Paraplegias and their sequelae are among the most far-reaching events in an affected person's life. The neurological deficits and impairments resulting from damage to the spinal cord affect not only the motor system, sensation, and autonomic functioning of a patient but also have serious psychosocial sequelae. Whereas traumatic paraplegias are epidemiologically well documented, only very limited statistical data are available regarding the incidence of non-traumatic acute and subacute paraplegias. Acute and subacute paraplegic syndromes that were not obviously caused by a harmful external event (injury) are so uncommon that targeted diagnostic and therapeutic action does not always ensue because of limited experience with this clinical problem. This is particularly noteworthy since the dynamic of a developing paraplegia is often assessed wrongly. Cervical paraplegias had a poor survival prognosis up to the middle of the 20th century, but the situation has improved substantially thanks to progress in acute care and rehabilitation medicine (1). Patients with acute or subacute paraplegias do mostly not present with an easily recognizable clinical picture of a total paraparesis or tetraparesis with impaired motor and sensory function, and loss of control over bladder and rectum. Rather, in individual cases, especially while clinical symptoms are newly developing, unspecific neurological symptoms may dominate that may hinder correct diagnosis. Although the therapeutic options in traumatic paraplegias are still limited, some treatments exist for non-traumatic paraplegias, which address the underlying cause. These may crucially influence the prognosis of the paraplegic syndrome. Early diagnosis is therefore vitally important. This article presents the most important differential diagnoses of the non-traumatic acute or subacute paraplegic syndrome and the resulting consequences for diagnostics and treatment (box).

Ischemic spinal cord lesions

After vascular disorders of the spinal cord, ischemic lesions are more common than spinal hemorrhage. Compared with cerebral ischemias, spinal ischemias are rare. The reasons include the fact that the spinal arteries are often not overly affected by atherosclerosis and the particularities of arterial blood supply to the spinal cord with its marked extraspinal and

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**SUMMARY**

*Introduction:* Awareness of symptoms and possible causes of acute and subacute non-traumatic paraplegia are crucial to avoid any diagnostic delay of these medical emergencies. *Methods:* Selective literature review. *Results:* Possible causes of acute paraplegia are vascular spinal disease (primary or secondary ischaemic myelopathy, spinal haemorrhage, vascular malformation), inflammatory spinal disease, and spinal tumors. These must be distinguished from diseases presenting with acute paraplegia but not involving the spinal cord, such as polyradiculitis, hyperkalaemic and hypokalaemic paralysis, and psychogenic paraplegia. Lumbar puncture and the recording of somatosensory and motor evoked potentials may also be helpful in establishing the diagnosis. Initial evaluation of an acute paraplegia should seek evidence of cord compression, in which case urgent surgical decompression is mandatory. Spinal MRI should be obtained at the earliest opportunity. *Discussion:* Rapid diagnosis and early treatment of non-traumatic paraplegia are crucial determinants of prognosis. In many cases, this can prevent the development of an irreversible paraplegia, especially if the patient presents with incipient symptoms. *Dtsch Arztebl 2006; 103(44): A 2948–54.*

Key words: paraplegia, myelitis, spinal ischemia, spinal hemorrhage, spinal tumour
intraspinal collateralization. The blood supply to the spinal cord comes from three longitudinal arteries – the unpaired anterior spinal artery and the paired posterior spinal arteries – which are linked to each other via an expansive network of anastomoses and which are supplied mainly by the vertebral arteries and the posterior inferior cerebellar arteries, the arteries to segments C5 to C8, and the major anterior radicular artery (which most commonly branches off between Th10 and L1). The blood supply to the anterior two-thirds of the spinal cord comes from the sulcocommissural arteries, which branch off from the anterior spinal artery, that of the posterior third from penetrating branches of the posterior spinal arteries.

The clinical picture of acute spinal cord ischemia develops within just a few minutes to several hours and is determined by the vascular supply system that is involved (2). One has to differentiate between the anterior spinal artery syndrome, the arteria sulcocommissuralis syndrome, and the arteria spinalis posterior syndrome. The initial symptom of the anterior

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**Differential diagnosis of acute and subacute non-traumatic paraplegic syndromes**

1. **Vascular disorders of the spinal cord**
   1.1 Ischemic disorders of the spinal cord
      1.1.1 Primary ischemias: atherosclerosis, vasculitis
      1.1.2 Secondary ischemias: vascular compression secondary to space occupying lesions, disorders of the aorta
   1.1.3 Decompression sickness
   1.2 Spinal hemorrhages: epidural hematoma, subdural hematoma, subarachnoid hemorrhage, intraparenchymal hemorrhage (hematomyelia)
   1.3 Spinal vascular malformations: dural arteriovenous fistula, perimedullar fistula, intramedullar arteriovenous angioma, cavernoma

2. **Inflammatory disorders of the spinal cord**
   2.1 Without compression of the medulla
      2.1.1 Acute transverse myelitis: viral, bacterial, fungal, accompanying or following infection, after vaccination
      2.1.2 Myelitis in chronic inflammatory disorders of the central nervous system (e.g., multiple sclerosis, neuroborreliosis)
      2.1.3 Myelitis in systemic diseases (such as Behçet’s disease)
   2.2 With medullar compression
      2.2.1 Epidural abscess
      2.2.2 Subdural abscess
      2.2.3 Spondylodiscitis

3. **Toxic or allergic disorders of the spinal cord**
   3.1 Subacute myelo-optico-neuropathy (SMON) caused by clioquinol
   3.2 Late myelopathy after chemonucleolysis

4. **Non-inflammatory spinal space occupying lesions**
   4.1 Disc prolapse
   4.2 Neoplasms

5. **Non-spinal disorders**
   5.1 Acute polyradiculitis Guillain Barré
   5.2 Hyperkalemic or hypokalemic paralyses
   5.3 Parasagittal cortical syndrome (e.g., bilateral infarction in the area receiving its blood supply from the the anterior cerebral arteries)
   5.4 Psychogenic paraplegic symptoms
spinal artery syndrome as the most common vascular spinal cord syndrome are most commonly belt-like or radicular pain, followed by flaccid tetraparesis or paraparesis, disturbed function of bladder/rectum, and isolated loss of pain and temperature sensation, while vibration sense and proprioception are mostly intact. The syndrome is most commonly located in the middle to lower thoracic spine. The arteria sulcocommissuralis provides the blood supply to the right and left sides of the anterior two thirds of the spinal cross-section, so that once ischemia occurs, anterior spinal artery hemisyndrome develops. In case of the very rare arteria spinalis posterior syndrome, which in most cases affects both posterior spinal arteries simultaneously, the dorsal columns are typically affected, resulting in static and locomotor ataxia, sometimes also (if the lateral funiculus is affected) central paresis with pyramidal signs (3).

**Figure 1:** Male patient aged 74 years with a spinal dural arteriovenous fistula. Clinically progressing paraparesis over two years, with episodic, acutely deteriorating, impaired sensitivity only in the feet.

a) T2, TSE sagittal. Longitudinal increase of the signal (light area) as a sign of edema and glioses resulting from venous congestion in the spinal cord.
b) T2, TSE axial. The changes (light area) are positioned centrally in the spinal cord in the axial image.
c) T2 3D GRE. The congested veins (shaded) are showing up snaking and dilated.
d) Arteriovenous fistula in contrast-medium enhanced magnetic resonance angiography (arrow).
Etiologically, in acute spinal cord ischemia, primary vascular disorders of the spinal cord are differentiated from secondary spinal cord ischemias (4). In case of primary ischemias, mention must be made of atherosclerotic vascular changes as well as the fact that in vasculitides or collagenoses, spinal vessels are affected. Principally, this can happen in all systemic vasculitides. It has often been described in patients with systemic lupus erythematoses and Sjögren’s syndrome (5, 6).

Among the secondary spinal cord ischemias, the first to be mentioned is the compression of spinal cord arteries through intraspinal expansion processes (such as tumors, disc prolapses, abscesses). In acute or subacute paraplegic symptoms, these will have to be ruled out immediately by magnetic resonance imaging since they usually require instant surgical decompression. Further important causes that have to be ruled out by differential diagnoses are disorders of the aorta, such as aortic dissection or extensive aortic aneurysms. These may result in ischemic lesions in the middle and lower thoracic spine (7). Symptoms may be fluctuating. If paraplegic symptoms occur at this level, however, these possible causes of spinal ischemias will have to be considered and further diagnostic tests using sononography and computed tomography should be initiated. Timely treatment may prevent further damage to the spinal cord in this context. A rare cause of spinal ischemia is decompression sickness in divers. The paraplegic syndrome caused by decompression sickness is not always reversible (8).

Spinal hemorrhages and vascular malformations
In analogy to intracranial hemorrhages, spinal hemorrhages may also lead to epidural and subdural spinal hematomas and spinal subarachnoid hemorrhages and parenchymal hemorrhages (hematomyelia). The most common cause of spinal hemorrhages include spinal cord trauma, often combined with an increased tendency to bleed (for example, in hemophilia or during anticoagulant treatment) as the predisposing factor. Cases of spontaneous spinal epidural or subdural hematoma have been described previously (9, 10). Spontaneously occurring spinal subarachnoid hemorrhages are mainly bleeds of arteriovenous angiomas of the spinal cord, occasionally also of spinal tumors (11). Bleeding aneurysms, however, are rare and occur only high up, in the cervical spine, in the area receiving its blood supply from the vertebral arteries. These run through the foramen magnum immediately adjacent to the transition area between cervical medulla and medulla oblongata. Clinically, acute radicular back pain is the main symptom, accompanied by headache, nausea and vomiting, and mostly incomplete paraplegic symptoms. In the same way, spontaneous intraparenchymal spinal hemorrhages occur mainly on a background of arteriovenous malformations or tumors, sometimes also of clotting disorders (11). With regard to their clinical symptoms, these cannot be distinguished from ischemic spinal cord lesions. Differentiation from ischemia is confirmed by spinal magnetic resonance scan.

Spinal vascular malformations, such as dural spinal fistula or intramedullar arteriovenous malformations may result in spinal cord symptoms both through hemorrhages and through ischemia due to venous congestion or through ischemias via steal effects. The lead symptom is slowly progressing paraplegic symptoms with periodic, acute or subacute, occasionally transient, deterioration. A diagnosis can usually be made non-invasively, by spinal magnetic resonance imaging and magnetic resonance angiography (figure 1). A selective spinal digital subtraction angiogram (DSA) is still seen as essential, to determine the exact location of the fistula, to document venous drainage, and to plan further treatment (12).

Inflammatory spinal cord disorders
Among the inflammatory spinal cord disorders that may lead to acute or subacute paraplegic symptoms, myelitides have to be differentiated from inflammatory space occupying lesions. These result in compression and therefore secondary damage to the spinal cord. Because of the different therapeutic consequences, in cases of suspected inflammatory spinal cord disorders, compression of the spinal cord has to be ruled out immediately by spinal magnetic resonance imaging (13). Among the myelitides, transverse myelitis can be distinguished from myelitis in a case of chronic inflammatory disorder of the central nervous system or a systemic disorder.

Acute transverse myelitis usually presents with paraplegic symptoms, accompanied by or immediately following a febrile (viral) infection. An absence of fever does not definitely
rule out a diagnosis of acute myelitis, however. In some patients, the symptoms are the
direct result of the viral infection; in others, they are the result of the immunologically
mediated process caused by the viral infection. Such an immunologic mechanism is also
the cause of post-vaccination myelitis (14). Acute myelitis is only rarely caused by infection
with bacteria or fungi. Myelitides may also occur in chronic inflammatory disorders of the
central nervous system, e.g. as an episode in multiple sclerosis or in neuroborreliosis.
Additionally, they may manifest in systemic diseases such as Behçet’s disease. Acute polio
infection (Polio myelitis anterior) and herpes zoster with involvement of the spinal cord
(Polio myelitis posterior) are seen as special forms of myelitis. These typically do not affect
the entire spinal cross-section and are therefore characterized clinically by motor symptoms
only (flaccid paralysis or incomplete paraplegic symptoms) (15, 16).

Diagnostically, analysis of the cerebrospinal fluid is advised in all forms of myelitis,
after spinal cord compression has been ruled out by imaging techniques. In addition to
confirming the diagnosis, this also differentiates between acute inflammatory processes
and chronic inflammatory processes (13). Especially the identification of oligoclonal banding
has a high positive predictive value in regard to the development of multiple sclerosis (17).
To differentiate myelitis as an episode of a chronic inflammatory neurological disorder
from acute transverse myelitis, multimodal evoked potentials (visual evoked potentials,
VEP; somatosensory evoked potentials, SEP) can contribute valuable additional information:
in multiple sclerosis, these characteristically show clear latency delays as a sign of the
primary demyelinating disease process, whereas in acute transverse myelitis, typically
what is found is a cross-sectional loss of SEP or a reduction in amplitude without any sign
of a latency delay (18). In neuroborreliosis, which mostly manifests as myeloradiculitis or
myeloradiculopolyneuritis, electrophysiologically, a combination of neurological pathological
changes occurs.

Among the space occupying inflammatory disorders of the spinal cord that are accompanied
by acute or subacute paraplegia, spondylodiscitides must be differentiated from epidural
abscesses and the very rare subdural abscesses. Such space occupying inflammatory lesions
develop in most cases from hematogenic spread of inflammatory foci in different locations
or in general infections. Causative strains are most often staphylococci, more rarely
streptococci or gram-negative strains, occasionally even mycobacteria (19). Predisposing
factors – such as diabetes mellitus, immunosuppression, consumptive diseases, or alcohol
or drug dependence – have an important role. Additionally, iatrogenic abscesses may develop
– e.g., after epidural injections. Spondylodiscitides occur most commonly on the thoracic
and lumbar spine; their symptoms are often unspecified or they are clinically silent, so that
they are recognized only when neurological symptoms develop through nerve root
involvement or paraplegic symptoms (figure 2). The diagnostic gold standard in case of
suspected spondylodiscitis is the magnetic resonance scan (20). Treatment of paraplegic
symptoms owing to this cause consists of immediate surgical decompression.

The clinical course of an epidural abscess – most commonly located on the thoracic spine
– in the initial phase is characterized by severe local back pain that may be radiculating or
not. General signs of infection are common but may be absent. Further, subacute paraplegic
symptoms develop, with increasing weakness and loss of sensation in the legs, positive
Babinski signs, and increasingly disrupted control over bladder/rectum. The slower course,
the presence of other foci of infection, and the initial back pain are diagnostic pointers in the
distinction from acute transverse myelitis. A spinal epidural abscess is a medical emergency
that requires immediate surgery and antibiotic treatment after the diagnosis has been
confirmed by magnetic resonance imaging, because the prognosis depends crucially on the
extent of the paralyses that have already occurred preoperatively (21).

Non-inflammatory spinal space occupying lesions
Much like the inflammatory space occupying lesions described above, non-inflammatory
spinal space occupying lesions may result in acute or subacute paraplegic symptoms.
In addition to secondary spinal cord ischemia, a possible mechanism is the compression of
spinal vessels and thus direct pressure damage to the spinal cord. Although clinically, in
spinal tumors, slowly progressing spinal cord symptoms predominate, they may manifest
as an acute or subacute paraplegic syndrome, especially in fast-growing tumors, e.g.
metastases or pathological vertebral fractures (22). A further cause of acute or subacute
paraplegic symptoms is a median disc prolapse. This is localized mostly in segments L3/L4 or L4/L5 and, through compression of the caudal fibers, leads to polyradicular flaccid paralysis and impaired sensation and loss of control of bladder/rectum. Such a prolapse may also, however, be localized in the thoracic segments (23).

Non-spinal disorders
In the differential diagnosis of non-traumatic paraplegias, disorders have to be considered that do not primarily affect the spinal cord but whose symptoms may create the false impression of a spinal cord disorder. These include acute Guillaumin-Barré syndrome (GBS), hypokalemic or hyperkalemic paralyses, parasagittal cortical syndrome in case of bilateral ischemia in the area of the anterior cerebral arteries, and psychogenic paraplegic syndromes.

Acute GBS is characterized by ascending flaccid paraparesis, sometimes accompanied by impaired sensitivity, which may create the impression of a sensory level, as well as by loss of independent muscular reflexes. The symptoms typically start distally in the legs and may quickly develop into paraparesis or tetraparesis. Often this has been preceded by an infection, but it may also occur spontaneously. The pathogenesis is that of an autoimmune reaction...
Algorithm for emergency diagnostics in patients with onset of acute or subacute paraplegia, adapted from (13)
against components of the peripheral nerves, especially the myelin sheath. In addition to the typical clinical presentation, demyelination of the peripheral nerves or nerve roots can be proved by delays in neural transmission. Additionally, a "dissociation albuminocytologique" may be found in the cerebrospinal fluid, a disruption to the blood-brain barrier, with raised protein levels in the cerebrospinal fluid while the cell count remains normal. This constellation is, however, not specific for GBS but may also appear as cerebrospinal fluid occurring below the block in spinal cord compression.

Acquired hypokalemic or hyperkalemic paralyses that manifest as acute or subacute flaccid parapareses or tetrapareses with loss of muscular reflexes but are not accompanied by impaired sensation, may be confused with spinal disorders (24). The cause is in most cases an endocrine or toxic disturbance of the serum sodium concentration. Hereditary dyskalemic periodic paralyses, however, are not a genuine differential diagnosis because these manifest in infancy and can easily be recognized in the family history because of their autosomal dominant transmission.

Bilateral ischemia of the anterior cerebral arteries can mimic acute paraplegic symptoms. These arteries supply the parasagittal cortex and the leg area of the somatosensory and motor cortex, so that after a bilateral infarction, pareses and impaired sensation of the lower limbs may result. Damage to this area caused by other factors may also result in a parasagittal cortical syndrome with bilateral leg paralysis, e.g., in the case of fast-growing tumors. The diagnosis is made by cranial computed tomography or magnetic resonance imaging.

Psychogenic pareses may also occasionally take an acute course. In such cases, current conflicts or traumatizing experiences can often be elicited. Directional in the clinical investigation are pareses of changing severity while reflexes and muscle tone remain normal. Of great clinical use in such cases are somatosensory and motor evoked potentials (SEP and MEP), which in individual cases may prove objectively the functional continuity of motor and somatosensory pathways. It has to be borne in mind that a normal MEP from the affected muscles excludes a high-grade paresis or paralysis. Low-grade pareses may occasionally be accompanied by a normal MEP (25).

Conclusions for clinical practice

The occurrence of acute or subacute paraplegia is a medical emergency and requires instant diagnostic action to clarify the etiology and to be able to start therapy immediately. This is especially important as the prognosis can be improved in many cases by fast therapeutic action, and the extent of the resulting deficiency may be appreciably reduced. The central diagnostic tool is magnetic resonance imaging, to exclude non-inflammatory or inflammatory space occupying lesion with spinal cord compression, which requires immediate surgical decompression. The importance of magnetic resonance imaging, however, is not by any means limited to the exclusion of a space occupying lesion but allows direct diagnosis of ischemic or inflammatory spinal cord lesions. Spinal computed tomography on the other hand is the inferior diagnostic method compared with magnetic resonance imaging because of its inferior resolution of soft tissue. In non-traumatic paraplegias, spinal computed tomography is indicated only in individual cases, additionally to magnetic resonance imaging, to obtain information about bone structures. Further important diagnostic procedures include analysis of cerebrospinal fluid to prove or exclude an inflammatory pathogenesis and the evoked potentials (SEP and motor evoked potential, MEP). In case of consistent clinical signs, secondary damage to the spinal cord has to be considered – e.g. in aortic aneurysm – and the diagnostic methods have to be adapted accordingly.

Because of the narrow time window, the rule is that patients with a developing bilateral paralysis of the limbs should be transferred as soon as possible to a neurological or neurosurgical ward, where spinal magnetic resonance imaging is possible at all times and where a further diagnosis can be made speedily. The diagram shows an algorithm for the recommended diagnostic procedures. Only sound knowledge of the multiple possible differential diagnoses can guarantee optimal care and help prevent lasting paraplegia.

Conflict of Interest Statement

The authors declare that no conflict of interest exists according to the Guidelines of the International Committee of Medical Journal Editors.

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