MISDIAGNOSIS OF SPINAL CORD INJURIES— THE PHYSIATRIST'S POINT OF VIEW

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'The trouble with doctors is not that they don't know enough, but that they don't see enough.'

(*Sir Dominic J. Corrigan—1853*)

'Make it compulsory for a doctor using a brass plate to have inscribed on it, in addition to the letters indicating his qualifications, the words: Remember that I, too, am mortal.'

(*G. B. Shaw—from 'The Doctor's Dilemma'*)

Abstract. Thirteen different patients involving spinal cord pathology with delayed or false diagnosis are presented and these diagnostic pitfalls are discussed. Many seemingly simple explanations for para or quadriplegia are suddenly quite complex upon scrutiny. The problem of proper diagnosis is capital in instituting proper therapy, advising the patient and his family, and unfortunately in today's medical climate, for medical legal reasons.

Key words: Spinal cord injuries; Misdiagnosis; Pitfalls in diagnosis of spinal cord injuries or diseases.

Case Reports

We previously reported two cases of spinal cord tumours occurring some 30 years after childhood poliomyelitis (Ohry *et al.*, 1975). The first case involved a 34-year-old woman with a retroperitoneal neurogenic sarcoma. The second patient, a 52-year-old woman, had a meningioma removed from the D10 level at age 30. At first the progressive neurologic deterioration was thus described: 'late post-poliomyelitis amyotrophic lateral sclerosis like picture' (Mulder *et al.*, 1972), rather than neoplastic aetiology. These cases help support our hypothesis relating old polio-virus infection and tumours (Ohry *et al.*, 1978).

The third case was a 22-year-old man who was admitted to the orthopaedic department with a 7-week history of severe low back pain. Conservative therapy was unsuccessful. Myelography showed a stop of dye flow at the D12 level. Laminectomy of D12-L3 vertebrae showed a brown bulge at the L2 level. A diagnosis of ependymoma or glioma was made without biopsy confirmation. The neurologic picture deteriorated and repeat myelography showed continued dye flow. A course of radiation was given to the lumbar vertebrae with dramatic improvement of the neurologic picture and disappearance of pain. Sixteen years later the neurologic signs returned, with urinary disturbances, lower limb weakness, and low back pain. The diagnosis at this time was radiation myelopathy. After 3 months, with worsening of the neurologic picture, he was operated on again and a herniated nucleus pulposus was found. No evidence of arachnoiditis or tumour was noted. The neurologic picture improved markedly. No neurogenic bladder was demonstrated on cystometrogram. The conclusion here is that there were two episodes of disc herniation, and that the first was misdiagnosed as 'tumour'.

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The fourth case, a 25-year-old man, had poliomyelitis at the age of 1 year. The residuum was paresis of one leg. During military service he had episodes of urinary calculi without urinary or sexual disturbances. At the age of 23 he had had a severe subarachnoid haemorrhage from a small aneurism of the inferior branch of the right temporal artery. He refused surgery, but after a few months another angiography, this time of the spinal arteries, showed severe arteriovenous malformation of the lower dorsal cord. A second subarachnoid occurred and he underwent a D11-L2 laminectomy with the excision of a huge A-V malformation. He suddenly became a D11 paraplegic, with partial recovery. Retrospectively it appears the 'polio' and subarachnoid haemorrhage were probably several episodes of bleeding, and part of the natural history of his A-V malformation (Tobin *et al.*, 1976).

Case five is a 39-year-old man who in 1958 presented with 'multiple neuritis'. He was subsequently diagnosed as cauda equina syndrome secondary to vascular malformation. His symptoms included spasms, pains, and weakness. After urinary retention appeared, myelography was reported as showing 'vascular malformation'. No treatment was given. Over the course of the following years his neurological picture worsened and he suffered all the complications of paraplegia: pressure sores with plastic repair, urinary tract infections, hydronephrosis, urinary calculi, urethral disturbance, perineal abscess, all of which resulted in a permanent suprapubic cystostomy. In 1971 he had a bout of pleuro-pericarditis with no isolation of bacterial, viral or mycobacterial agents. By 1977 he had progressed to C7-8 complete quadriplegia. Stiffness of his spine had been noted 3 years ago, and present X-ray studies showed a typical bamboo-spine. Retrospective evaluation of his X-ray studies points to a relentless progression of changes seen in ankylosing spondylitis. Retrospection of the old myelograph showed some non-specific changes. The pleuro-pericarditis, the diminishing of the sacro-iliac joint, and the bamboo spine give the diagnosis of Bechtereu's disease, causing spinal cord injury.

Case six deals with a 38-year-old blacksmith who had a 10-year history of premature atrial contractions, and a 4- to 5-year history of low back pain complaints. After lifting some heavy object his back pain suddenly worsened. Examination at that time showed a slight weakness in the legs with mild urinary tract disturbances. Bed rest and analgesics were the therapy then. Within 3 weeks he returned to his job. Soon thereafter spastic paraparesis with clonus and bilateral Babinski signs was noted. After an unsuccessful attempt at myelography, the patient refused further therapy or examination. However, a few days later he returned to the hospital incontinent and consented to a lumbar laminectomy of the L4-5 vertebrae with removal of a disc. He presently walks with a 'spastic' gait, using two canes. There remains some sphincter incontinence. Here, diagnosis seems to have been correct from the start, but treatment was too slow for a long time.

Case seven is a 22-year-old woman with hebephrenic schizophrenia hospitalised after a suicide attempt (jumping from a height of 12 metres). She was then returned to the psychiatric institution. It was only after an episode of falling that the neurologic signs of urinary disturbance, spastic and weak lower limbs were noted. She was re-examined and X-rays of the back revealed fracture dislocation of D5 on D6. Laminectomy and stabilisation with Harrington's rod were performed. She eventually recovered, walks unaided with an almost normal gait and has complete sphincter control. The hebephrenic-schizophrenic behaviour which resulted in her re-admission as a psychiatric patient delayed the discovery of the vertebral fracture-dislocation and ultimate paraparesis.

Case eight is a 27-year-old Russian-born immigrant with a history of childhood poliomyelitis. At age 16 she complained of hemiparesis, fatigue and headache which her physician related to the polio. Her condition deteriorated. She was operated on and a cervical neurinoma (Schwannoma) was removed. During the next 10 years she showed increasing neurologic signs. She had three subsequent operations, each with removal of more new growths from her spinal cord. Finally she became a complete C7 quadriplegic.

Case nine concerns a 35-year-old man wounded from a bomb blast in 1973. After a brief interval of unconsciousness he awoke with full memory but with headaches,

dizziness and odd sensations throughout his body. During the following days he complained of transient limb paralysis, incontinence and sexual inadequacy. Psychiatric evaluation concluded in 'shell shock'. Three years later he was admitted for psychiatric evaluation with complaints of urinary frequency, partial impotence, cervical and limb pains and disturbances in concentration. Physical examination revealed tenderness in the cervix and in the D4-5 area, and mild hyperaesthesia of the penis. Cystomanometry indicated an upper motor neuron type bladder, with autonomic dysreflexia occurring during bladder filling. Psychiatric evaluation revealed that the behavioural disorder reflected no concern with impotency. We concluded that he suffered from a mild myelopathy, a sphincter disturbance and a mild neurotic reaction secondary to cervical spine lesion.

The tenth case is a 66-year-old widow who suffered from hypertension and palpitations. She had been treated with valium and digoxin 1 year prior to admission. She developed mild paraparesis. Despite continual complaints not associated with any specific physical findings except a non-specific weakness in the lower extremities, her problem was felt to be psychosomatic. One month later she developed motor and sensory deficits in the lower limbs and loss of sphincter control. Myelography revealed a block at D2 and on surgical exploration a meningioma was removed. The final neurologic picture was that of Brown-Sequard syndrome with spastic gait and some bladder disturbances.

The 11th case is a 20-year-old Arab man. In 1967 he had been hospitalised with classical dysentery, rectal prolapse, malnutrition and dehydration. He had also had an episode of bloody diarrhoea. Treatment with fluids and antibiotics had improved his general condition and the rectal prolapse was repaired. In 1968-69 he had developed a picture of general fatigue, quadriparesis and areflexia. He had been diagnosed as having 'dermatomyositis' and after slight improvement, discharged. In 1972 urologic investigation showed a normal IVP and the incidental finding of spinabifida was noted. Voiding cystography showed a contracted bladder with evidence of an upper motor neuron lesion of the bladder. In 1976 he still had urinary complaints. Dermatomyositis is not the diagnosis here. The urinal tract problem and the rectal prolapse may have resulted from the spinabifida, and the episode in 1968-69 may have been a viral myelitis.

The 12th case deals with a 54-year-old man who had had poliomyelitis at age 20 which left one leg affected. His spinal column had been irradiated for this as was then accepted, and months later a skin cancer which had developed over the irradiated area had to be excised. In 1968 a basal cell carcinoma was removed from his forehead. In 1971 he became incontinent and his neurologic condition deteriorated. Both legs became weak, necessitating short leg braces and Canadian crutches, and later long leg braces. He presently is a flaccid D10-11 paraplegic with a lower motor neuron bladder. In 1973 he underwent a transurethral resection of the prostate in an attempt to diminish the high residual urine. Myelography showed many sites of 'arachnoiditis'. Here a possible explanation for the late deterioration might be spinal cord irradiation.

The 13th case is a 53-year-old former concentration camp inmate who immigrated from Rumania following World War II. Complaints of low back pain with unsuccessful symptomatic therapy for 2 weeks were followed by a course of traction in another hospital. This was stopped after 2 days because of unbearable pain. Urinary retention and lower limb paralysis followed, and myelography revealed a block at L3. An L2-L4 laminectomy was performed but only severe arachnoiditis was noted. No change occurred in the physical condition. She remained a D11 paraplegic. A second myelography later at our centre showed a block at the D11 level. A second, more rostral (D11-12) laminectomy 4 days after the first operation revealed a neurinoma attached to the D11-12 roots. Neurologically the patient remained a D11 paraplegic. The first, inconclusive laminectomy should have been followed immediately by the second, higher myelography.

A 54-year-old woman, healthy except for a cystocele, had suffered a mild contused shoulder in a car accident. Several weeks later she had developed disturbances in urination. Introvenous pyelography and renal function studies were normal. She began

voiding using pressure above the pubis to empty her bladder. The problem worsened. Independent neurological examination 2 years later revealed slight deltoid muscle flattening, and a slightly positive Babinski sign on the left, with normal sensation. X-ray studies of the lumbosacral spine showed discogenic injury to L5-S1, and areas having bilateral stenosis with anterior, lateral, and posterior osteophytes. Five years after the accident she was seen in our clinic. She had a large, atonic bladder. Residual urine was 1100 cc. No bulbocavernosus sign was noted. EMG showed no anal sphincter function, and cystometry revealed an atonic bladder. Cystoscopy showed trabeculation on the bladder wall and cystocele. Presently she is using intermittent catheterisation to empty the residual urine. Here the problem was probably not related to the cystocele, but rather to some spinal injury (of the lower motor neuron type) in the cauda equina area.

Discussion

Examination of our clinical material shows many patients either diagnosed late in the course of their illness or incorrectly diagnosed. The exact percentage of falsely diagnosed spinal cord injuries can be determined by careful examination of all case records to establish the date of onset and the time of diagnosis of spinal cord injury. This is vital for both treatment and prognosis, and to help establish criteria of compensation and needs. Retrospectively, of course, this is quite easy to do, and emphasises how easy it is to fall into traps in the course of the disease.

The literature dealing with misdiagnosis of spinal cord injuries and other neurologic diseases is not abundant. Cumes (1977) described a patient with incontinence, voiding difficulty and a history of several urinary tract infections. Misdiagnosis concluded in stricture and posturethral valves. Early treatment included urethral dilatation, transurethral resection and a left ureteral implantation. Final diagnosis was sacral dysgenesis with associated occult-spinal dysplasia, which was concluded after discovery of a small pockmark over the sacrum and an abnormal sacral X-ray. Patient number 11 had been diagnosed as having dermatomyositis, although later an occult spina bifida was found. It is also conceivable that the viral myelitis had entered the story as a further complication, and both the spina bifida and the myelitis resulted in urinary tract problems. It appears certain that the dermatomyositis was not the primary problem or root diagnosis.

Oesen *et al.* (1974) presented a case report in which the post-mortem findings included a malignant tumour whose extramedullary growth surrounded the spinal cord in cuff-like fashion, causing a compressing syndrome. Subsequent irradiation, followed by surgical intervention (D4, 5, 6) laminectomy with incomplete tumour excision, led to neurologic deterioration. He died 14 years later. Two years prior to death, his situation had worsened and he had claimed that post-surgical irradiation was the cause of this worsening. Post-mortem examination showed teleangiectasis covering the skin of his back (histology of his first operation 14 years before had showed Schwann cell sarcoma). Only connective tissue was noted around the cord, but traces of irradiation myelopathy were found in his spinal cord. Patient number 12 also suffered from what now appears to be irradiation myelopathy from spinal cord irradiation after poliomyelitis.

Multiple sclerosis is frequently misdiagnosed (Finelli, 1976; Kahl, 1975) as meningitis (Johnson, 1975; Sandler, 1975), cervical injury (Tomlin *et al.*, 1975), and tumours (Segal *et al.*, 1975). The pitfalls in diagnosis of lesions at the junction of the medulla and spinal cord have previously been discussed (Blumenthal, 1975; Kubera *et al.*, 1976). Mulder *et al.* (1976) discussed patients with amyotrophic lateral sclerosis (ALS) who survived more than 5 years.

Patient number 5 presented a possible vascular malformation of the cord with severe ankylosing spondylitis. Whether the first or second disease caused his spinal cord damage is pure conjecture. The literature on the neurologic complications of Bechterew's disease is sparse (Shenkin *et al.*, 1976; Young *et al.*, 1977). The vascular malformation may be considered as a cause of the development of a spine or ridge over the position surface of the vertebral body. This in turn can trap nerve roots as they are adjacent to the anterior tract. Our patient with flaccid paralysis and lower motor neuron type bladder offers evidence to support this theory.

Many of our misdiagnosed patients began their story with low back pain (Maruta *et al.*, 1976) resisting medical and surgical treatment. Such cases should be treated by combined supportive psychotherapy and physiotherapy with appropriate antidepressants.

In conclusion, we stress the need for re-examination of all clinical material to prevent the misdiagnosis of spinal cord injury and unnecessary complications of treatment, mistreatment or neglect.

SUMMARY

In this paper we describe 13 different patients with spinal cord pathology with delayed or false diagnosis.

We discuss the problem of misdiagnosis of these conditions. Retrospective and critical examination of clinical material is essential to establish criteria for proper treatment and prognosis of spinal cord problems. We stress the need for re-examination of all clinical material to prevent misdiagnosis of spinal cord injury and unnecessary complications of treatment, mistreatment or neglect.

RÉSUMÉ

Treize cas de diagnostics faux ou tardifs de pathologies de l'épine dorsale sont présentés et ces pièges diagnostiques sont discutés. De nombreuses causes, simples à première vue, de paraplégie ou de quadriplégie se révèlent complexes à l'observation. Le problème du diagnostic correct est capital pour instituer une thérapie adéquate, conseiller le patient et sa famille et malheureusement, dans le climat médical d'aujourd'hui, pour des raisons médico-légales.

ZUSAMMENFASSUNG

Dreizehn Fälle falscher oder später Diagnose pathologischer Erscheinungen am Rückenmark werden vorgestellt und diese diagnostischen Fallen werden diskutiert. Viele anscheinend einfache Gründe der Para- oder Tetraplegie enthüllen sich plötzlich als kompliziert bei gründlicher Untersuchung. Das Problem der richtigen Diagnose ist sehr wichtig für eine gute Therapie, für die Beratung des Kranken und seiner Familie und leider, im heutigen medizinischen Klima, für medizinisch-juristische Gründe.

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