

CASE OF THE WEEK

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CLINICAL PICTURE

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65 years old male patient with vascular risk factors (diabetis, hypertension, increased blood viscosity etc) presented clinically with a mainly motor clinical presentation in the form of atrophy of small muscles of the hand and evidence of upper motor neuron manifestations in the lower limbs and with a clinical picture characterized by a painless onset with a chronic fluctuating course. Meticulous clinical examination reveled mild segmental sensory manifestations at C5,C6 spinal segments. In this patient, increased whole blood viscosity was in the form of increased fibrinogen level, increased platelet aggregability, relative increase in the hematocrit value, reduced RBCs deformability...etc. Evidence of spinal cord claudication was present in the form of aggravation of weakness in the lower limbs when walking and a sensation of hotness felt in the hands when the patient walks for a distance.

RADIOLOGICAL FINDINGS

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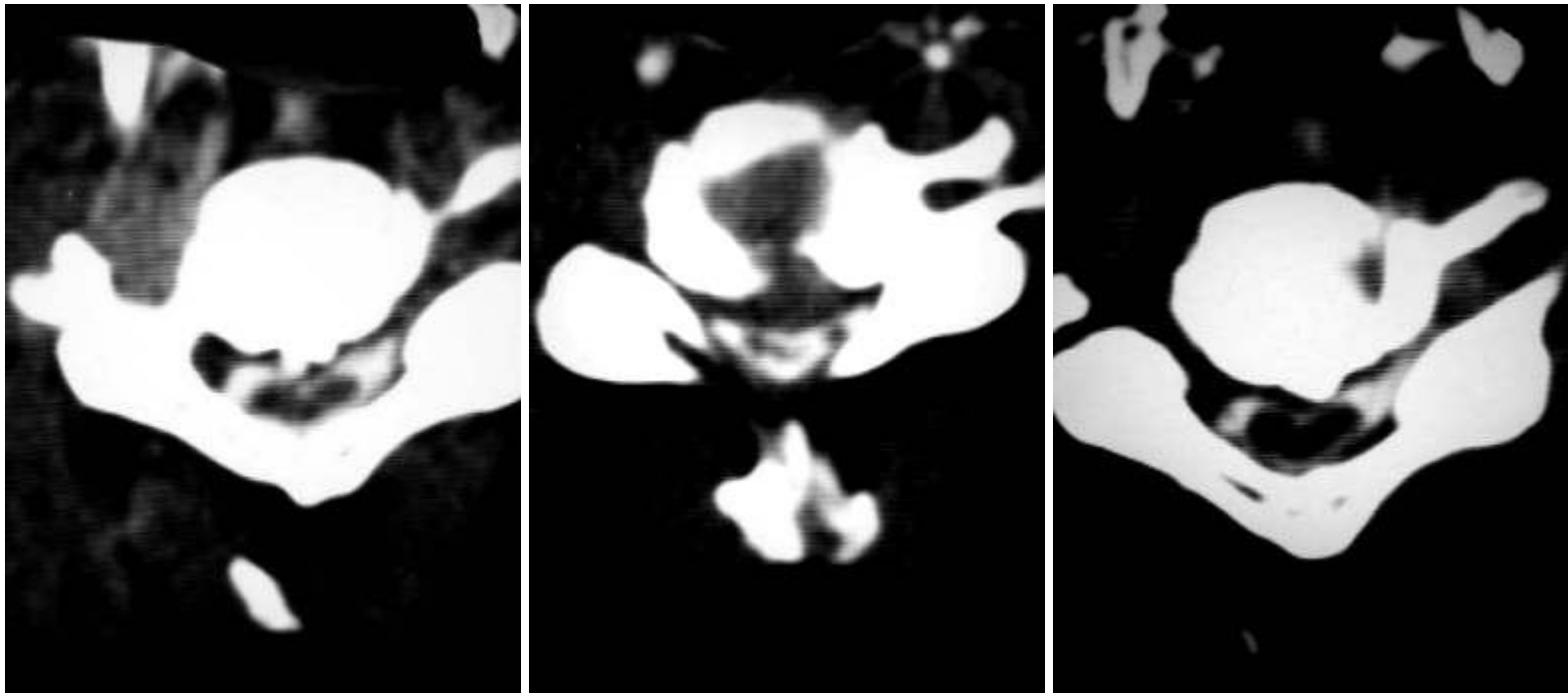


Figure 1. CT myelography at C4,C6,C7 cervical spinal segments showing evidence of spondylitic changes in the form of posterior osteophytes, cervical canal stenosis, luschka joint hypertrophy, and segmental spinal cord atrophy at C5,C6 spnal segments

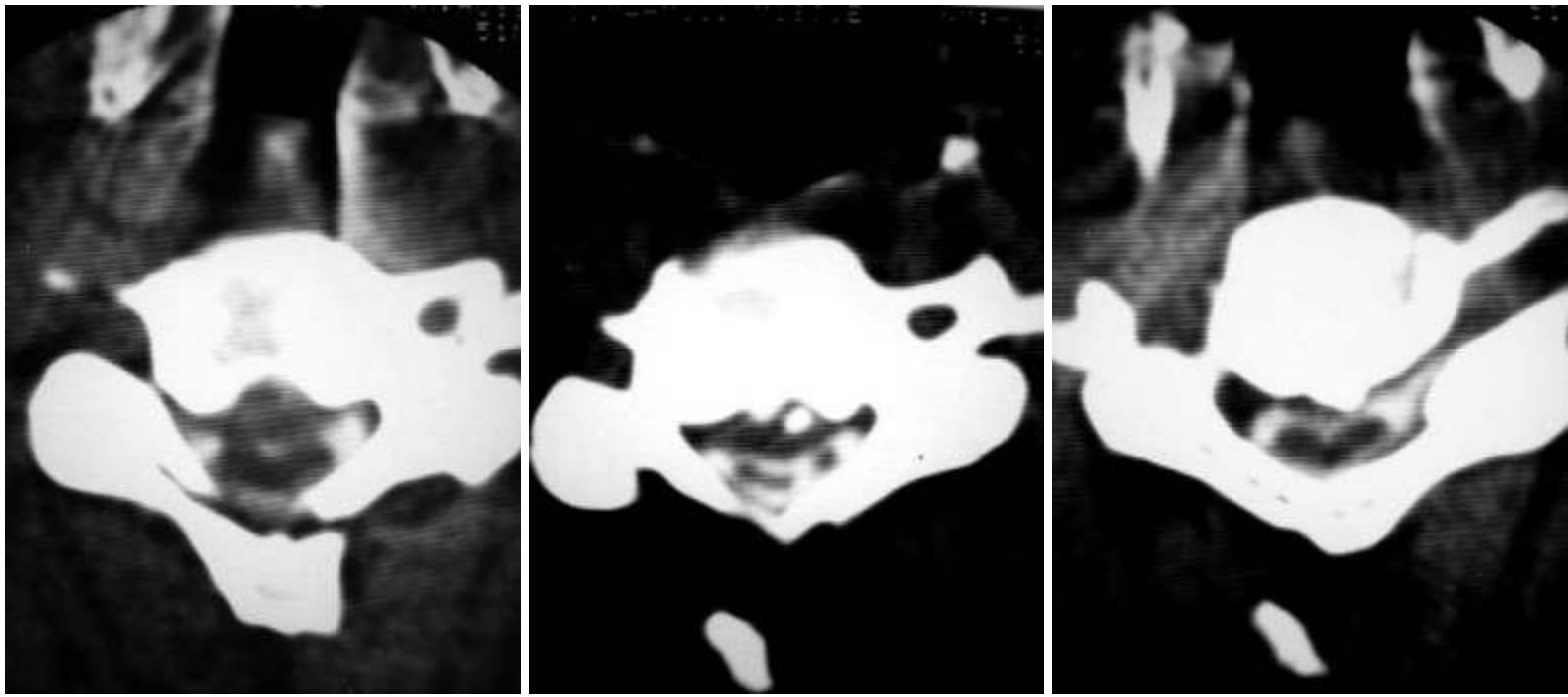


Figure 2. CT myelography at C4,C6,C7 cervical spinal segments showing evidence of spondylitic changes in the form of posterior osteophytes, cervical canal stenosis, luschka joint hypertrophy, and segmental spinal cord atrophy at C5,C6 spinal segments. Notice the calcified soft disc herniation.

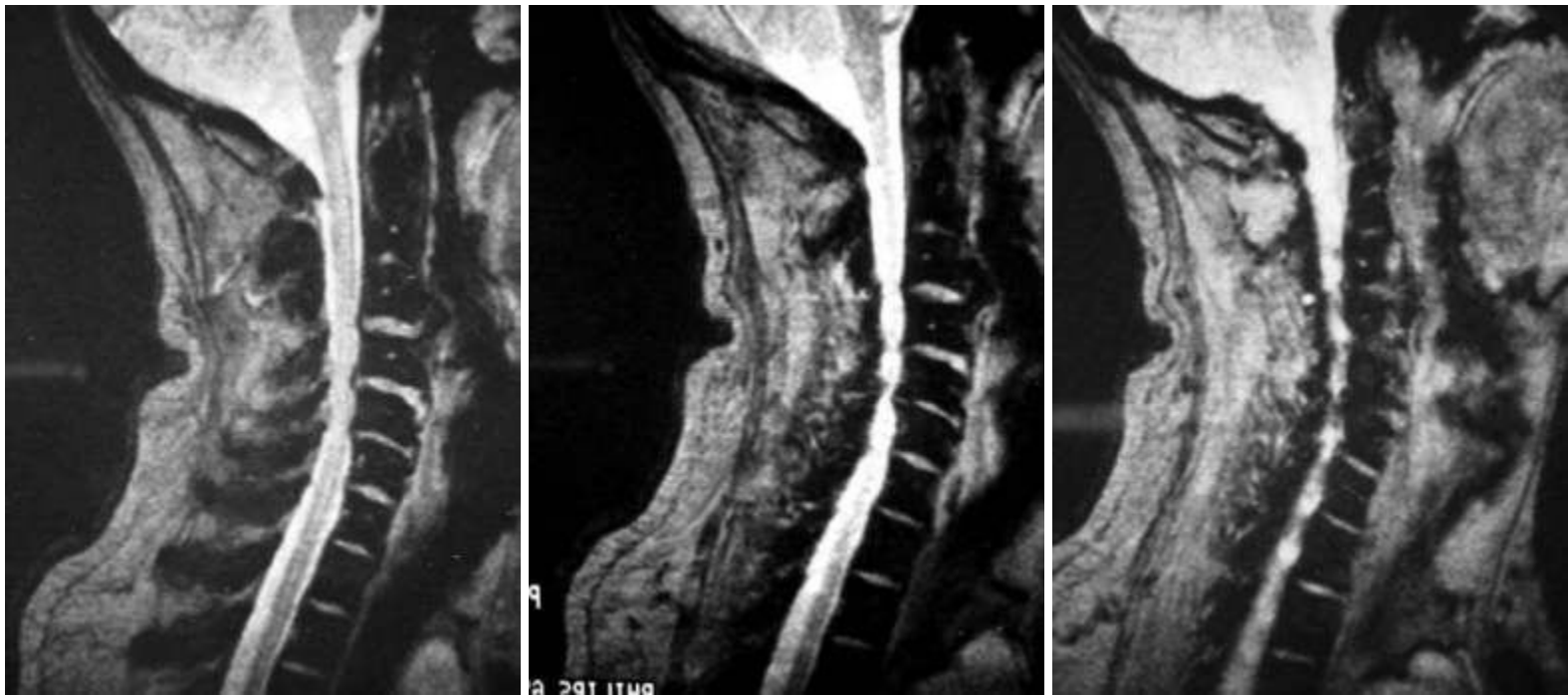


Figure 3. MRI T2 images showing evidence of cervical disc degeneration and cervical canal stenosis

In the above reported case evidence of spondylitic cervical canal stenosis, with posterior osteophytes, calcified hard disc herniation and C5,C6 segmental spinal cord atrophy was demonstrated. The clinical picture was in the form of a mainly motor clinical manifestations and a remitting course with evidence of spinal cord claudication.

Because the vascular spondylitic myelopathy has a sudden painless onset and a fluctuating course with remission and exacerbation, it was frequently misdiagnosed as multiple sclerosis. However major differences are present

between myelopathy due to disc disease and that due to multiple sclerosis as follows

- Unlike multiple sclerosis, myelopathy due to cervical spondylosis had a sudden onset with the clinical picture developing over just a few hours.
- Unlike multiple sclerosis, the duration of relapses in myelopathy due to cervical spondylosis is very short (on the average few hours to one or two days).
- Unlike multiple sclerosis, relapses of myelopathy due to cervical spondylosis shared a similar clinical presentation in every single patient i.e. the disease was disseminated only in time and never in place. And although signs and symptoms might be severer on recurrent episodes (mainly due to the cumulative effect of structural damage and/or the functional disturbances caused by each ischaemic episode), however the disease used to recur in the same anatomical site (dorso-lumber spinal segments) and is never disseminated in place.
- Unlike multiple sclerosis, the clinical picture of myelopathy due to cervical spondylosis is mainly motor (in the form of weakness and atrophy) and sensory manifestations, though definite, are detected only by careful examination.

In fact the "mainly motor clinical picture" was occasionally a potential source for another misdiagnosis which is motor neuron disease or motor neuropathy. However myelopathy due to cervical spondylosis can easily be differentiated from motor neuron disease because of the relapsing remitting course, and because of the existence of definite, though subtle, sensory manifestations. Also the existence of impotence, bladder disturbances and occasional back pain are points against the diagnosis of primary motor neuron disease.

The predominance of motor manifestations in myelopathy due to cervical spondylosis is in fact another point favouring its ischemic aetiology. It is clear that when ischaemia occurs, the most vulnerable region of the spinal cord is the grey matter because its metabolic rate is three to five times greater than the metabolic rate of the white matter. This would account for the many cases reported in literature of paraparesis with little sensory manifestations and for instances of lower motor neuron syndromes of an ischaemic basis.

In cervical spondylotic myelopathy patients the motor weakness is characteristically increased by walking and relieved by rest and this is another point favouring the ischemic aetiology of myelopathy due to degenerative disc disease. Normally walking is associated with marked increase of blood flow to the spinal cord and cauda roots to meet the increased metabolic rate of these neural structures, physiologically the spinal cord microvascular bed will dilate to accommodate the increased blood flow. Cervical canal stenosis (induced by disc disease) and the associated segmental arteriosclerosis will hinder this normal physiological "exertion induced hyperaemia" of the neural structures resulting in a temporary spinal cord "ischaemic dysfunction on exertion".

Although the prognosis following a single ischaemic episode is good , however repetition of the ischaemic episodes will ultimately result in spinal cord atrophy with irreversible neurological deficits The patient, following admission, received medical treatment for diabetes, hypertension, antiplatelet medications and medications that improve RBCs deformability, reduce whole blood viscosity and fibrinogen level (like pentoxifylline, bezafibrate etc) and he was referred to surgery once diagnosed radiologically.

DIAGNOSIS:

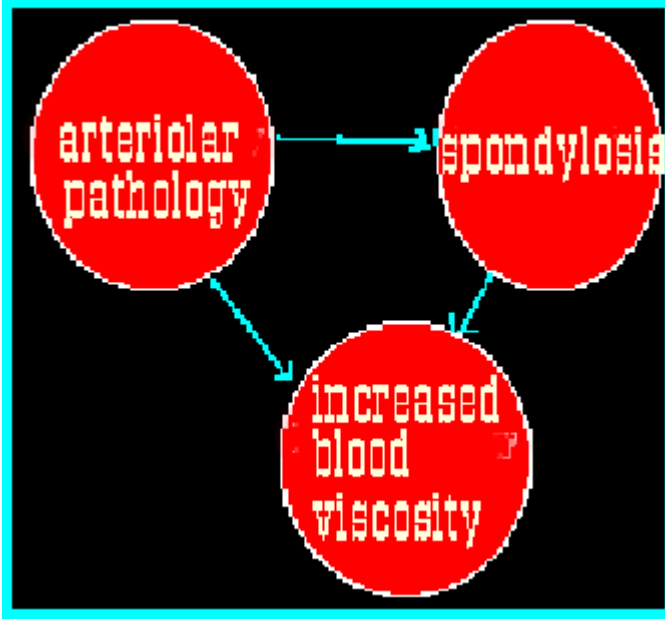
DIAGNOSIS: CERVICAL SPONDYLOTIC VASCULAR MYELOPATHY

DISCUSSION

DISCUSSION:

Patients with spondylitic cervical myelopathy are classified, clinically, into two main groups. The painless

myelopathy group (due to vascular aetiology) and the painful myelopathy group (due to compression of the spinal cord). Clinically the painless myelopathy group is characterized by a fluctuant course with remission and exacerbations and a mainly motor clinical presentation.



In the painless myelopathy group, the natural history of the disease is determined by the interaction of three main pathogenic factors. Spondylitic factor, vascular factor and haemorheological factor associated with increased whole blood viscosity (increased haematocrit value and serum fibrinogen).

Figure 4. The painless cervical spondylitic myelopathy is the interaction between three main pathogenic factors

The spondylitic factor ultimately results in bony and soft tissue hypertrophy that causes cervical canal stenosis, and encroach upon the subarachnoid space, reducing its volume. Lack of the CSF cushioning effect will cause embarrassment of the spinal circulation at the level of cervical enlargement since optimum blood supply to the spinal cord needs an optimum CSF cushioning effect.

The second factor is the vascular arteriolar factor. Apparently the painless myelopathy group comprises a group of patients where the incidence of vascular risk factors, (such as essential hypertension, NIDDM, hyperlipidaemia (Type IV), hyperfibrinogenemia and increased platelet aggregation...the metabolic syndrome) is very high. The incidence of arteriosclerosis is known to be high among patients with vascular risk factors.⁴

This is consistent with the necropsy findings of Manen, 1966² and Jillinger 1967¹. The authors reported arteriolosclerosis, lipohyalinosis and fibrosis of the perforating intramedullary vessels and the fine vessels lying on the surface of the spinal cord in patients with cervical spondylitic vascular myelopathy.⁴

These changes were maximum in the cervical enlargement and the overlapping zone in the cervico-dorsal region, making the spinal cord especially vulnerable at this zone to vesico-circulatory disorders, mainly of extra-medullary origin, which cause critical decrease in spinal cord flow. It should be mentioned that the area of the spinal cord between C4 and D1 is a watershed area with marginal blood supply. This last field zone is most likely to suffer from insufficiency of blood and has been shown to be a preferential zone for vascular damage.⁴

According to the necropsy results of Jillinger, 1967¹ the arteriosclerotic changes in the overlapping cervico-dorsal region were isolated findings. They did not depend on age and were negatively correlated with arteriosclerosis in the rest of the body. A finding that probably denotes that the injurious effect of the spondylitic changes accelerate the arteriosclerotic changes in the region of the cervical enlargement.

However it should be noted that both cervical spondylosis and arteriosclerosis are slowly progressive pathology and they can not be held responsible for the sudden onset of the clinical symptomatology seen in the painless myelopathy group. No compressing agents (like soft disc herniation, or osteophytes) are demonstrated radiologically, in this group of patients, that can explain the clinical symptomatology in terms of compression of the spinal cord and/or an important radicular artery. In short both cervical spondylosis and arteriosclerosis serve by furnishing the

background for the ultimate determinant of the clinical symptomatology.

Cervical spondylosis will result in canal stenosis, loss of the CSF cushioning effect and embarrassment of the spinal circulation in the region of the cervical enlargement. Arteriosclerosis will result in reduction of the caliber of the radicular and the perforating intramedullary arterioles with loss of the auto-regulatory physiological process. Flow in the perforating arteries is dependent on the auto-regulatory process of the penetrating intramedullary arterioles on one hand and the whole blood viscosity on the other hand. Loss of the auto-regulatory process secondary to advanced arteriosclerosis will simply mean that the spinal cord perfusion, in the vulnerable region of the cervical enlargement, will fluctuate with fluctuation of the whole blood viscosity, (the ultimate determinant of the vascular myelopathy).⁴

Whole blood viscosity is a collective terminology that reflects the influence of various factors that include mainly the corpuscular and the plasmatic components of the blood. Whole blood viscosity is usually increased in patients with the vascular spondylitic myelopathy.

Blood viscosity is mainly determined by the hematocrit value and the plasma viscosity is mainly determined by the plasma fibrinogen level. High values of lipid have also be found to increase whole blood viscosity.⁴

Increase platelet aggregation also increases whole blood viscosity. The behavior of the red blood cells was also found to affect the blood viscosity. Increased red cell aggregation and reduced red cell deformability increase whole blood viscosity. The RBCs deformability is invariably reduced and the aggregability is invariably increased in the presence of high fibrinogen level and high haematocrit values. Fibrinogen in particular is a strong RBCs aggregant agent. Inverse correlation is present between the red cell deformability and the haematocrit value and serum fibrinogen level.

Increase of the whole blood viscosity is a common finding in essential hypertension and NIDDM. The vascular resistance of the perforating blood vessels of the spinal cord and the brain is dependent upon the ratio between the whole blood viscosity over the caliber of the blood vessel. Increase of the whole blood viscosity results in high vascular resistance to blood flow and subsequently low perfusion pressure and neuronal tissue ischaemia. Stenosis of the perforating blood vessel secondary to arteriosclerosis further aggravates the problem. Inverse correlation is present between the neuronal tissue blood flow and serum fibrinogen level and the haematocrit value.⁴

Hyperfibrinogenemia and increased RBCS, and platelet aggregation reflect a hypercoagulable state with increased thrombotic tendency that selectively affects the small perforating blood vessels and the microcirculation of the brain and spinal cord. Microvascular occlusion can occur either by local aggregation of hyperaggregable platelets or by red cell aggregation with impaction of rigid red blood cells in the microcirculation. This is more likely to occur with the existence of high red cell mass (Hch value) that can displace the hypersensitive platelets Towards the arteriolar wall resulting in platelet aggregation and thrombus formation. Thrombus formation is enhanced if the arteriolar wall is abnormal (arteriolosclerosed).⁴

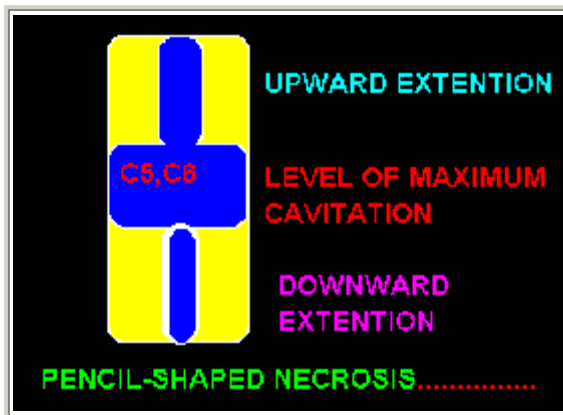
The high blood viscosity observed in the painless myelopathy patients should simply mean, especially when coupled with arteriosclerosis of the small perforating blood vessels, that the blood flow to the spinal cord at the level of cervical enlargement is subjected to high vascular resistance that could ultimately results in low perfusion pressure and chronic ischaemia. The increased thrombotic tendency observed in those patients should mean that the chronic ischaemia state could be interrupted by acute thrombotic microvascular occlusion that can result, pathologically, in spinal cord lacunar infarction at the level of cervical-enlargement and clinically in lower cervical painless myelopathy of sudden onset and regressive course. The acute thrombo-occlusive episodes are responsible for the intramedullary cavitations observed in the painless myelopathy group.⁴

NEUROIMAGING FINDINGS IN THE PAINLESS MYELOPATHY GROUP

In general two pathological subtypes are observed in this group, spinal cord atrophy and spinal cord cavitations, both are very segmental and both are the end result of spinal cord ischemia.

- Two types of spinal cord of cavitations are observed in the painless myelopathy group

| | |
|-------------|--|
| CAVITATIONS | 1-Pencil shaped necrosis: the cavitation is maximum at the level of C5,C6 spinal segments and extends one ore two segments above and/or below |
| CAVITATIONS | |
| CAVITATIONS | 2-Central gray matter cavitation: bilateral ,symmetrical,rounded cavitations, localized in the bilateral anterior horns of spinal segments C5,C6 |



Pencil shaped necrosis and central gray matter cavitations, most probably represent spinal lacunar infarctions. The spinal cord atrophy observed in those patients is very segmental and exclusively localized to the level of cervical enlargement between C4- C8 with maximum changes at C5,C6 segment. The spinal cord above C4 and below C8 is often normal. The segmental spinal cord atrophy observed in these patients is the result of long standing chronic ischaemia interrupted by recurrent thrombo-occlusive episodes.⁴

All the atrophic and cystic changes are exclusively limited to the spinal cord area between C4 and C7. The C5-C6 segments are most frequently involved. The C5-C6 segments are the most vulnerable to vascular

damage as they represent a watershed area with higher incidence of segmental arteriosclerosis.¹

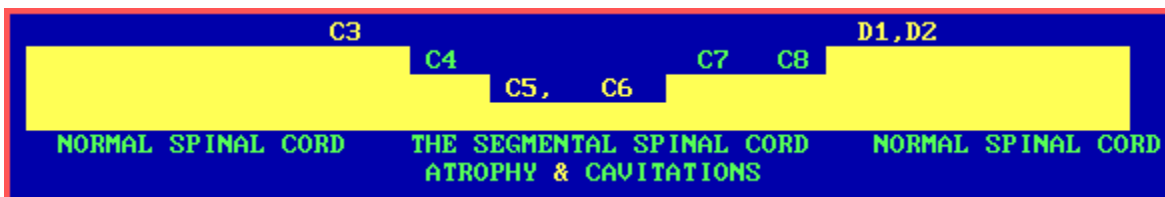


Figure 5. In cervical spondylitic myelopathy, both cystic and atrophic changes are exclusively localized to the level of cervical enlargement with maximum changes at C5,C6 spinal segments

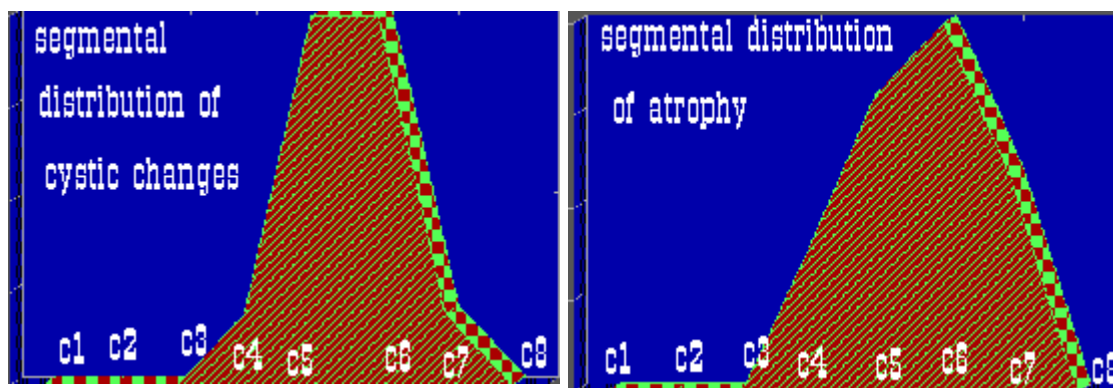


Figure 6. In cervical spondylitic myelopathy, both cystic [left image] and atrophic [right image] changes are exclusively localized to the level of cervical enlargement with maximum changes at C5,C6 spinal segments

The central gray matter cavitations demonstrated in the painless group group represent lacunar infarctions involving the bilateral anterior horns. It results clinically in a purely LMN picture (proximal muscles amyetrophy).¹

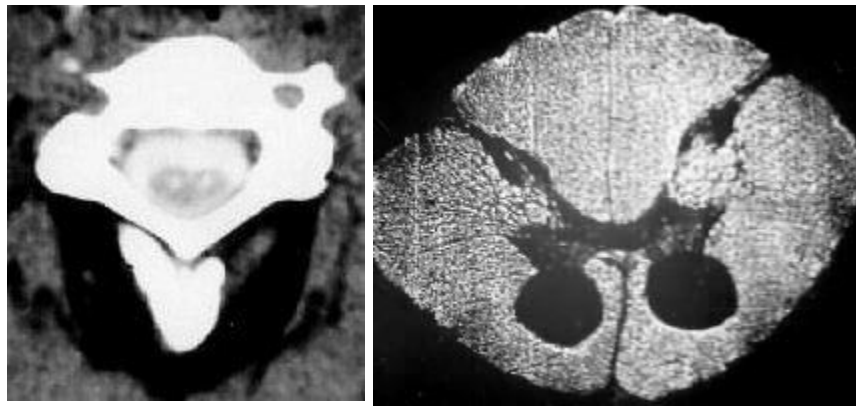


Figure 7. CT myelography showing central gray matter cavitations as bilateral symmetrical, well defined rounded zones of intramedullary contrast accumulations in the presumed anatomical areas of anterior horns [right image is a schematic representation]



The association between spondylitic myelopathy and spinal cord atrophy and/or cavitation is well known. The ischaemic aetiopathogenesis of myelopathy in this group of patients is further substantiated by the observation that relapses of myelopathy are intimately coupled temporally with rise of whole blood viscosity and thrombotic tendency. Relapses occur more frequently in the summer time. Dehydration is more common in summer time, it results in contraction of the plasma volume and rise of the haematocrit value and subsequently blood viscosity.

Figure 8. MRI T1, T2 images showing pencil-shaped necrosis

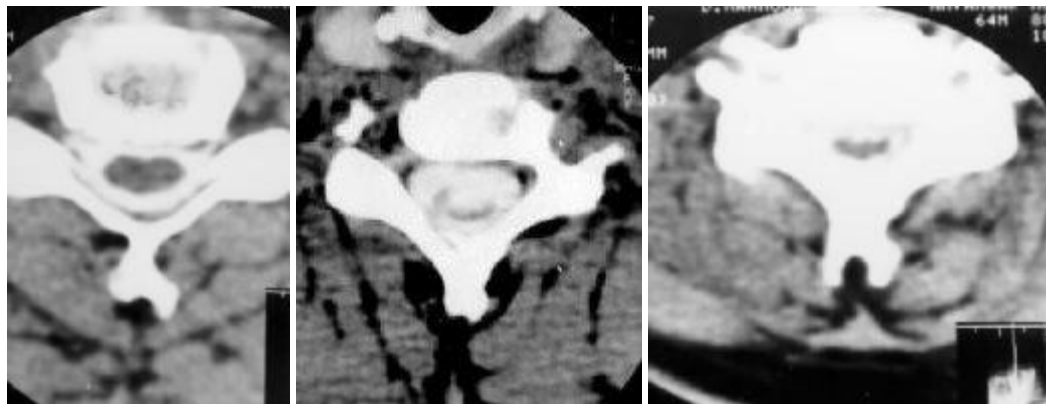


Figure 9. CT myelography showing normal spinal cord at the level of cervical enlargement (A) and spinal cord atrophy in cervical spondylitic myelopathy (B,C), notice that the atrophic segments are flattened, collapsed, with irregular contour and wide subarachnoid spaces

The ischaemic aetiology of myelopathy in the painless myelopathy group is consistent with the necropsy findings of Jellinger, 1967¹. The author reported, in spondylitic myelopathy patients, white matter ischaemic demyelination, neuronal degeneration and a diffuse lacunar state similar to those seen in the basal ganglion in the hypertensive small vessel disease of the brain. It should also be mentioned that increased whole blood viscosity is also the ultimate aetiopathogenic factor in hypertensive microvascular brain disease (diffuse lacunar state, leukoaraiosis, etc.).⁴

The haemorheological profile of vascular spondylitic myelopathy is similar to the haemorheological profile of hypertensive micro-vascular brain disease (Lacunar infarction, leukoaraiosis etc.) previously reported by many authors⁴. The haemorheological parameters tended to drop down to the normal levels following the acute phase in myelopathy patients and this has also been reported in ischaemic brain disease.

The hypertensive micro-vascular brain disease is found to be similar in many ways to the spondylitic vascular

myelopathy regarding vascular risk factors, the vascular arteriolar pathology, parenchymatous pathology and the haemorheological profile. Similarities are listed in in the following table.

| | |
|--------------------------------------|---|
| Vascular risk factors | NIDDM,hypertension,hyperlipidaemia ,old age,LVH,are common in both diseases |
| Vascular arteriolar pathology | Lipohyalinosis of the fine penetrating arterioles is common in both diseases |
| Pathological findings | Neuronal degeneration, ischaemic demyelination and a diffuse lacunar state are common in both diseases |
| Haemorheological profile | Increased whole blood viscosity and thrombotic tendency of the blood are common in both diseases |

Because the vascular spondylitic myelopathy has a sudden painless onset and a fluctuating course with remission and exacerbation, it was frequently misdiagnosed as multiple sclerosis or transverse myelitis. The demonstration of haemorheological abnormalities in those patients should suggest important lines of treatment. Antiplatelet medications, drugs that improve RBCs deformability, reduce whole blood viscosity and fibrinogen level (like pentoxifylline, bezafibrate etc) will be of great value in those patient. Also maintenance of good body hydration is of paramount importance especially in the older age group. Control of risk factors like hypertension and NIDDM is essential.

Although the prognosis following a single ischaemic episode is good. however repetition of the ischaemic episodes will ultimately result in spinal cord atrophy with irreversible neurological deficits (So patients with the spondylitic vascular myelopathy probably needs prophylactic treatment like cerebrovascular patients following the first acute myelopathic episode).

In general the vascular aetiology is the most common cause of the spondylitic myelopathy. The spondylitic vascular myelopathy is present mainly in males and is characterized, clinically, by a painless clinical picture and radiologically by the presence of segmental spinal cord atrophy or cavitations. Incidence of vascular risk factors is high among patients with vascular myelopathy with frequent haemorheological abnormalities denoting increased whole blood viscosity. Although central disc herniation can cause a painless clinical picture, but this can easily be excluded radiologically. The roles played by the spondylitic process, the vascular pathology and the haemorheological abnormalities in the pathogenesis of the spondylitic vascular myelopathy are summarized in following table.

| Pathological factor | Effect |
|---|--|
| Cervical spondylosis | Cervical canal stenosis with reduction of the CSF subarachnoid spaces,lack of the CSF cushioning effect will cause embarrassment of the circulation at the level cervical enlargement |
| Sclerosis of the intramedullary arterioles | Reduction of the caliber of the intramedullary arterioles with loss of the autoregulatory process, this increases the vascular resistance and results in low perfusion pressure and chronic tissue ischaemia. Arteriolosclerosis is maximum at the level of cervical enlargement |
| Increased whole blood viscosity | Increased fibrinogen level,haematocrit value,and platelet aggregability. This increases whole blood viscosity and subsequently the vascular resistance and results in low perfusion pressure and chronic tissue ischaemia, it also increases the thrombotic tendency of the blood and results in recurrent acute microvascular thrombo-occlusive episodes |

SUMMARY

CT SCAN IMAGING OF THE SPONDYLITIC VASCULAR MYELOPATHY

CT
SCAN

CT
SCAN

CT
SCAN

- Intrathecal enhancement is necessary
- In spinal cord atrophy, the spinal cord is deformed, with wide subarachnoid spaces
- In pencil shaped necrosis, intramedullary contrast accumulation occurs at C5,C6 with occasional extension of cavitation one or two segments Above or below
- In central gray matter cavitation there is bilateral,symmetrical, rounded and well-defined accumulation of contrast material in the regions of the anterior horns.

CERVICAL SPONDYLOSIS WITH SIGNIFICANT NECK PAIN ASSOCIATED WITH MYELOPATHY AND/OR RADICULOPATHY

- Neck pain in association with cervical spondylosis is either due to



1- **Acute neck pain** : Is commonly due to acute soft disc herniation, this results clinically in radiculopathy and or myelopathy.

2- **Chronic neck pain**: This is commonly due to foraminal stenosis due to facet joint hypertrophy and or luschka joint disease.

Figure 10. luschka joint hypertrophy resulting in marked foraminal stenosis

SUMMARY

SUMMARY

Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in older persons. The aging process results in degenerative changes in the cervical spine that, in advanced stages, can cause compression of the spinal cord. Symptoms often develop insidiously and are characterized by neck stiffness, arm pain, numbness in the hands, and weakness of the hands and legs. The differential diagnosis includes any condition that can result in myelopathy, such as multiple sclerosis, amyotrophic lateral sclerosis and masses (such as metastatic tumors) that press on the spinal cord. The diagnosis is confirmed by magnetic resonance imaging that shows narrowing of the spinal canal caused by osteophytes, herniated discs and ligamentum flavum hypertrophy. Choice of treatment remains controversial, surgical procedures designed to decompress the spinal cord and, in some cases, stabilize the spine are successful in many patients.

Cervical spondylotic myelopathy (CSM) is the most common spinal cord disorder in persons more than 55 years of age in North America and perhaps in the world. As the number of older persons in the United States increases, the incidence of CSM will most likely increase. In a prospective study designed to more accurately define the incidence

of CSM, 23.6 percent of 585 patients with tetraparesis or paraparesis admitted to a United Kingdom regional neuroscience center had CSM.⁵ The overall prevalence in this population is unknown.

Pathophysiology of CSM

Spondylosis refers to the degenerative changes that occur in the spine, including degeneration of the joints, intervertebral discs, ligaments and connective tissue of the cervical vertebrae. There are three important pathophysiologic factors in the development of CSM: (1) static mechanical; (2) dynamic mechanical; and (3) spinal cord ischemia. Static mechanical factors result in the reduction of spinal canal diameter and spinal cord compression. With aging, the intervertebral discs dry out resulting in loss of disc height. This process puts greater stress on the articular cartilage of the vertebrae and their respective end plates. Osteophytic spurs develop at the margins of these end plates (*Figure 11*). Osteophytes stabilize adjacent vertebrae whose hypermobility is caused by the degeneration of the disc.



Figure 11. Axial computerized tomography scan showing ventral osteophytes pressing into the spinal canal.

The disc also calcifies, further stabilizing the vertebrae. Osteophytes increase the weight-bearing surface of the end plates and, therefore, decrease the effective force being placed on them. In addition to osteophytic overgrowth, the ligamentum flavum may stiffen and buckle into the spinal cord dorsally. Osteophytic overgrowth ventrally and, in some cases, buckling of the ligamentum flavum dorsally can cause direct compression of the spinal cord resulting in myelopathy (clinically evident spinal cord dysfunction). Symptoms are believed to develop when the spinal cord has been reduced by at least 30 percent.

Dynamic mechanical factors relate to the fact that the normal motion of the cervical spine may aggravate spinal cord damage precipitated by direct mechanical static compression. During flexion, the spinal cord lengthens, thus stretching over ventral osteophytic ridges. During extension, the ligamentum flavum may buckle into the spinal cord causing a reduction of available space for the spinal cord (*Figure 12*).

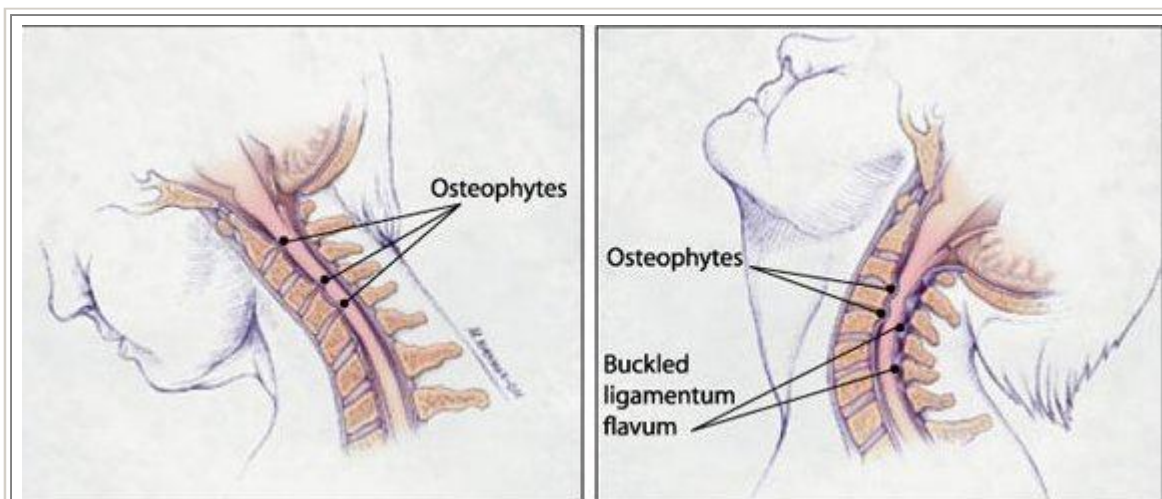


Figure 12. Dynamic mechanical factors in cervical spondylotic myelopathy. (*Left*) During flexion, the spinal cord is stretched over ventral osteophytic ridges. (*Right*) During extension, the ligamentum flavum may buckle into the spinal cord reducing space for the cord.

Spinal cord ischemia probably plays a role in the development of CSM, particularly in later stages. Histopathologic changes in the spinal cord consistent with ischemia have been observed in patients with CSM. However, the precise mechanism for spinal cord ischemia is not completely understood. Other factors associated with the development of spondylosis include heavy labor, posture and genetic predisposition. Also, 70 percent of patients with Down syndrome have an increased incidence of spondylosis by 50 years of age.

Clinical History

Patients with CSM will generally have these symptoms: neck stiffness; unilateral or bilateral deep, aching neck, arm and shoulder pain; and possibly stiffness or clumsiness while walking. CSM usually develops insidiously. In the early stages of CSM, complaints of neck stiffness are common because of the presence of advanced cervical spondylosis. Other common complaints include crepitus in the neck with movement; brachialgia, which is characterized as a stabbing pain in the pre- or postaxial border of the arm, elbow, wrist or fingers; a dull "achy" feeling in the arm; and numbness or tingling in the hands.

Common complaints of patients with cervical spondylotic myelopathy include neck stiffness, crepitus in the neck with movement, brachialgia, a dull "achy" feeling in the arm, and numbness or tingling in the hands.

Pain following a stereotypical dermatomal distribution is referred to as a radiculopathy rather than a myelopathy. For example, in patients with a disc herniation between the sixth and seventh vertebrae, pain radiates into the shoulder, upper arm, elbow, and index and middle fingers. It is typically unilateral. Numbness and weakness follow the same distribution. Some patients will exhibit signs and symptoms of radiculopathy and myelopathy.

The hallmark symptom of CSM is weakness or stiffness in the legs. Patients with CSM may also present with unsteadiness of gait. Weakness or clumsiness of the hands in conjunction with the legs is also characteristic of CSM. Symptoms may be asymmetric particularly in the legs. Loss of sphincter control or frank incontinence is rare; however, some patients may complain of slight hesitancy on urination.

• Addendum

- A new version of this PDF file (with a new case) is uploaded in my web site every week (every Saturday and remains available till Friday.)
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