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SUMMARY

In the present study 11 surgically confirmed patients with the clinico-radiological diagnosis of degenerative dorsal disc herniation are included. All patient were subjected to (1) full clinical examination (2) study of some haemorheological parameters and vascular risk factors (3) plain x ray dorsal spine (4) Myelography with postmyelography CT scan of the dorsal spine (5) MRI examination of the dorso-lumbar spine. Both MRI and CT myelography were done according to the suspected clinical spinal levels of each patient. Standard T1,T2 and proton density weighted images were used in MRI examination. All patients were subjected to both MRI and CT myelography. The cardinal clinical signs and symptoms of dorsal disc herniations, in this study, were "a mainly motor presentation", the presence of remissions and exacerbations of myelopathy and the presence of two levels in every single patient ( one at the conus-epiconus and anther sensory level at D6-D7 spinal segments). History of spinal trauma was characteristically absent and dorsal pain was minor and present in only 4 cases (36%). A
higher incidence of vascular risk factors and haemorheological changes consistent with increased whole blood viscosity and increased thrombotic tendency of the blood were found in patients with dorsal disc disease. Plain X ray dorsal spine and dorsal myelography were either normal or showed non specific minor changes in the majority of cases. Both CT myelography and MRI demonstrated partially or heavily calcified disc herniations in the lower dorsal vertebral segments with a maximum occurrence at D11, D12 level. Central gray matter cavitations at the level of disc herniation and spinal cord atrophy 6 spinal segments above the level of disc herniation were demonstrated in one case. The impact of these findings on our understanding of pathogenesis of myelopathy in degenerative dorsal disc disease and on the management of these cases will be discussed.

INTRODUCTION

Thoracic disc herniation presents a challenge in diagnosis and therapeutic decision making. Autopsy studies have suggest an incidence of thoracic disc herniations between 7% to 15% in individual died without a neurological history suggestive of dorsal disc disease, Abbot and Retter 1956, Arseni and Nash 1960. The incidence of asymptomatic dorsal disc herniation detected by incidental MRI or CT scan examinations in patients not presented with any neurological manifestations ranges between 11% to 13%, William and Cherryman, 1988, William et al, 1989, Awwad et al, 1991. According to Awwad et al, 1991, spinal cord deformation, flattening or distortion could be present, radiologically, even to a marked degree in asymptomatic individuals. Clinically, however, symptomatic dorsal disc herniations compose less than 1% of all spinal disc herniations and this simply means that the majority of dorsal disc herniations are asymptomatic. The majority of symptomatic disc herniations are calcified hard discs when first discovered and this simply means that they have been silently there for a long time before causing symptoms, Arce and Dohrmann, 1985 (a,b), Metwally,1991. History of trauma is absent in over 90 % of patients with symptomatic dorsal disc herniations, Arce and Dohrmann, 1985 (a,b), Metwally,1991, Dietze and Fessler, 1993. The existence of remissions and exacerbations of symptomatology in patients with symptomatic dorsal disc herniations could be confusing and very misleading, Arce and Dohrmann, 1985 (a,b), Dietze and Fessler, 1993. The aim of this study is to look for the aetiological factors that are responsible for symptom formation in degenerative dorsal disc herniations and that can explain the peculiar and protean clinical picture in patients with symptomatic dorsal disc herniations. Patients with symptomatic dorsal disc herniations are compared with an age matched group of patients with other space occupying pathology in the same anatomical locations (the dorso-lumbar regions) to see the exact role played by the dorsal disc herniations, as being space occupying lesions at these anatomical sites, in symptom formation and to see whether the space occupying nature of dorsal disc herniations and their common anatomical sites are solely responsible for the clinical picture or whether other aetiological factors are implicated, in conjunction dorsal disc herniations, in symptom formation. The impact of our findings on the pathogenesis and management of myelopathy in dorsal disc herniations will be discussed.

MATERIAL AND METHODS

In the present study 11 surgically confirmed patients with the clinical and radiological diagnosis of dorsal disc herniation are included. All patients were subjected to

1- Full clinical examination.

2- ECG.

2- Serum fibrinogen, haematocrit value, serum lipid and serum lipid electrophoresis, blood sugar and platelet aggregation (haemorheological and vascular risk factors)

3- Plain x ray dorsal spine.

4- myelography and post-myelographic CT scan.

5- MRI.
With regard to the platelet aggregation methodology, the ADP induced platelet aggregation and the light transmission technique was used. A platelet rich plasma suspension was obtained from each patient and a normal control.

The transmission of light through the platelet suspension was continuously monitored on a pen-ink recorder. The addition of ADP resulted in the formation of an increasingly large platelet aggregates, which in turn allows for increasing light transmission through the platelet suspension. Percentage of light transmission is the measure of platelet aggregation. Each patient is controlled by a normal relative. The result is either increased platelet aggregation (light transmission) above the normal control (when difference in light transmission is demonstrated between the patient and the normal control) or not (within normal platelet aggregability) when no difference in light transmission is demonstrated between the patient and the normal control (Koski, 1987, Metwally and Refaat 1995). FIGURE (1) PLATELET AGGREGATION FOR PATIENTS AND CONTROL

Both MRI and CT myelography were done according to the suspected clinical spinal levels of each patient as will be explained later. Standard T1,T2 and proton density weighted images were used in MRI examination. It should be noted that all patients were subjected to both MRI and CT myelography.

Nine patients with spinal tumours at the dorsolumbar regions were taken as controls (mean age 44 years). All patients were males and all were surgically confirmed. All patients were subjected to the same investigations done to the dorsal disc herniation patients, however both MRI and CT myelography were not done to every patients. see table (7)

RESULTS

- Results of clinical examination

The clinical picture of patients with degenerative dorsal disc disease was characterized by weakness in the lower limbs (bilateral with lateralization to one side). The weakness was proximal with atrophy in the quadriceps muscles (bilateral with lateralization to one side). Mild lower dorsal pain was demonstrated in only four patients (36%), the pain characteristically increased by walking and relieved by rest. Impotence was present in all patients (100%). Bladder disturbances, in the form of precipitancy of micturation, was present in 3 patients (27%). Marked fluctuation in the intensity of the clinical symptomatology was a cardinal and a very characteristic feature in all patients (100%) to the point that some of these patients were misdiagnosed as multiple sclerosis.

The whole clinical picture, of each patient, was composed of periods of marked increase of weakness in the lower limbs that alternates with periods of marked improvement of weakness. The improvement was spontaneous and unlike multiple sclerosis was more rapid (one or two days of bed rest can result in marked clinical improvement). Periods of aggravation of symptomatology usually had a sudden onset and are occasionally preceded by physical exertions. Initially exacerbation of symptomatology was of short duration with almost complete reversal of clinical symptomatology, however with the passage of time the patients started to notice that periods of exacerbation of symptomatology tended to be more frequent and to have a longer duration. Some residual neurological deficits, in the form of persistent proximal weakness and atrophy in the quadriceps muscles, are left behind in a cumulative fashion after each exacerbation. The approximate number of episodes of exacerbation of symptomatology and the duration of disease for each patient (time between onset of symptoms to clinical presentation to us) are listed in table (1). Approximately each patient had two episodes of exacerbation per year.

TABLE (1) The approximate number of episodes of exacerbation of symptomatology and the duration of disease
for each patient

<table>
<thead>
<tr>
<th>patient number</th>
<th>age</th>
<th>sex</th>
<th>duration of illness</th>
<th>approximate number of episodes of exacerbation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>male</td>
<td>1 year</td>
<td>3 episodes</td>
</tr>
<tr>
<td>2</td>
<td>39</td>
<td>male</td>
<td>1 year</td>
<td>4 episodes</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>male</td>
<td>1.5 years</td>
<td>2 episodes</td>
</tr>
<tr>
<td>4</td>
<td>47</td>
<td>male</td>
<td>1 year</td>
<td>3 episodes</td>
</tr>
<tr>
<td>5</td>
<td>42</td>
<td>male</td>
<td>2 years</td>
<td>5 episodes</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>male</td>
<td>2.5 years</td>
<td>5 episodes</td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>male</td>
<td>3 years</td>
<td>4 episodes</td>
</tr>
<tr>
<td>8</td>
<td>51</td>
<td>male</td>
<td>5 years</td>
<td>8 episodes</td>
</tr>
<tr>
<td>9</td>
<td>55</td>
<td>male</td>
<td>5.7 years</td>
<td>7 episodes</td>
</tr>
<tr>
<td>10</td>
<td>53</td>
<td>male</td>
<td>6 years</td>
<td>9 episodes</td>
</tr>
<tr>
<td>11</td>
<td>69</td>
<td>male</td>
<td>8 years</td>
<td>12 episodes</td>
</tr>
</tbody>
</table>

It must be noted that the exacerbations were painless in all patients, even in the subgroup of patients with mild dorsal pain. The pain in this group was chronic, its intensity used to fluctuate (being aggravated by walking and relieved by rest) and it was not temporally related to motor exacerbations.

Weakness in the lower limbs was aggravated by walking and relieved by rest in 9 out of the 11 patients (82%) and this constituted another characteristic feature. No history of spinal trauma was demonstrated in any patient.

Clinical neurological examination, in all patients with dorsal disc disease, was characterized by a combination of upper and lower motor neuron manifestations in the lower limbs, mainly in the form of mild to moderate atrophy of the quadriceps muscle (bilaterally and asymmetrically) with diminished knee and occasionally ankle jerk reflexes. Extensor planter response was present bilaterally in all patients.

Radicular sensory manifestations (mainly in the form of diminished superficial sensations in the L2, L3, L4 spinal segments and the saddle shaped area) were present in all patients, however sensory manifestations were not a prominent complaint. Sensory manifestations were detected only by careful clinical examinations and were occasionally most prominent contralateral to the side with more weakness. Motor manifestations (in the form of weakness and atrophy) predominated the clinical picture in all patients. The cremasteric reflex was diminished in all patients. All the clinical signs were bilateral with marked asymmetry and lateralization to one side. A sensory level at D6, D7 spinal segments was demonstrated in all patients.

Examination of the upper limbs was normal and no other neurological abnormalities were demonstrated in any patients. It is interesting to note that all patients were hypertensive on admission with a mean blood pressure of (195/115). All patients were admitted during an episode of symptom exacerbation.

The clinical picture of patients with spinal tumours in the dorso-lumbar regions was similar to that of the dorsal disc disease with the following differences

1-Patients with spinal tumours had a disease course characterized by persistent gradual progression of clinical signs and symptoms. Periods of spontaneous improvement have never been reported by any patient.

2-The clinical picture of patients with spinal tumours in the dorso-lumbar regions was collectively pointing to a lesion in the cauda-conus part of the spinal cord, as it was the case in patients with dorsal disc herniation (with a combination of both upper and lower motor neuron manifestations in the lower limbs together with diminished sensation in the saddle shaped area, lost cremasteric reflex etc.). However, and unlike patients with dorsal disc
disease, no sensory level at D6, D7 spinal segments was demonstrated in any patient with spinal tumours in the dorso-lumbar regions.

3-None of the patients with spinal tumours in the dorso-lumbar regions reported vertebral pain or weakness aggravated by walking and relieved by rest. In this group both pain, when present, and weakness were persistent and progressive.

4-Non of the patients with spinal tumours in the dorso-lumbar regions were hypertensive on admission.

5-Sensory manifestations were as prominent as the motor manifestations.

The existence of a sensory level at D6, D7 spinal segments was confusing in patients with dorsal disc disease. Although the clinical picture was collectively pointing to a lesion in the cauda-conus part of the spinal cord (with a combination of both upper and lower motor neuron manifestations in the lower limbs together with diminished sensation in the saddle shaped area, lost cremasteric reflex etc.), however a sensory level at D6, D7 spinal segments is far above the previous level and this frequently led to an erroneous diagnosis of a long lesion, such as arteriovenous malformation or syringomyelia, or multiple lesions.

- Results of haemorheological studies and vascular risk factors

Results of ECG, Serum fibrinogen, haematocrit value, serum lipid and serum lipid electrophoresis, blood sugar and platelet aggregation of patients with dorsal disc disease are summarized in table (2)

Table (2) Results of haemorheological studies and vascular risk factors

<table>
<thead>
<tr>
<th>NUMBER</th>
<th>age</th>
<th>sex</th>
<th>ECG</th>
<th>fibrinogen</th>
<th>lipid</th>
<th>platelet aggregation</th>
<th>diabetes</th>
<th>haematocrit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>M</td>
<td>normal</td>
<td>650mg/dl</td>
<td>normal</td>
<td>INCREASED</td>
<td>+</td>
<td>49</td>
</tr>
<tr>
<td>2</td>
<td>39</td>
<td>M</td>
<td>normal</td>
<td>560mg/dl</td>
<td>normal</td>
<td>INCREASED</td>
<td>normal</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>M</td>
<td>normal</td>
<td>400mg/dl</td>
<td>normal</td>
<td>INCREASED</td>
<td>+</td>
<td>48</td>
</tr>
<tr>
<td>4</td>
<td>47</td>
<td>M</td>
<td>normal</td>
<td>610mg/dl</td>
<td>normal</td>
<td>INCREASED</td>
<td>normal</td>
<td>44</td>
</tr>
<tr>
<td>5</td>
<td>42</td>
<td>M</td>
<td>LVH</td>
<td>570mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>49</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>M</td>
<td>normal</td>
<td>480mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>47</td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>M</td>
<td>LVH</td>
<td>600mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>42</td>
</tr>
<tr>
<td>8</td>
<td>51</td>
<td>M</td>
<td>LVH</td>
<td>580mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>43</td>
</tr>
<tr>
<td>9</td>
<td>55</td>
<td>M</td>
<td>LVH</td>
<td>550mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>40</td>
</tr>
<tr>
<td>10</td>
<td>53</td>
<td>M</td>
<td>LVH</td>
<td>630mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>47</td>
</tr>
<tr>
<td>11</td>
<td>69</td>
<td>M</td>
<td>LVH</td>
<td>600mg/dl</td>
<td>IV hyperlipidemia</td>
<td>INCREASED</td>
<td>+</td>
<td>40</td>
</tr>
<tr>
<td>mean</td>
<td>46</td>
<td>-</td>
<td>-</td>
<td>566mg/dl</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>44.272</td>
</tr>
</tbody>
</table>

It is interesting to note that 6 out of eleven patients with dorsal disc disease (55%) showed evidence of hypertensive left ventricular hypertrophy (LVH), 9 patients (82%) were diabetic, 7 patients (64%) had type IV hyperlipidemia (increased triglyceride and reduction of high density lipoprotein). All patients had elevated serum fibrinogen, see table (3) and increased platelet aggregation compared with the normal control. see table (2)

None of patients with spinal tumours had evidence of hypertension or LVH. Also non of these patients were diabetic or showed evidence of hyperlipidemia. Both serum fibrinogen (mean value 260 mg/dl), See table (3), and platelet aggregation were normal in this group.
The mean haematocrit value of patients with dorsal disc disease was higher than that of patients with spinal tumours (mean 37) and the difference was statistically significant. See table (4)

### Table (3) fibrinogen in patients with dorsal disc disease compared with that of spinal tumours

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>dorsal disc disease</th>
<th>spinal tumours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Mean</td>
<td>566</td>
<td>260</td>
</tr>
<tr>
<td>ST</td>
<td>106</td>
<td>26.1</td>
</tr>
</tbody>
</table>

T VALUE=7.68, DF=27, P>0.001

### Table (4) haematocrit value of patients with dorsal disc disease and spinal tumours

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>dorsal disc disease</th>
<th>spinal tumours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Mean</td>
<td>44.272</td>
<td>37</td>
</tr>
<tr>
<td>ST</td>
<td>2.64</td>
<td>1.034</td>
</tr>
</tbody>
</table>

T VALUE=10.20, DF=26, P>0.001

To put things together, it is quite evident that patients with dorsal disc disease had a higher incidence of vascular risk factors (non insuline dependent diabetes, hypertension, hyperlipidemia, LVH and increased platelet aggregation). Haemorheological changes consistent with increased whole blood viscosity and increased thrombotic tendency of the blood were also found in this group of patients with dorsal disc disease (increased haematocrit value, increased fibrinogen level, increased platelet aggregation etc.).

It should be noted that non of the patients with dorsal disc disease showed evidence of cerebral, cardiac or peripheral ischaemia either by history or clinical examination, also non of them were known to be diabetic or hypertensive before admission.

- **Results of radiological studies**

Results of radiological studies for patients with dorsal disc disease are summarized in table (5)

### Table (5) : Results of radiological studies

<table>
<thead>
<tr>
<th>No</th>
<th>PLAIN X RAY</th>
<th>MYELOGRAPHY</th>
<th>CT MYELOGRAPHY</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal in patients number 1,3,4,5,7</td>
<td>1- No abnormalities detected at the region corresponding to the sensory level</td>
<td>1-No abnormalities detected at spinal segments corresponding to the sensory level.</td>
<td>2-Centrolateral disc herniation , the disc</td>
</tr>
<tr>
<td>Case</td>
<td>MRI Findings</td>
<td>Disc Calcification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-------------</td>
<td>-------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-8</td>
<td>Normal</td>
<td>Minor indentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>patients number 2,6,8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>calcified disc herniation (left in patients number 1,2,4,5,8 and right in patients number 3,6,7) inducing canal stenosis and spinal cord deformation and displacement</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>is isointense, to hypointense relative to the normal disc material on the T1 images and hypointense on the T2 images, the herniated disc is inducing canal stenosis and spinal cord deformation and displacement.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Normal</td>
<td>Minor indentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1- No abnormalities detected at spinal segments corresponding to the sensory level.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2- left dorso lateral heavily calcified disc herniation inducing canal stenosis and spinal cord deformation and displacement</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3- The disc is signal void on all pulse sequences.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Normal</td>
<td>Minor indentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1- No abnormalities detected at spinal segments corresponding to the sensory level.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-Central heavily calcified disc herniation inducing canal stenosis and spinal cord deformation and displacement</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3- The disc is signal void on all pulse sequences.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Disc calcification</td>
<td>Complete myelographic blockade</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-Central heavily calcified disc herniation at level D11, D12.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-Spinal cord atrophy with central gray matter cavitation at the level of disc herniation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3-Spinal cord atrophy 6-7 segments above the herniated disc (pencil-shaped necrosis).</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1- The disc is signal void on all pulse sequences.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-The spinal cord is atrophic 6 spinal segments above the level of disc herniation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3-Central grey matter cavitations could not be appreciated by MRI.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Plain X ray of the dorsal spine was either normal or showed non specific degenerative changes in all cases with dorsal disc disease. Disc calcification was demonstrated in only one case (case number 11). See table (5).

Both MRI and myelography with postmyelography CT scan were done to all patients with dorsal disc disease. Both the dorso-lumbar regions and the spinal segments corresponding to the sensory level were scanned.

FIGURE [2] DORSAL DISC CALCIFICATION

Myelography was normal in 5 cases (45%) even when examined in retrograde. It showed minor indentation that can easily be overlooked or underestimated in anther 5 cases(45%) and complete myelographic blockade in only one case(10%) (case number 11). See table (5).

Scanning the spinal cord at spinal segments corresponding to the sensory level did not reveal any pathological changes in the spinal cord, subarachnoid spaces, epidural spaces, vertebrae or paravertebral regions in any patient except in one patient (patient number 11). In this particular patient the spinal cord was markedly atrophic with wide subarachnoid spaces at spinal segments corresponding to the sensory level. Progressive
downward scanning revealed that the spinal cord was atrophic from the D6 spinal segment down to the conus-epiconus region of the spinal cord at the dorso lumbar vertebral region. A heavily calcified disc herniation was demonstrated at D11,D12 level in this patient and the spinal cord showed the characteristic central gray matter cavitations opposite to disc herniation, Metwally and Refaat 1995. In this particular case the spinal cord was atrophic at least 6 spinal segments above the level disc herniation. See table (5).

Both MRI and CT myelography demonstrated dorsal disc herniation inducing variable degrees of canal stenosis, spinal cord displacement and/or deformation. The D11, D12 level was the most frequently involved level, see table (6). Although disc calcification was demonstrated in only one case by plain x ray, however evidence of disc calcification was demonstrated in all herniated discs by CT scan. The signal intensity of the T1,T2 weighted images was variable according to the degree of disc calcification (mostly isointense, to hypointense relative to the normal disc material on the T1 images and hypointense on the T2 images). Heavily calcified discs were signal void on all pulse sequences.

Table 6 Percentage of dorsal disc herniations at the various dorsal disc levels

<table>
<thead>
<tr>
<th>level</th>
<th>Patients number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>D9-D10</td>
<td>Patient number 7,1 ( 2 patients)</td>
<td>18%</td>
</tr>
<tr>
<td>D10-D11</td>
<td>Patient number 6 (one patient)</td>
<td>9%</td>
</tr>
<tr>
<td>D11-D12</td>
<td>Patient number 2,3,4,5,8,9,11 ( 7)</td>
<td>64%</td>
</tr>
</tbody>
</table>
In MRI examination, the most useful sequence was the sagittal T1 pulse sequence. It should be noted that MRI examination failed to demonstrate the central grey matter cavitations that were clearly demonstrated by CT myelography. We always felt that CT myelography (compared with MRI) is much better in identification of the exact shape and location of the herniated dorsal disc material. It should be noted that only a single disc herniation was found in every patient and multiple herniations was not demonstrated in any patient.

The mere fact that all herniated discs were calcified points to the chronicity and the long duration of disc disease that probably antedates the onset of clinical symptoms by a long time. This is consistent with our findings since the herniated dorsal discs in patients number 9,10,11 were heavily calcified while those of other patients were only partially calcified. Patients number 9,10,11 had the longest duration between the onset of symptoms to the time of clinical presentation to us. see table (1,5)

In patients number 11,9,7,4,1 at least two neuroimaging studies (CT myelography and/or MRI), done during previous episodes of exacerbations, were presented by the patients when they finally came to us. Comparison between the neuroimaging studies done before presenting to us with those done after clinical presentation to us did not reveal any add-on pathological structural differences except in patients number 11. In this patient the only differences revealed by comparison were the spinal cord atrophy and the central grey matter cavitations which were demonstrated when the patient presented to us and were not seen in the radiological study (CT myelography) done 8 month earlier. It must be stressed, however, that no intradural pathologies were demonstrated in any patients.

Results of radiological studies for patients with spinal tumours are summarized in table (7)

<table>
<thead>
<tr>
<th>NUMBER</th>
<th>PLAIN X RAY</th>
<th>MYEOGRAPHY</th>
<th>CT MYEOGRAPHY</th>
<th>MRI</th>
<th>SURGICAL FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NORMAL</td>
<td>Complete myelographic blockade at D11, D12</td>
<td>CT MYEOGRAPHY AND PLAIN CT SCAN SHOWED EXTRADURAL MASS WITH PARAVERTEBRAL EXTENSION</td>
<td>NOT DONE</td>
<td>DUMB-BELL NEUROFIBROMA AT D11,D12</td>
</tr>
<tr>
<td>2</td>
<td>NORMAL</td>
<td>Complete myelographic</td>
<td>EXTRADURAL HYPODENSE</td>
<td>NOT DONE</td>
<td>DERMOID TUMOUR AT D10, D11, D12</td>
</tr>
</tbody>
</table>
Results of surgical findings in group with dorsal disc disease

A partially or heavily calcified hard discs were removed surgically from all patients, no other pathologies were found. The dura was not opened and no comment could be made on the spinal cord status. Postoperatively all patients showed good clinical improved (except patient number 11) and follow up for the following year showed that the exacerbations that used to recur at least twice yearly ceased to occur.

DISCUSSION

Despite the protean presentations of dorsal disc herniations, attempts have been made to define subsets of characteristic clinical syndromes. Benson and Bynes, 1975, Arce and Dohrmann, 1985 (a,b), described two groups of patients depending on the association with either acute spinal trauma or degenerative disc disease. When associated with acute spinal trauma, patients presents within one day to one month from their injury with spinal cord or radicular symptomatology caused by soft disc herniation. With prompt treatment the prognosis in these patients is good and complete recovery is the rule. The spinal trauma in this group is commonly in the form of a fall landing on feet and buttocks often with an element of axial spinal twisting motion or rotation while falling, Patrick, et al, 1965.

Spinal cord compression by the space occupying nature of the herniated soft discs is the aetiopathogenic factor in this case and the clinical picture is straight forward and usually in the form of paraparesis or paraplegia with a level following a definite spinal trauma. This group of patients constitute less than 10% of all symptomatic dorsal disc herniations. Arce and Dohrmann, 1985 (a,b)
When associated with degenerative changes of the dorsal discs and spine, patients reported a prolonged history (up to 26 years in some patients, Arce and Dohrmann, 1985 a,b) and only 20% gave a history of suggestive of spinal trauma. Patients belonging to this group (with degenerative dorsal disc disease) constitute over 90% of cases with dorsal disc herniations. The prognosis of surgical treatment in this group is variable and generally not as good as the group of patients with traumatic dorsal disc herniation. Arce and Dohrmann, 1985 (a,b), Dietze and fessler, 1993

From the anatomical and biomechanical point of view the dorsal spine is divided into three divisions (1) D1-D4 or upper dorsal spine, (2) D5-D9 or middle dorsal spine and D10-D12 or dorso lumbar spine. Dorsal disc herniations in the upper dorsal spine are extremely rare and require violent trauma, Gelch, 1978, Hann, 1980, Lloyd et al, 1980, Kumar and Nuckley, 1986, Fessler, et al, 1991, Fessler and Dietze, 1992. Herniations in the middle dorsal spine are also uncommon, and mostly traumatic because of the stabilizing effect of the rib cage, Andriacchi et al, 1974. The dorso-lumbar division is a transition zone of vertebral configuration and facet orientation, with the highest mobility which predisposes it to degenerative changes and failure under relatively minor stress, White and Panjabi 1990, Maiman and Pintar, 1991. Degenerated discs are weak discs and might herniate spontaneously or because of minor trauma that can pass unnoticed by the patients. In fact this would explain why most (over 90%) of herniated dorsal discs are present in the dorso-lumbar division and occur in the absence of a definite spinal trauma. Epstein, 1983, Arce and Dohrmann, 1985 (a,b), Lesoin, et al, 1986, White and Panjabi. 1990

Degenerative dorsal disc herniations are commonly hard (calcified) on symptom formations, thus denoting that they have been silently there a long time before the clinical presentation. The protean clinical manifestations of degenerative dorsal disc herniations have commonly led to delayed diagnosis and misdiagnosis and can not be explained solely in terms of "a space occupying lesion (the herniated discs) compressing the spinal cord and inducing paraplegia or paraparesis with a level". Carson et al, 1971

The cardinal clinical signs and symptoms of dorsal disc herniations in this study are "a mainly motor presentation", the presence of remissions and exacerbations of myelopathy and the presence of two levels in every single patient (one at the conus -epiconus and anther sensory level at D6-D7 spinal segments). History of spinal trauma was characteristically absent and dorsal pain was minor and present in only 4 cases (36%). Delayed diagnosis, and occasionally misdiagnosis, was the rule in all cases. Misdiagnosis included multiple sclerosis, motor neuron disease, motor neuropathy or myopathies. Although fluctuations of clinical myelopathic symptomatology were reported by many studies, however, no attempt was made to explain this phenomenon in any of these studies. Arseni and Marisius, 1970, Arce and Dohrmann, 1985 (a,b), Maiman, et al, 1984, Stillerman and Weiss, 1991 (see table 8)

Table (8) Comparison between the clinical picture of dorsal disc disease in this study and some other studies

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean duration of symptoms</td>
<td>4 years</td>
<td>No comment</td>
<td>No comment</td>
<td>4 years-26 years</td>
<td>1 years</td>
<td>4 years</td>
</tr>
<tr>
<td>Weakness in the lower limb aggravated by walking</td>
<td>90%</td>
<td>No comment</td>
<td>No comment</td>
<td>No comment</td>
<td>No comment</td>
<td>No comment</td>
</tr>
<tr>
<td>Dorsal pain</td>
<td>36%</td>
<td>78%</td>
<td>57%</td>
<td>No comment</td>
<td>8%</td>
<td>81%</td>
</tr>
<tr>
<td>History of trauma</td>
<td>0%</td>
<td>22%</td>
<td>10%</td>
<td>12%</td>
<td>15%</td>
<td>11%</td>
</tr>
<tr>
<td>Cauda-conus syndrome</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>
*MEAN DURATION OF SYMPTOMS REFERS TO THE DURATION BETWEEN THE ONSET OF CLINICAL SYMPTOMS TO THE TIME OF THE CORRECT CLINICAL DIAGNOSIS.

Consistent with the view point of Chamber 1988, Vanderburgh and Kelly 1993, all dorsal disc herniations, in this study, occurred at D9-D10 through D12-L1 with the single most common level at D11-D12. see table (6)

Although the presence of disc calcification in the dorsal region should direct the attention to the possibility of dorsal disc herniation, however and according to our results, disc calcification in a way that can be demonstrated by plain x ray is present only in the minority of cases and its absence does not exclude the diagnosis of degenerative dorsal disc herniations. Singounas and Karvounis, 1979, Otani et al 1988, Ogilvie, 1991, Dietze and Fessler, 1993

Myelography was normal in 45% of cases and showed only minor indentation of the contrast column (that can easily be overlooked or underestimated) in another 45% of cases. According to love and Mefer, 1950, Baker et al, 1965, Metwally,1991, Dietze and Fessler, 1993, myelography is not uncommonly normal in symptomatic dorsal disc herniations and it leads to the correct preoperative diagnosis of dorsal disc herniation in only 56% of cases, Dietze and Fessler, 1993. In our opinion the myelographic findings can only be explained by the fact that the volume of the herniated dorsal disc material is usually small, inducing dorsal canal stenosis rather than complete myelographic blockade and major spinal cord compression. This simply means that the herniated dorsal disc material contributes to the overall clinical picture by inducing dorsal canal stenosis rather than through major compression of the neural structure. Logue, 1952, Love and Schorn, 1965, McAllister and Sage, 1976, Barnett, et al, 1987

Post myelography CT scan was the gold standard for the diagnosis of all the herniated dorsal discs in this study. All herniated disc were partially or heavily calcified (degenerative, non-traumatic). See table (5) This is consistent with the view point of Chowdhary, 1987, Fessler at al, 1991, Hamilton and Thomas, 1990, stillerman and weiss, 1991. According to the view point of Sekhar and jannetta, 1983, Chin et al, 1987, shikata et al, 1988, Dietze and Fessler, 1993, multiple dorsal disc herniations in a single patient is such a rare entity and was demonstrated in single patients in this study. See figure 6, Although intradural migration of disc fragments were reported before in dorsal disc herniations, Chowdhary 1987, however it has not been recorded in any of the patients examined in this study.

Consistent with the view point of Vanderburgh and kelly, 1993, we found the sagittal T1 MRI images to be the most useful sequence in identifying dorsal disc herniations. Our recent experience, however, has demonstrated that the T2 images are equally informative. See figure (6), Although calcification, which is very common in degenerative dorsal disc herniations, results in signal void in all pulse sequence, however it can diminish the conspicuity of the herniated dorsal discs on MRI, Ross et al, 1987. Because MRI has a high false positive

By comparing cases with spinal tumours with cases with dorsal disc disease it was quite evident that major differences between the clinical picture of each group were present. These differences can neither be explained by the anatomical site (both were sharing common anatomical sites) nor by the space occupying nature of the offending pathology (radiologically spinal tumours were occupying more space than the herniated discs and subsequently were exerting more pressure on the neural structures)

Although the clinical picture of patients with spinal tumours were straight forward and can easily be explained in terms of space occupying lesions compressing the nearby neural structures, however things for dorsal disc herniations were more complex and indeed "not that much simple". In degenerative dorsal disc disease other factors must be working, behind curtains, and in conjunction with the calcified herniated discs in the pathogenesis of myelopathy and in clinical symptoms formation. In this situation the epidural calcified discs are simply the iceberg of the whole problem.

In this study, and unlike patients with spinal tumours in the dorso-lumbar region, patients with dorsal disc disease had a higher incidence of vascular risk factors (non insuline dependent diabetes, hypertension, hyperlipidemia, LVH and increased platelet aggregation). Arteriosclerosis is common when vascular risk factors are present. Haemorheological changes consistent with increased whole blood viscosity and increased thrombotic tendency of the blood were also found in this group of patients with dorsal disc disease (increased haematocrit value, increased fibrinogen level, increased platelet aggregation etc.).

As a point of departure as quick over view on the blood supply of the dorsal spinal cord will be given. The dorsal spinal cord receives from 2-4 anterior radicular arteries which commonly arise from the aortic intercostals. However the principal arterial blood supply of the dorso-lumbar spinal cord is the artery of adamkiewicz. This vessel usually arises on the left side some where between D9-L1 and then enters the spinal canal through the corresponding intervertebral foramen and then it ascends for a variable distance to reach the midline, mostly at D10. It then divides into a small, narrow ascending branch (to the mid dorsal spinal segments) and a larger descending branch (to the dorso-lumbar spinal segments), Sekhar and jannetta, 1983, Lesoin, et al, 1986. The dorsal spinal cord has the greatest length between radicular arteries, which means that occlusion of one of these vessels may seriously compromise the blood supply to the corresponding spinal segments. Arce and Dohrmann, 1985 (a,b ), Metwally, 1991.

For dorsal disc herniation below D8, Maiman et al, 1984, performed selective angiography of the artery of adamkiewicz and found evidence of irregularity, elongation and tortuosity consistent with selective arteriosclerotic changes of this important radicular artery. Angiography for preoperative assessment of the

Figure [6] MRI T2 images showing herniated dorsal discs at D11,D12 and D12,L1. The D11,D12 herniation is indenting and mildly displacing the spinal cord. Notice the spinal cord signal changes at D11,D12 disc level (possibly due to spinal cord edema or ischemic changes). The herniated disc at D11,D12 disc level is signal void at all pulse sequences due to calcification.
status and the exact anatomical localization of this artery was thus suggested by some authors. Perot and Munro, 1969, Abramovitz, 1993

This is consistent with the necropsy findings of Manen, 1966 and Jellinger 1967. 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 relation to the vascular myelopathy caused by cervical spondylosis. According to the necropsy results of Jellinger, 1967 these arteriosclerotic changes 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 accelerates the arteriosclerotic changes in the region of the cervical enlargement. Metwally and Refaat 1995.

By putting things together it is quite apparent that, In dorsal disc disease, the natural history of myelopathy is determined an interaction between 3 main pathogenic factors, spondylitic factor, vascular factor and haemorheological factor.

The spondylitic factor ultimately results in bony and soft tissue hypertrophy that causes dorsal canal stenosis, and encroaches upon the subarachnoid space, reducing its volume. Lack of the CSF cushioning effect will cause embarrassment of the spinal circulation at the level of disc herniation, since optimum blood supply to the spinal cord needs an optimum CSF cushioning effect. The spondylitic factor probably also accelerate the arteriosclerotic changes in the spinal radicular arteries as will be explained later. Metwally and Refaat 1995

The second factor is the vascular factor. The dorsal spondylitic myelopathy patients comprised a group of patients where the incidence of vascular risk factors was found to be very high. The incidence of arteriosclerosis is known to be high among patients with vascular risk factors. The artery of adamkiewicz, the major blood supply to the dorso-lumbar spinal cord was found to be arteriolosclerosis on selective spinal angiography in patients with dorsal spondylitic myelopathy. Perot and Munro, 1969, Maiman et al, 1984, Abramovitz, 1993. Arteriolosclerosis of the spinal cord radicular arteries in relation to spondylosis was also reported on postmortem studies by Manen, 1966, Jellinger 1967. Although Manen, 1966 and Jellinger 1967 reported their findings in cervical spondylitic vascular myelopathy, however and by extrapolation, their findings that can also applicable to myelopathy caused by dorsal disc disease. To the best of out knowledge no similar postmortem studies are available in myelopathy caused by degenerative dorsal disc disease probably because of the rarity of the disease.

Because Manen 1966 reported that arteriolosclerosis of the spinal cord radicular arteries in relation to spondylosis was an isolated phenomenon not dependant on age and was negatively correlated with arteriosclerosis in the rest of the body, Metwally and Refaat 1995 suggested the possibility that the injurious effect of the spondylitic changes accelerates the arteriosclerosis in the radicular arteries suppling the myelopathic segments of the spinal cord. Endothelial injury is universally accepted to be the triggering factor of arteriosclerosis, Spence, 1995, and it is possible that the continuous wear and tear of spondylosis induce a vascular endothelial injury of the radicular arteries, the arteries lying on the surface of the spinal cord or the intramedullary arteries thus setting up the substrate for the development of this segmental and focal arteriosclerosis of the spinal arteries. In this respect spondylosis could be regarded as an independent vascular risk factor that, unlike the classical risk factors, acts locally rather than systemically. More researches are needed to define the exact role played by spondylosis in the development of spinal arteriosclerosis.

The findings of manen, 1966 are also consistent with our findings since we did not find evidence (on clinical background) of cerebral, cardiac, or peripheral ischemia in any of the examined patients. The presence of this local vascular risk factor (spondylosis) is probably responsible for the acceleration of the segmental spinal arteriosclerosis that undoubtedly resulted in the earlier development of focal, segmental spinal cord ischaemic episodes (of sudden onset and regressive course). Absence of evidence of systemic arteriosclerosis, even in the presence of systemic risk factors, should simply mean that the role played by spondylosis in the development of focal spinal arteriosclerosis is a major and a determinant role and not just a minor role contributing to that of the systemic risk factors in the development of arteriosclerosis.

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 patients with dorsal disc disease. By comparing the current radiological studies with previous studies, in some patients, no additional compressing agents (like new soft disc herniation, change in the size of the existing herniated discs, or the development of new osteophytes) were demonstrated radiologically that can explain the clinical symptomatology in terms of compression of the spinal cord and/or an important radicular artery.

The mere fact that all herniated disc were calcified means that they have been there for a long time before the development of clinical symptomatology. Autopsy studies have suggestive an incidence of thoracic disc herniations between 7% to 15% in individual died without a neurological history suggestive of dorsal disc disease, Abbot and Retter 1956, Arseni and nash 1960. The incidence of asymptomatic dorsal disc herniation detected by incidental MRI or CT scan examinations in patients not presented with any neurological manifestations ranges between 11% to 13%, William and Cherryman, 1988, William et al, 1989, Awwad et al, 1991. According to Awwad et al, 1991, spinal cord deformation, flattening or distortion could be present, radiologically, even to a marked degree in asymptomatic individuals.

This simply means that a calcified dorsal disc herniation, in it self, might remain asymptomatic unless other factors are added to the clinical picture (i.e. focal spondylitic spinal arteriosclerosis and increase in the whole blood viscosity and thrombotic tendency of the blood) and the net clinical picture in those patients is the resultant of the interactions between those three factors (spondylosis, focal segmental spondylitic spinal arteriosclerosis and increase in the whole blood viscosity and thrombotic tendency of the blood). In short both spondylosis and arteriosclerosis serve by furnishing the background for the ultimate determinant of the clinical symptomatology. (increase in the whole blood viscosity and thrombotic tendency of the blood)

Dorsal spondylosis will result in canal stenosis, loss of the CSF cushioning effect and embarrassment of the spinal circulation in the dorso-lumbar segments of the spinal cord. 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. In response to increase in the whole blood viscosity, the spinal microvascular bed dilates to accommodate more blood, thus the spinal cord perfusion will be kept at a constant value despite the normal daily fluctuation of the whole blood viscosity. Loss of the auto-regulatory process, secondary to advanced arteriosclerosis, will simply mean that the spinal cord perfusion, in the vulnerable dorso-lumbar segments of the spinal cord, will fluctuate with fluctuation of the whole blood viscosity. Powers, 1992, Metwally and Refaat 1995.

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. Grotta, et al., 1982, Schneider et al., 1987. Blood viscosity is mainly determined by the hematocrit value and the plasma viscosity is mainly determined by the plasma fibrinogen level. High values of serum lipid have also been found to increase whole blood viscosity. Pearson et al., 1981, Pearson, 1987, Stoltz et al., 1981, Grotta et al., 1982, 1985. Increase in the platelet aggregation also increase 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. Lowe, 1987.

Although RBCs deformability and aggregability were not selectively tested in the present study, however the RBCs deformability is invariably reduced and their 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. Grotta, et al, 1985, Pearson, 1987.

The significant increase of the haematocrit values and the serum fibrinogen, especially when coupled with increased platelets aggregation and hyperlipidaemia, is an indicator of increased whole blood viscosity in patients with degenerative dorsal disc disease. Ott et al., 1979, Pearson et al., 1981, Stoltz et al., 1981, Bartoli et al., 1982, Grotta et al., 1982, 1985.
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. Powers, 1992, Metwally and Refaat 1995. Inverse correlation is present between the neuronal tissue blood flow and serum fibrinogen level and the haematocrit value. Grotta et al., 1985, Schneider et al., 1987.

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 hyperaggragable platelets, Pearson, 1987, or by red cell aggregation with impaction of rigid red cells in the microcirculation. Lowe, 1987. This is more likely to occur with the existence of high red cell mass (Hematocrit 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 (arteriolsclerosis) Koski, 1987, Schneider, et al., 1987.

The high blood viscosity observed in patients with dorsal disc disease should simply mean, especially when coupled with arteriosclerosis of the small perforating blood vessels, that the blood flow to the spinal cord, at the dorso-lumbar segments, is subjected to high vascular resistance that could ultimately result in low perfusion pressure and chronic ischaemia. The increased thrombotic tendency observed in those patients should mean that the chronic ischemic state could be interrupted by acute thrombotic microvascular occlusions that can result, pathologically, in spinal cord lacunar infarction and clinically in dorso-lumbar painless myelopathy of sudden onset and regressive course.

The acute thrombo-occlusive episodes are responsible for the intramedullary cavitations observed in patient number 11. Those cavitations, most probably, represent lacunar infarctions in the presumed anatomical sites of the anterior horns. The segmental spinal cord atrophy observed in this patient could be the result of long standing chronic ischaemia interrupted by recurrent thrombo-occlusive episodes. By analogy the sensory level that was demonstrated in all patients (other than patient number 11) could be the result of chronic dorsal spinal cord ischemia in the distribution of the artery of adamkiewicz that has not, yet, reached a magnitude sufficient to induce gross structural spinal cord ischemic changes (atrophy) that can be demonstrated radiologically.

The association between spondylitic myelopathy and spinal cord atrophy and/or cavitations was described before Tsuji, 1982, Jestico 1983, Furguson and Caplan, 1985, Penning et al., 1986, Jinkens et al., 1986. All these reports used only CT myelography and non used MRI. The pathology was collectively described without sufficient specification and no correlation with the clinical picture was made. A possible vascular aetiology for the spinal cord atrophy and/or cavitations was vaguely proposed by Furguson and Caplan, 1985 and Jinkens et al., 1986, but without defining in which way this vascular aetiology is implicated in the pathogenesis. No haemorheological study was done in any of the previous reports. However it should be noted that all previous studies were localized to the cervical region (cervical spondylitic myelopathy) and to the best of our knowledge non ever reported central gray matter cavitations or segmental spinal cord atrophy in dorsal disc disease.

The study of Metwally and Refaat 1995 is probably the only study where the radiological studies (both MRI and CT scan were used) were correlated with the clinical picture, vascular risk factors and haemorheological parameters. However, and still again, the authors only studied the cervical spondylitic myelopathy. It is interesting to note that although central gray matter cavitations and/or segmental spinal cord atrophy were demonstrated radiologically in all patients( with painless cervical spondylitic vascular myelopathy) in the study of Metwally and Refaat 1995, it was demonstrated in only one patient in this study (patient number 11) even though patients in both studies were sharing common vascular risk factors and haemorheological parameters. Basically the blood supply to the dorsal spinal cord is richer than the blood supply to the cervical enlargement which is known to be a watershed area with marginal blood supply. This last field zone (the cervical enlargement) is most likely to suffer from insufficiency of blood and has been shown to be a preferential zone for vascular damage. Tuli, 1975, Jellinger, 1967.
It looks like that the richness of the blood supply to the spinal cord is another factor determining the probability and the magnitude of vascular damage in the spondylitic vascular myelopathy. Spinal cord zones with a poorer blood supply are more likely to suffer earlier and to a graver degree than spinal cord zones with a richer blood supply. Consistent with the results of Metwally and Refaat 1995, we also found that exacerbation of myelopathy in dorsal disc disease to be intimately coupled temporally with rise of whole blood viscosity and thrombotic tendency. Metwally and Refaat 1995 also reported that the haemorheological parameters tended to drop down to the normal levels following the acute phase in spondylitic vascular myelopathy patients (thus explaining the spontaneous and rapid clinical improvement). It looks like that increased whole blood viscosity and thrombotic tendency of the blood are the ultimate determinant factors in the development the spondylitic vascular myelopathy.

The ischaemic aetiology of myelopathy, in patients with dorsal disc disease, is consistent with the necropsy findings of Jellinger, 1967. The author reported, in the spondylitic vascular 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.). Elshazli, 1984, Schneider, et al., 1987.

The haemorheological profile of vascular spondylitic myelopathy is also similar to the haemorheological profile of hypertensive micro-vascular brain disease (Lacunar infarction, leukoaraiosis etc.) previously reported by Elshazli, 1984, Schneider et al., 1987. The haemorheological parameters tended to drop down to the normal levels following the acute phase in myelopathy patients, Metwally and Refaat 1995 and this has also been reported in ischaemic brain disease, Koski, 1987.

The hypertensive micro-vascular brain disease was 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 table (9).

<table>
<thead>
<tr>
<th>Vascular risk factors</th>
<th>Hypertension, NIDDM, type IV hyperglycaemia, old age, LVH are common in both diseases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathological findings</td>
<td>Neuronal degeneration, ischaemic demyelination, diffuse lacunar state are common in vascular myelopathy (Jillenger, 1967) and in hypertensive microvascular brain disease (Hachinski, et al., 1987, Leifer, et al., 1990).</td>
</tr>
<tr>
<td>Haemorheological profile</td>
<td>Increased whole blood viscosity and increased thrombotic tendency are common in vascular myelopathy (Metwally and Refaat 1995, this study) and in ischaemic brain disease (Koski, 1987, Schneider et al., 1987).</td>
</tr>
</tbody>
</table>

Spinal cord ischaemia is far much less well studied compared with cerebral ischaemia. Practically little attention was given in literature to the exact spinal cord pathological findings in the spondylitic vascular myelopathy, due to dorsal disc disease, and how is it correlated with any vascular aetiology and/or haemorheological abnormalities and in which way the spondylitic process is related to the whole problem.

Although Perot and Munro, 1969, Maiman et al. 1984, Abramovitz, 1993 demonstrated, on selective spinal
angiography, that the artery of adamkiewicz is commonly arteriosclerosed in patients with degenerative dorsal disc disease, however they did not mention whether their patients had evidence of vascular risk factors or haemorheological abnormalities or not. They did not comment on the probable aetiopathogenic factors responsible for the development of this selective and focal arteriosclerosis. They also did not mention the impact of their finding on the clinical picture or on the management of dorsal disc disease.

In fact the present study, to the best of our knowledge, is probably the only study where the dorsal spinal cord ischemic pathology was correlated with the haemorheological factors. This correlation was made necessary since the incidence of hypertension and NIDDM was found to be high among patients with spondylitic myelopathy due to degenerative dorsal disc disease. Haemorheological abnormalities are known to be common in diabetes and hypertension. Also the presence of calcification in all herniated dorsal discs and the static nature of the structural pathology on follow up studies had made it necessary to search for another aetiology of the ischaemic episodes. Calcified discs are chronic pathologies and can not be held responsible for relapsing and remitting myelopathies.

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 dorsal disc disease and that due to multiple sclerosis as follows

- Unlike multiple sclerosis, myelopathy due to dorsal disc disease 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 dorsal disc disease is very short (on the average few hours to one or two days).

- Unlike multiple sclerosis, relapses of myelopathy due to degenerative dorsal disc disease 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 degenerative dorsal disc disease 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 anther misdiagnosis which is motor neuron disease or motor neuropathy. However myelopathy due to dorsal disc disease 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 dorsal disc disease is in fact anther 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. Marcus et al., 1977, Dawson and Potts, 1991.

In 82% patients the motor weakness was characteristically increased by walking and relieved by rest and this is anther point favouring the ischemic aetiology of myelopathy due to degenerative dorsal 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, Dawson and Potts, 1991. Dorsal canal stenosis (induced by the dorsal 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". Spinal cord claudication due to spinal canal stenosis was described before. Dawson and Potts, 1991

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 (patient number 11). Patient number 11 was the oldest with the highest total number of ischaemic episodes, see table (1). All patients, 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 they were referred to surgery once diagnosed radiologically.

Operatively the calcified discs were removed resulting in decompression of the spinal canal. Postoperatively all patients improved except patient number 11, probably due to the existence of significant preoperative spinal cord atrophy. All patients were followed up for at least one year postoperatively and they all showed persistent gradual improvement (of course except patient number 11) and recurrent episodes of myelopathy ceased to occur in all patients (including patient number 11). Spinal canal decompression will widen the spinal canal and restores the volume of the subarachnoid spaces thus restoring the cushioning effect of the CSF which is mandatory for optimum blood flow to the spinal cord. Focal spinal cord ischemia will undoubtedly improve following optimization of the cushioning effect of the CSF in the subarachnoid spaces, Bennett and McCallum, 1977, Safdari and Baker, 1985, Ebersold, 1986, Ridenour, 1991. Medical treatment for diabetes, hypertension, increased blood viscosity was continued Postoperatively and undoubtedly contributed to the clinical improvement and stabilization of the patients condition.

It is interesting to note that although patient number 11 did not show any clinical improvement postoperatively, however his condition did stabilize and recurrent episodes of myelopathy ceased to occur. It looks like that surgical and medical treatment are indicated even in the presence of gross spinal cord atrophy as this will, at least, prevent future attacks of myelopathy that will undoubtedly make the patient’s condition even worse.

Delay in the diagnosis and treatment of dorsal disc disease is very common (an average of 4 years in this study and as long as 26 years in some other studies, Arce and Dohrmann, 1985 a,b). Delay in the correct diagnosis could have serious implications because, over time, the cumulative effect of ischaemic episodes will induce irreversible structural neuronal damage that will render any surgical or medical treatment useless in so far as reversing or improving the patient neurological status is concerned.

In our opinion this delay has actually stemmed from the following points

1-A misconception that "all cases of dorsal disc herniations are traumatic in nature, absence of history of trauma usually excludes the diagnosis of dorsal disc herniation", in fact this statement is mentioned here only to condemned since over 90 % of cases of dorsal disc herniations are degenerative and not traumatic. Benson and Bynes, 1975, Arce and Dohrmann, 1985 (a,b)

2-A misconception that "a degenerative dorsal disc herniation symptomatizes by inducing spinal cord compression and is solely responsible for the clinical picture", still again this statement is mentioned here only to condemned since degenerative dorsal disc herniations contribute to the overall clinical picture by inducing dorsal canal stenosis, embarrassment of spinal circulation and probably acts as a local risk factor accelerating radicular arteriosclerosis and that the net clinical picture is the result of the interaction between the spondylitic, the vascular and the haemorheological factors. Degenerative dorsal disc herniations might remain asymptomatic for life if other factors are not added to the clinical picture. William and Cherryman, 1988, William et al, 1989, Awwad et al, 1991

3- Failure to understand that the clinical picture of degenerative dorsal disc herniations is not just "paraplegia with a level" and that it superficially resembles multiple sclerosis.

In general the vascular aetiology is the ultimate pathogenic factor of the spondylitic myelopathy whether in the cervical of the dorsal regions. The spondylitic vascular myelopathy is present mainly in males and is
characterized, clinically, by relapsing remitting painless myelopathies and radiologically by the presence of segmental spinal cord atrophy or cavitations. Incidence of vascular risk factors was high among patients with vascular myelopathy with frequent haemorheological abnormalities denoting increased whole blood viscosity. 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 table (10).

TABLE [10] PATHOGENESIS OF THE SPONDYLITIC VASCULAR MYELOPATHY

<table>
<thead>
<tr>
<th>Pathological phenomenon</th>
<th>Detrimental effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>dorsal spondylosis &amp; dorsal canal stenosis</td>
<td>1. Loss of the CSF cushioning effect with embarrassment of spinal cord circulation in the region of cervical enlargement or the dorso-lumbar spinal segments.</td>
</tr>
<tr>
<td></td>
<td>2. Accelerate arteriolosclerosis in the region of spondylosis.</td>
</tr>
<tr>
<td>hypertensive vascular changes in the dorso-lumbar spinal segments or the cervical enlargement</td>
<td>1. Stenosis of the perforating intramedullary arterioles in the region of cervical enlargement or the dorso-lumbar spinal segments.</td>
</tr>
<tr>
<td></td>
<td>2. Loss of the auto-regulatory physiological phenomenon of the stenosed arterioles</td>
</tr>
<tr>
<td>increased whole blood viscosity and thrombotic tendency of the blood</td>
<td>1. Chronic ischaemia in the region of cervical enlargement or the dorso-lumbar spinal segments.</td>
</tr>
<tr>
<td></td>
<td>2. Acute microvascular thrombo-occlusive episodes in the region cervical enlargement or the dorso-lumbar spinal segments.</td>
</tr>
</tbody>
</table>

This study, taken together with the study of Metwally and Refaat 1995, casted important light on what we prefer to call "The spondylitic spinovascular disorders" and we consider this terminology to be the most accurate and the most descriptive of this emerging new concept in which spondylosis interacts with vascular risk factors, segmental spinal arteriosclerosis, and haemorheological factors (increased whole blood viscosity and increased thrombotic tendency of the blood) in the pathogenesis of segmental myelopathy and in symptom formations. Metwally, 2000

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