral cavity through bacteremia. The extensive prevertebral venous plexus in the vertebral column provides a sophisticated anatomical background for spreading of bacterial infection. In adults, discitis mostly originates from one of the neighboring endplates, which are necrotized by a septic embolus, while the disc is infected secondarily. Spread from contiguous tissue is rare and mainly occurs in adjacent infections, including retropharyngeal abscess, esophageal ruptures, and infected implants [17, 18].

7.2 Microbial agents

The most common causative agents of spinal infections are *Staphylococcus aureus* and *Staphylococcus epidermidis*, gram negative organisms and in general anaerobe bacteria. In infants the most common agents are *Staphylococcus aureus*, *Streptococcus agalactiae* and *Escherichia coli* and in childhood *Staphylococcus aureus*, Staphylococcus pyogenes and Hemophilus influenzae. *Staphylococcus aureus* is the most common causative agent in hematogenous osteomyelitis in patients of all ages [19]. *Mycobacterium tuberculosis* can cause Pott's disease and skeletal tuberculosis which nowadays are extremely rare. Fungal origin of spinal infection is rarely found and particularly include Aspergillus spp., Candida spp., and *Cryptococcus neoformans* [17].

7.3 Classification of spinal infections

The spinal infections can be classified into the following categories:

a. Pyogenic infections
   i. Vertebral osteomyelitis
   ii. Discitis
   iii. Spondylodiscitis
   iv. Spinal epidural abscess
   v. Facet joint arthritis

b. Granulomatous infections
   i. Tuberculous infections
   ii. Fungal infections
   iii. Parasitic infections

c. Postoperative wound infection

d. Spinal infection in the immunocompromised patients

7.3.1 Pyogenic infections

The source of infections is usually the genitourinary, gastrointestinal and respiratory tract and in one-third of cases, the source is unknown. Patients who are on prolonged steroid treatments or immunosuppressants have a higher risk for pyogenic infections.

7.3.1.1 Vertebral osteomyelitis

Vertebral osteomyelitis most commonly occurs via the hematogenous route. The disease can progress to abscess formation and can involve the paravertebral structures and spinal canal. Risk factors are diabetes, renal failure, rheumatoid arthritis, AIDS, malignancy and old age. The most common bacterial agents responsible for vertebral osteomyelitis are *S. aureus*, Streptococcus sp., *P. aeruginosa*, *E. coli* and Proteus sp.

7.3.1.2 Discitis

This is an infection or inflammation of the intervertebral disc space. The infection starts at the endplates and spreads to the disc secondarily. *S. aureus* is the most common causative agent.

7.3.1.3 Spondylodiscitis

An infection and inflammation of the endplates of the vertebrae, as well as the joining intervertebral disc. It commonly occurs in sepsis, post-tonsillectomy, urinary tract, gastrointestinal and respiratory tract infections. The most common organisms are staphylococci (40–60%) and tuberculosis (20%). **Figure 8** demonstrates a case of active and healed spondylodiscitis in one patient.

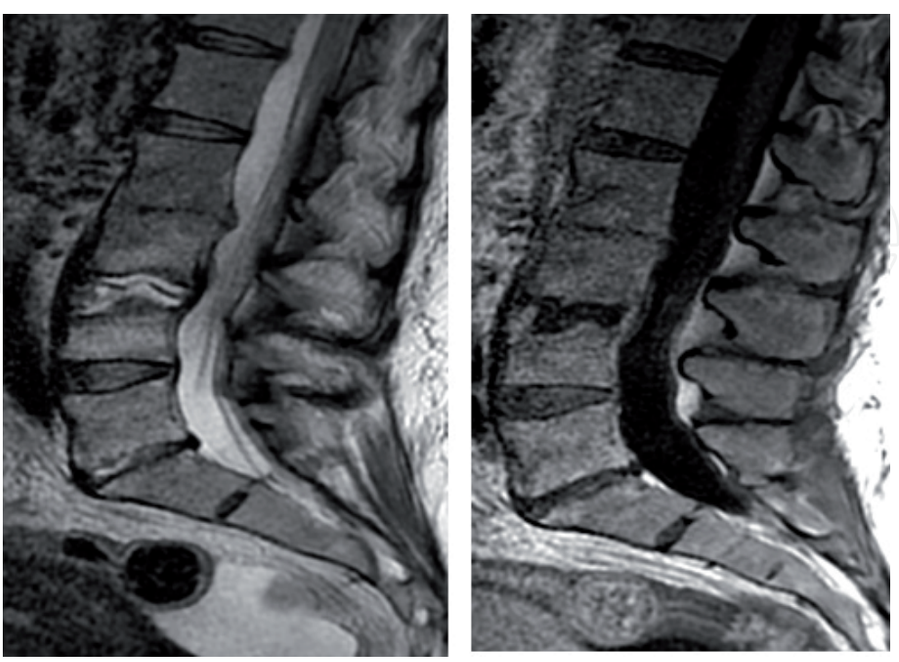

Figure 8.
Active spondylodiscitis at the level of LIII-IV and healed spondylodiscitis at the level of L II-III can be seen on both T1 and T2 sagittal images. Destruction of the disc and ossification of the two adjacent vertebral bodies are noted at the level of LII.

7.3.1.4 Spinal epidural abscess

Epidural abscesses are often complications of invasive spinal interventions, but spontaneous cases can also occur due to hematogenous spread or adjacent infected sites. The risk factors are diabetes mellitus, HIV, osteomyelitis, urinary tract infection, sepsis, soft tissue infections and spinal abnormality. *S. aureus* is the most common causative agent. The infection can come from contiguous areas, through hematogenous spread and from unknown distant locations as well [15, 17, 18].

Pain and muscle spasm are the most common symptoms of spine infection and the patient can be bedridden. In half of the cases, fever is the systemic manifestation of infection and early diagnosis is hindered by the fact that 30–70% of patients with spondylitis/spondylodiscitis do not show any signs of prior infection. Radicular pain and neurologic deficit are rare finding at the beginning but as a complication or progression of the situation serious neurological symptoms can occur [17].

Prevalence of spinal epidural abscess based on localization is 35% in thoracic region and 48% at lumbar region. Laboratory findings include elevated erythrocyte sedimentation rate and C-reactive protein, in about 40% of cases there are total or PMN leukocytosis [19]. X-ray shots are not very informative for diagnosing epidural abscesses, after 3–4 weeks the inflammation starts spreading around and degenerating the adjacent bony vertebra, and it is only then when destruction of bony parts can be visualized on X-ray shots; X-ray does not rule out the presence of a space occupying lesion in the spinal canal and therefore, post Gadolinium MRI scans are necessary to have a precise diagnosis. In cases where MRI scans are unavailable then CT scans or myelography can be done to gain more information. MRI scans remain the gold standard for confirming diagnosis when spinal epidural abscess formation is suspected. Epidural abscesses appear hypointense on T1 sequences and hyperintense on T2 and STIR sequences. Rim enhancement in post Gd scans can also be visualized. In **Figure 9** an epidural spinal abscess can be seen at the level of L IV – SI; Surgical excision and drainage of the abscess was done along with intensive iv. antibiotic treatment.

7.3.2 Granulomatous infection

Infectious diseases of spine which is caused by bacteria, fungi and parasites and it is accompanied by formation of granulomas. Granulomas are mixture of transformed macrophages, matrix and other inflammatory cells. Most cases are due to hematogenic spread of microorganisms to the spinal structures but spread from adjacent infected tissues are also a common pathway of infection [20].

7.3.2.1 Tuberculous infections

The spine is the most common site of skeletal tuberculosis and *Mycobacterium tuberculosis* is the most common causative agent. The lower thoracic spine is the segment frequently involved in tuberculous infections. The infections is a result of past hematogenous foci, contiguous disease or lymphatic spread from pleural disease, it gradually enlarges and spreads to involve two or more adjacent vertebrae by extension beneath the anterior longitudinal ligament or directly across the intervertebral disc.

X-ray shots reveal anterior wedging of two adjacent vertebral bodies with destruction of the intervening disc. On MRI scans post Gd sequences show subligamentous, Dural or discal contrast enhancement whereas T1 sequence shows a hypointense and T2 sequence a hyperintense marrow. Contrast enhanced MRI is the preferred modality of choice and if unavailable then contrast enhanced CT scans are the modality of choice.

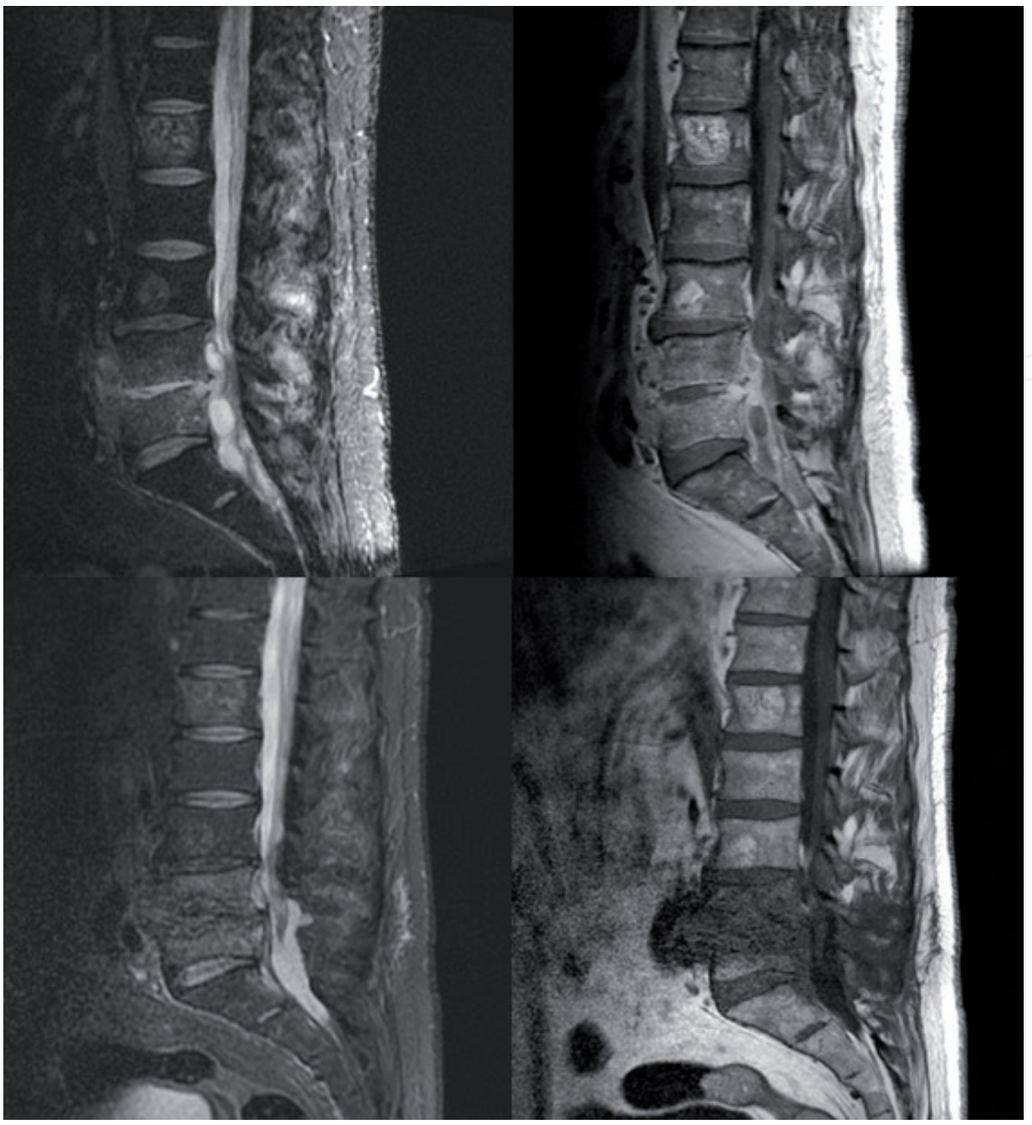

Figure 9.
A case of spontaneous epidural abscess formation, the patient was referred to the neurosurgical word due to progressive paraparesis; lab test results revealed leucocytosis and elevated CRP and PCT values. MRI scans show diffusion restriction (upper left image – STIR sequence) in the epidural region at the level of LIV-SI as well as LIV disc and LIV LV vertebral bodies, the post Gd T1 image (upper right) shows contrast enhancement of the lesion in the periphery. Control MRI scans after 6 weeks (lower images): On STIR (lower left image) sequence significant reduction of diffusion restriction is seen, T1 Contrast scan (lower right image) show no sign of contrast enhancement. The presence of hemangiomas at the level of LI and LIII vertebral bodies are noted.

7.3.2.2 Fungal infections

Fungal infections of the spine are rare and occur mainly as opportunistic infections in immunocompromised patients. They form noncaseating lesions. The common fungal agents causing fungal infections are Candida species, *Cryptococcus neoformans* and Aspergillus species. In fungal vertebral osteomyelitis epidemic fungi, such as Coccidiodes immitis and Blastomyces dermatitidis are the typical causative agents.

7.3.2.3 Parasitic infection

The parasites that have been reported to cause infections of the spine are Echinococcus granuosus (hydatid disease), Toxoplasma gondii (toxoplasmosis) and rarely Taenia solium (cysticercosis). The parasitic spine infection is extremely rare, and few cases have been reported in the literature.

7.3.2.4 Postoperative wound infection

Postoperative wound infections after spine surgeries are common complications and their occurrence rate vary depending on the type and site of surgery. Simpler surgeries such as lumbar microdiscectomy or sequestrectomy have lower rates of postoperative infection (~0.6–3%) whereas more complicated surgeries such as instrumented fusions have a higher occurrence rate (~6–18%). If left untreated or not diagnosed properly and in time, long term complications such as pseudoarthrosis, spinal deformities, chronic pain and even in severe cases sepsis and death can occur. Deep wound cultures accompanied by CBC and evaluation of CRP, PCT and ESR helps diagnosing postoperative SSIs. Picture modalities such as contrast enhanced MRI and CT scans are important in adding vital information for proper diagnosis, but they can also be very misleading [21].

Treatment of postoperative SSIs in spine surgery is no exception than other SSIs, early proper diagnosis, targeted antibiotic therapy and in severe cases surgical debridement and/or revision of surgery is needed to avoid long term complications. Sepsis and septic shock can be catastrophic outcomes of SSIs, therefore prompt response after diagnosing SSIs is a vital part of a good prognosis. Screening patients for multi resistant skin flora and proper bathing before surgery alongside with proper disinfection of the surgical site at the time of surgery and keeping an aseptic environment are modifiable factors which play an extensive role in SSIs. Factors such as prolonged steroid treatment, or use of immunosuppressant drugs, DM, chronic autoimmune diseases, alcoholism, malnutrition and acquired autoimmune defects are contributing to a higher rate of SSIs.

7.3.2.5 Spinal infection in the immunocompromised

As mentioned in Section 3, immunocompromised patients are at a greater risk for developing postoperative infections and spontaneous infections in general. The factors and strategies mentioned in Section 3 management and treating immunocompromised patients are all applicable for spine surgery as well. Patients with immune deficiencies should be considered for rare parasitic, fungal, and bacterial spine infections such as Cryptococcus, Mycobacterium and Echinococcus infections.

Figure 10 shows a case of spontaneous spondylodiscitis in a patient who was on chronic use of methylprednisolone tablets for treating rheumatoid arthritis.

7.3.2.6 Diagnosis of infection

ESR and CRP are both good indicators for determining inflammation in the body. ESR is more specific for tuberculosis infection, whereas CRP is an indicator for any inflammatory condition, including bacterial infections. Procalcitonin (PCT), a promising marker to distinguish between bacterial and nonbacterial infection, shows lower sensitivity than CRP in patients with spinal infection. Identification of the causative organism is essential, and if MRI or CT scans are suggestive of spinal infection then direct CT-guided biopsy or blood cultures should be obtained to identify the causative agent and clarify the diagnosis.

7.3.2.7 Imaging

The characteristics of spinal infection foci on picture modalities are as follow:

- X-ray: endplate irregularities and erosion in the vertebral endplates.
  Disadvantages: It is only after 2 to 4 weeks that the radiographs appreciate

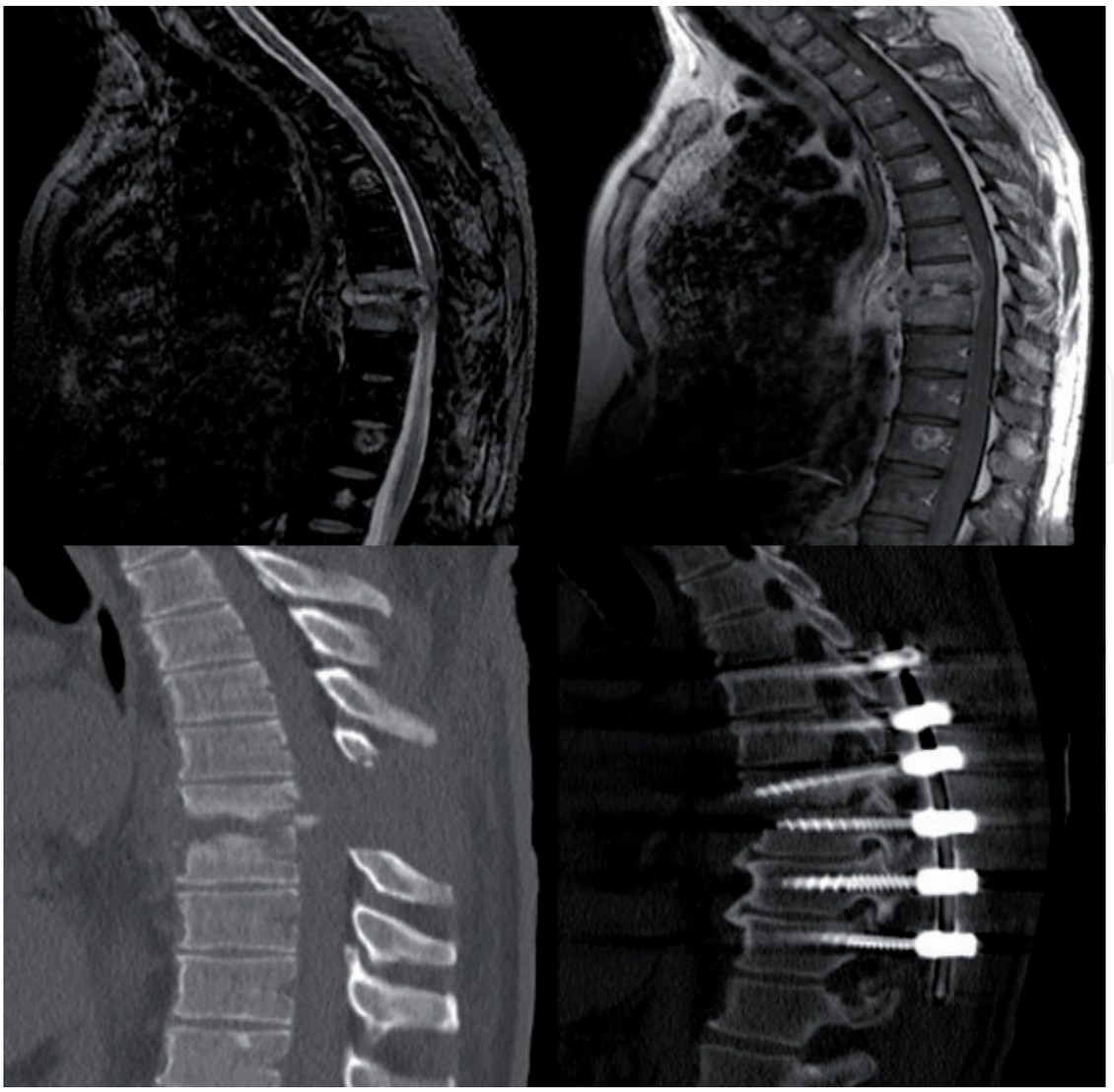

Figure 10.
A 40 years old male patient was brought to the emergency department due to sudden loss of lower extremities tone and severe paraparesis. Lab results were in favor of inflammatory process and the MRI scans have confirmed the diagnosis of spondylodiscitis. The STIR sequence (upper left image) shows diffusion restriction in the Th VIII-IX vertebral bodies as well as in the sub ligamental region which is causing spinal cord compression. Post Gd T1 sequence (upper right image) shows contrast enhancement of the lesion). The patient was admitted for acute surgery and first intraoperative CT scan (lower left image) shows the extent of posterior bony decompression (laminectomy) and the on the lower right image the final postoperative control scan is seen after percutaneous transpedicular fixation was done to achieve stability. It is important to mention that multiple lesions in the vertebral bodies were seen which typically assemble hemangiomas, but due to the complexity of this case and the fact that the patient was on prolonged steroid use, septic emboli and Pott's disease had to be ruled out. This was done by taking samples from the lesions and performing PCR, culturing, and sending samples for histopathological examination. All results were in favor of hemangiomas and an ongoing spondylodiscitis.

any changes. A positive radiograph may help in diagnosis, but a negative radiograph does not rule out the diagnosis of spinal infection.

- CT: Is the best way to demonstrate bone destruction and fragmentation. It can detect early changes like end-plate erosions, much sooner than radiography. It is superior in detecting bony fragments, new bone formation, bony sclerosis, and calcification. It can detect early changes like end-plate erosions, much sooner than radiography.

- MRI: It is considered the gold standard and is the most useful imaging modality in the evaluation of spinal infection. It can detect the form of increased

vertebral marrow water content and micro destruction of trabecular bone. Typical findings in patients with spondylodiscitis are hypointense discs and vertebral bodies in T1-weighted images and hyperintense signals of the same structures in T2-weighted images. Although MRI is the gold standard in diagnosis of spinal infection, there is no pathognomonic finding on MRI that dependably discriminates between spinal infection and possible neoplasm [22].

Being a noninvasive and safe procedure follow-up MRI is good for assessing therapeutic-response and to guide clinical decisions. New MRI methods like diffusion-weighted imaging are useful in spinal cord abscess analysis. Contrast obtained pictures are a must in order to visualize the extent of epidural and meningeal inflammation. Diffusion tensor and fiber-tracking imaging methods are in use for assessing spinal cord integrity in long standing cord compression cases [19].

7.3.2.8 Management of spinal infections

Clinical picture and presentation of spinal infections vary widely, but usually the onset is insidious and axial back pain and spasm are the main symptoms. Fever, chills, weight loss, anorexia and malaise are not always present and neurological deficit presents usually late but may present acutely with epidural abscess formation causing paralysis or cauda equina syndrome. WBC, ESR and CRP are nonspecific, but may be helpful in monitoring the response to treatment.

X-ray and CT scan can show us the bony destruction, it takes a few weeks, but MRI is more sensitive and may show changes, early in the course of the disease. It can include bone marrow edema, endplate irregularity, fluid in the disc space, destruction of bone and adjacent disc, and epidural and/or paraspinal soft-tissue infection and abscess formation.

Nonsurgical Treatment consists of appropriate antibiotic treatment which can result in termination of the infection, but precise bacteriological diagnosis and culturing is required from blood culture or aspirated samples in order to have a target antibiotic therapy which is more efficient. It is important to start antibiotic therapy before significant bone destruction occurs, to avoid any long-standing unfavorable biomechanical consequences and spinal instabilities.

The following steps are required for diagnosing spinal infections correctly early in order to have the best maximized therapy:

1. Biopsy – to identify organism and to obtain its sensitivity (needle biopsy with cultures – percutaneously via transpedicular approach or CT guided biopsy)

2. antibiotics – once the tissue sample is obtained, empirical antibiotic therapy must be started using broad-spectrum antibiotics such as Clindamycin or Rifampicin for at least 6 weeks intravenously and then followed by 6–8 weeks of oral antibiotic treatment. Antibiotic treatment should be modified based on antibiograms if needed, to achieve the best targeted antibiotic therapy efficiency [23].

3. pain medication

4. Thoraco-lumbar spinal orthosis (TLSO) brace – to reduce pain.

Nonoperative treatment is more likely to yield good results if patients are younger than 60 years, having normal immunologic status and the treatment is started early in the course of the disease.

Monitoring response to treatment is crucial and factors like improvement in pain reduction, muscle spasm, general sense of wellbeing, as well as progressive drop in the inflammatory markers like ESR and CRP are good indicators of sufficient treatment. Repetition of MRI scans can be helpful, but in early phases of treatment (within 4 weeks), after starting antibiotic therapy, it can be misleading, due to the fact that the effect of the antibiotic therapy can only be detected on the tissues adjacent to the spine in this period of time. It is recommended to perform post Gd MRI scans after four weeks of continuous iv. antibiotic treatment [24].

When non invasive treatments fail to achieve proper results, surgical treatment needs to be considered in order to control the disease progression. The fundamental goals are drainage of the pus and debridement of granulation and bony stabilization if necessary. Most cases can be managed non-surgically with antibiotics and immobilization, especially if the patient lacks neurological symptoms and if the spine retains its stability. Accordingly, we have to treat the patient surgically when

1. there is progression of disease despite antibiotic therapy

2. spinal instability

3. spinal epidural abscess

4. antibiotic refractory chronic infection [25]

Decompression of neural elements, removal of inflammatory tissue and infected bone (to decrease bioburden) with instrumented fusion can give the best result.

8. Conclusion

Postoperative infections are common and challenging to treat. Understanding the nature of these infectious diseases and their management can increase survival rate and prevent catastrophic results. Treating these infections can be surgical or conservative depending on presenting signs and symptoms and severity of infection. Proper targeted antibiotic treatment alongside surgical interventions are necessary in most cases to achieve the best result. Untreated or poorly treated cases lead to septic reactions and multi organ failure. Screening patients for immunodeficiencies, comorbidities, and specific individual bacterial patterns can significantly reduce postoperative infection rates.

Conflict of interest

The authors declare no conflict of interest.

Author details

Seyed Arad Senaobar Tahaei[1]*, Seyyed Ashkan Senobar Tahaei[2], Zoltan Mencser[1] and Pal Barzo[1]

1 Department of Neurosurgery, University of Szeged, Hungary

2 Department of Pharmacognosy, University of Szeged, Hungary

*Address all correspondence to: red.stradivarius@gmail.com

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