Uncommon Disorders of the Lower Spinal Region A Report on Eleven Patients*

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Introduction. Prompt diagnosis of lower spinal diseases is essential for successful treatment. Appropriate medical therapy or surgical decompression arrests progression and reverses existing neurologic deficit. Delayed or improper diagnosis seriously jeopardizes the patient's ultimate neurologic status. Too often early symptoms are disregarded or passed over as insignificant.

Diseases of the lower spinal region caused by congenital malformation, trauma, spondylosis, demyelination, and metastasis are readily recognized; however, there are a variety of additional disease processes which affect the lower spine and its contents. These processes are either rapid and devastating or elusive and slowly progressive. A keen sense of awareness on the part of the physician will aid the recognition of these disorders. In this regard eleven patients with uncommon disorders of the lower spinal region are presented. All had delayed or problematic diagnoses and were encountered directly or indirectly during the past seven years. Viewed collectively these infrequently occurring disorders of the lower spine appear with some regularity. This review shows that adherence to tenets of neurologic localization, deduction of pathologic processes, and use of available laboratory tests often lead to the correct diagnosis.

The disease states presented are divided into five categories: metabolic, vascular, tumorous, infectious, and those due to physical agents. Some, but not all, additional disorders within these categories are discussed.

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Metabolic Disorders.

Patient 1. A 53-year-old steel worker complained of stiffness in his right ankle and knee for three months. He was treated with heat and analgesics for arthritis. His leg became clumsy and his knee "buckled" on several occasions. Then his right foot began to "drag" and became swollen.

Examination showed that he walked slowly with short steps. There was increased tone in both legs. His only weakness was a minimal loss of power in both hamstring muscles. Deep tendon reflexes were all brisk except for the ankle jerks which were absent. Superficial abdominal reflexes were present. Vibratory sense was slightly reduced at the ankles. Position sense was impaired only in the toes. Romberg test was positive. Remaining sensory and neurologic examinations were normal. Atrophic papilla were seen along the lateral border of the tongue. The thyroid gland was mildly enlarged. Patches of vitiligo on his hands, forearms, chest, and face had appeared and gradually progressed over the previous 23 years.

Laboratory data included a hematocrit of 38.6%. Peripheral smear contained macrocytes and multisegmented, large polymorphonuclear cells. Mean corpuscular volume (MCV) was 111, mean corpuscular hemoglobin (MCH) was 35.6, and mean corpuscular hemoglobin concentration (MCHC) was 32. Two grams of valine taken orally caused methylmalonic acid to appear in the urine and indicated a deficiency of vitamin B_{12} acting as a coenzyme for conversion of methylmalonic acid to succinic acid.[1] Aspiration of

sternal bone marrow revealed a megaloblastic erythropoiesis. Gastric analysis showed a 28 cc/hour basal acid secretion rate and an alkaline pH in response to histamine stimulation. Serum vitamin B_{12} assay was 22 micrograms (<85 micrograms/ml occurs in pernicious anemia). Schilling test showed less than 5% excretion of radioactive tagged vitamin B_{12}.

The patient was treated daily with 100 μg vitamin B_{12} intramuscularly. Within one week his gait was normal. All his neurologic signs disappeared except for his hyperactive deep tendon reflexes. In retrospect he acknowledged slow thinking and irritability for the previous 6 months.

Discussion. The typical presentation of subacute combined degeneration of the spinal cord or vitamin B_{12} myelopathy is with weakness of the legs and paresthesias of all four limbs.[2] These neurologic findings are consistent with pathologic changes in dorsal and lateral columns. In these zones diffuse, asymmetric, spongy foci of degeneration occur in both axons and myelin sheaths. Impairment of vibratory sense often extends to the trunk while position sense is usually lost in the joints of the toes and feet. Pyramidal signs of weakness and spasticity are initially limited to the legs along with extensor plantar signs. Depressed deep tendon reflexes in the ankle and knee are thought to reflect involvement of peripheral nerves.

This patient presented with a subtle form of asymmetric spasticity. Attention to a slight reduction in the hematocrit prompted discovery of a macrocytic anemia and vitamin B_{12} deficiency. Proper therapy was then initiated. Myelography was avoided, and prompt improvement in the neurologic condition resulted. Since 15% to 25% of patients with vitamin B_{12} deficiency present with neurologic difficulties, subacute combined degeneration of the spinal cord must always be kept in mind for patients with signs and symptoms below the foramen magnum. The 23-year progression of vitiligo is of interest in respect to the autoimmune hypothesis of pernicious anemia.[3] Immunologic destruction of melanocytes is a proposed cause of vitiligo, and presumably a similar mechanism causes atrophy of the intrinsic factor secreting mucosa of the stomach.

There are other metabolic disorders which affect the spinal cord. Folic acid deficiency mimicking the neurologic and hematologic presentation of subacute combined degeneration has been reported.[4] Lateral column degeneration has followed portal-caval shunts or spontaneous portal systemic anastomoses in patients with severe cirrhosis of the liver.[5] In these patients spastic paraplegia is irreversible and accompanied by an encephalopathy. Pathology in this condition shows demyelination of the lateral columns which, unlike subacute combined degeneration, does not affect axons. The metabolic mechanism of the demyelination is not known.

Vascular Disorders.

Patient 2. A 10-year-old girl entered the Pediatric Clinic with a shuffling gait two months after repair of her aortic coarctation. Surgery went well, but atelectasis complicated the immediate postoperative period. Her chest x-ray cleared after one week and she was discharged. Upon arriving home her mother observed that her daughter walked slowly and was unable to climb stairs. Examination showed mild bilateral weakness in the iliopsoas and quadriceps muscles. Knee jerks and ankle jerks were hyperactive, and plantar responses were bilaterally extensor. There was a mild diminution to pain and temperature sensation in each leg, but position sense and vibratory discrimination were normal.

Discussion. The neurologic signs in this patient showed bilateral dysfunction of the pyramidal and spinothalamic tracts which, with normal dorsal column function, is indicative of the anterior spinal artery syndrome.[6] Intramedullary blood supply to spinal cord segments is similar throughout the length of the spinal cord. The radicular artery on the spinal nerve root gives a ventral branch to the single anterior spinal artery and a dorsal branch to paired posterior spinal arteries. A ring of superficially anastomosing vessels connects these two systems and supplies the underlying spinal cord surface. Reduction in blood flow to terminal sulcal and penetrating branches of the anterior spinal artery results in ischemic infarction of the clinically important pyramidal and spinothalamic tracts of the lateral columns. Occlusion of the anterior spinal artery results in infarction of the anterior columns, ventral horn, and central grey matter so that touch insensibility, segmental muscular atrophy, and incontinence of bowel and bladder occur. The dorsal columns and dorsal horns remain functional because of the separate blood supply of the paired and interconnecting posterior spinal arteries.

Current clinical and anatomic evidence indicate that circulation through the anterior spinal artery is

either interrupted or inadequate to allow for blood flow throughout the length of the spinal cord.[7] The nature and variability of radicular arteries further divide the spinal cord into vertical zones of blood supply.[8] Although each nerve trunk has a radicular artery, only seven or eight radicular arteries have sufficient size and flow to contribute to the anterior spinal artery circulation. The upper cord and its highest segments are supplied by the intracranial and transverse branches of the vertebral artery and in lower cervical and upper thoracic segments by deep cervical and ascending cervical branches of the subclavian artery. The lower cord is often supplied by a single large radicular artery between segments T-10 and L-2; the artery of Adamkiewicz. The middle spinal cord between segments T-4 and T-8 is supplied by a single radicular artery usually found at T-7. Thus the middle thoracic segment has little compensatory flow and is especially vulnerable to ischemic infarction.

Brewer reviewed 66 cases of bilateral lower limb weakness following repair of aortic coarctations.[9] Duration of aortic clamping and the number of intercostal arteries sacrificed were not related to the occurrence of spinal cord infarction. He proposes that anatomic variations in the anterior spinal artery and poor preoperative collateral circulation around the coarctation were the most important factors contributing to postoperative paraparesis. He points out that poor collateral flow to the cord contributes to the occasional spontaneous development of paraparesis in patients with aortic coarctation. Sacrifice of a low-lying artery of Adamkiewicz or of a second radicular artery in the lumbar region causes paraparesis during repair of abdominal aneurysms.[10] Occlusion of these arteries is proposed as a mechanism for paraparesis occurring with dissecting aortic aneurysms and saddle embolization of the lower aorta.[11]

Several other myelopathies occur due to alterations in spinal cord blood supply. Transient ischemic attacks and spinal cord infarction from arteriosclerotic radicular arteries are documented but are rare.[12] Spinal arteriovenous malformations occur. Their presentations are acute, intermittent, or progressive. Segmental cutaneous hemangiomas have been reported by Doppmann and associates to occur with underlying spinal arteriovenous malformation.[13] Recognition of these cutaneous angiomas is facilitated by a Valsalva maneuver and assists the angiographer in pinpointing the level of the major arterial blood supply to the malformation. Foix and Alajouanine described thrombosis in dilated, tortuous spinal arteries with thick walls as a unique cause of subacute necrotizing myelopathy. Clinically this disorder is characterized by a stuttering progression with leakage of red blood cells and protein into the spinal fluid. Later Alajouanine and others recognized these abnormal vessels as components of an arteriovenous malformation.[14] Thickened vascular walls found by pathologic examination differentiate this condition from the more rarely reported thrombosis of spinal veins.[15]

Claudication or exercise-induced limping occasionally results from spinal cord ischemia.[16] Paresthesias in lumbosacral dermatomes and cramping pain in the legs develop shortly after ambulation is begun or a posture involving extension of the lumbar spine is assumed. While at rest, the neurologic examination including the straight leg raising test is normal. Sensory loss and weakness develop with exercise rather than reduction of peripheral pulses. Myelography is required to confirm a congenitally narrowed lumbar spinal canal, further narrowed by minor disc protrusions that compromise blood supply and compress lumbosacral nerve roots. Epstein found a wide anatomic variation in this canal and concluded that sagittal narrowing of less than 13 mm often is associated with clinical evidence of root compression.[17]

Tumorous Disorders.

Patient 3. A 51-year-old woman complained of stiffness in her legs and burning in the soles of her feet six months before hospitalization. While toweling herself she discovered that she was numb in the lower back. Twice upon seeking consultation she was advised to lose weight. She came to the hospital when she began to drag her right leg while ascending stairs. Examination revealed mild weakness and spasticity in both legs with hyperactive deep tendon reflexes and extensor plantar responses. There was a positive Beevor's sign and a sensory level for pin prick to the umbilicus.

Discussion. This patient represents the frequency of spinal meningiomas occurring in middle-aged women. The thoracic area is by far the most common site for spinal meningiomas. Slow growth of these intradural tumors frequently contributes to their delay in diagnosis; hence insidious onset is typical. In this stage localized pain, determined by percussion of the spinous process of each vertebra, and early symptoms

of compression are vaguely defined and unaccompanied by frank neurologic signs. X-ray changes in thoracic vertebrae are often absent in contrast to the hyperostosis or bony erosion of the skull seen with intracranial meningiomas.

Mention should be made of the sign of segmental weakness of abdominal muscles which occurred in this patient. Beevor described migration of the umbilicus toward the functioning recti muscles of the abdomen and away from those weakened by interruption of their T-8 to T-12 thoracic root innervation. Usually upward movement of the umbilicus is recognized and occurs with lesions across the T-10 segment, but lateral migration of the umbilicus occurring with unilateral lower abdominal nerve paralysis is infrequently observed. Segmental loss of superficial abdominal reflexes ordinarily accompanies Beevor's sign.

Patient 4. A 32-year-old security agent had occasional pain in his right leg and foot for 14 years. He walked with a limp in his right leg. His condition was attributed to a congenital defect in association with spina bifida of his lumbar spine. For four years he had one to three band-like headaches per day lasting 25 to 30 minutes with occasional obscurations of vision lasting several seconds. He came to the hospital when he awoke momentarily blind for the second morning in three days. Examination showed papilledema, mild weakness in the flexors and extensors of the right foot, hypoactive knee jerks, and absent ankle jerks. Pain and temperature were slightly diminished in the left leg. Position and vibratory discrimination were bilaterally impaired in the toes.

Discussion. It is not surprising that the combination of papilledema and unilateral leg weakness led first to intracranial neuroradiologic studies in this patient with a lumbosacral ependymoma. The occurrence of papilledema with lower spinal tumors is recognized, but its mechanism is poorly understood.[18] Often cerebrospinal fluid (CSF) protein is elevated; it was 320 mg% in this patient. Curiously, nearly half of these patients have ependymomas. Despite the papilledema, ipsilateral weakness and crossed sensory loss to pain and temperature point directly to a spinal cord lesion. This is the Brown-Séquard syndrome and was undoubtedly demonstrated in the laboratory by Brown-Séquard himself when he taught at the Medical College of Virginia in 1855.

Since it was a source of confusion in this patient, a point should be made regarding the finding of congenital defects about the lower spine. In the presence of a neurologic deficit these anomalies should undergo a thorough evaluation. For example, spina bifida is sometimes associated with myelomeningoceles and intraspinal lipomas. Dermal sinuses are known to have fistulous connections to the subarachnoid space and serve as the source of recurrent meningitis.[19] A patch of hair, cutaneous nerves, or skin dimple in the lumbosacral region suggest the possibility of an underlying calcified septum and duplication of the cord which occurs in diastematomyelia.[20,21] All of these conditions have the potential for neurologic progression and are, once recognized, neurosurgically remediable.

Patient 5. For three years, a 48-year-old housewife complained of aching pain in the mid-thoracic region as she rose from a sitting or lying position. Occasionally pain became sharp and radiated down both thighs. A tight feeling developed in her right lower ribs as she stood up. For several years she received cortisone injections for "spinal arthritis." Six months prior to hospitalization, she often awoke with uncomfortable, tight, squeezing sensations in her legs. She began to have frequent falls. Examination showed a mild spastic paraparesis with a sensory level to segment T-8. Position and vibratory sense in the toes were absent. Myelography showed a ventral extradural block. At exploration, protrusion of a thoracic intervertebral disc was exposed.

Discussion. Changes in posture, as illustrated by this patient, affect both the blood supply and conduction capacity of a partially compressed spinal cord and its nerve roots. Increased intraspinal pressure caused by a cough or sneeze similarly produces intermittent symptoms. Thoracic pain occurring with a change in posture or coughing is a frequent symptom of patients with extrusion of a thoracic disc. In this disease unpleasant and segmental paresthesias were frequently found among 14 patients reported by Carson and associates.[22] He stated that recognition of this condition draws attention to the typical symptom complex, care in the myleographic diagnosis, and a wide lateral approach to the ventral disc at laminectomy.

Infectious Disorders.

Patient 6. A 25-year-old escapee from a Virginia prison developed low back pain which steadily intensified and spread down the back of each leg over five days. He entered a Washington hospital with bowel and bladder incontinence and limited movement of both legs. Due to the intense hyperesthetic pain, motor power and plantar responses could not be determined. Deep tendon reflexes were hyperac-

tive. Normal CSF pressures were found, and the CSF contained three red blood cells, protein of 150 mg%, and normal sugar. Myelography was normal. Over ten days he became paraplegic and anesthetic to the T-8 level. Deep tendon reflexes were now absent.

Discussion. Clinically this patient presents with the picture of acute transverse myelitis. Local or radicular pain often occur early, and loss of bowel and bladder function is common. Prognosis in acute transverse myelitis is frequently not as grave as the paraplegic state suggests. More than half the patients walk again and, if this is their first neurologic illness, they infrequently develop additional signs of multiple sclerosis.[23] Often a viral or vascular etiology is postulated, but at the turn of the century, in young men, syphilitic myelitis was the most common etiology. Syphilitic infection is considered the cause of this patient's myelitis because the pathology specimen obtained five years later shows a thickened arachnoid that is characteristic of syphilitic meningomyelitis. This thickening not only surrounded the entire cord in the lumbar region, but also extended along its length into the cervical region. Serum serology was positive, and he received more than adequate treatment for central nervous system (CNS) lues due to frequent urinary tract infections. Unfortunately, no CSF examination for syphilis is on record. This problem underlines the continued need to consider neurosyphilis in every instance of CNS disease and to obtain, as appropriate, both serum and CSF serology.

Patient 7. A 52-year-old diabetic was brought to the hospital in a delirious state. He complained of low back pain. There was costovertebral and mild, diffuse low back tenderness. He had no neurologic deficit. Temperature was 101.6 F. The urine contained white blood cells, bacteria, and acetone. Blood sugar was 380 mg% and electrolytes were normal. No acetone was found in the blood. He was treated for a urinary tract infection and dehydration. An attempt at lumbar puncture was aborted when it yielded only several drops of pus. A perinephric abscess was diagnosed and "drained." Three days later the patient became paraplegic.

It is unfortunate that the diagnosis of an epidural abscess was not made.

Discussion. Baker states that the typical course of epidural abscess is the progression from spinal ache to radicular pain, to weakness and then paraplegia.[24] Frequently these patients are delirious which obscures the clarity of this progression and probably explains the absence of the complaint of radicular pain in this patient. The combination of back pain, local tenderness, and fever should raise this possible diagnosis in every practitioner's mind since evolution to paraplegia may be quite rapid. Most patients initially have some neurologic deficit, but as in this patient, not all do. In some instances a site of infection such as a furuncle, dental abscess, or vertebral osteomyelitis is obviously apparent. With early surgical intervention neurologic recovery is expected and sometimes is complete.

Patient 8. A 48-year-old foundry worker had night sweats and cough with purulent sputum for three days. He was given tetracycline. Two days later he complained of diffuse back pain. The next day he became confused and was admitted to the hospital in a delirious state shouting and singing. He was afebrile, his neck was stiff, and ankle jerks were absent. There were no other abnormal neurologic findings. A lumbar puncture obtained yellowish fluid which was under normal pressure. CSF contained 480 white cells of which 90% were monocytes and 10% were polymorphonuclear cells. CSF sugar was 60 mg% and CSF protein was 4428 mg%. The next day the patient became febrile and paraplegic. Another lumbar puncture showed a similar spinal fluid formula. The patient died the following day.

Discussion. CSF protein that exceeds 0.5 gram is referred to as Froin's syndrome. This condition usually occurs with either complete spinal cord block or meningitis of a syphilitic or bacterial origin. If the spinal fluid has a high protein content and is allowed to sit, a pedicle of coagulated protein forms which should be stained for the tubercle bacillus. This patient had a rapidly progressive form of tuberculous meningitis for which he was treated on the basis of the elevated protein in the CSF. Wadia and Dastur, from their wide experience with tuberculosis meningitis in India, suggest that such a fulminant course results from a highly virulent organism or an unusual hypersensitive response to the tubercle bacillus.[25,26]

Disorders Due to Physical Agents.

Patient 9. An 18-year-old primigravida was in good health during her pregnancy. She entered an obstetric ward late one evening and eight hours later gave birth with epidural and brief inhalation anesthesia. Repeated catheterization was needed for relief of bladder distension. On the second post partum day she found it difficult to walk or rise up from a sitting position. This difficulty was ostensibly attributed to perineal pain. She was discharged and carried out of the hospital on the third post partum day. Upon her

return to clinic six weeks after delivery she reported her difficulty walking. Examination showed mild weakness in thigh extensors and leg flexors. There was a mild diminution of sensation in the saddle region about the buttocks and posterior thighs. Ankle jerks and knee jerks were intact, but deep tendon responses of the hamstring muscles were diminished. Plantar signs were flexor. The rectal sphincter was hypotonic. There was no complaint of low back tenderness and the straight leg raising test was normal. She reported gradual improvement in her disability, and six months later there was nearly complete resolution of her neurologic deficits.

Discussion. The neurologic findings in this patient are indicative of the cauda equina syndrome. This structure consists of low lumbar and conus medullaris nerve roots. The conus is composed of sacral segments of the terminal spinal cord. Literally the cauda equina nerve roots grossly resemble a horse's tail as they drape off the conus medullaris. For patients with low back pain, examination of the neural function of these sacral roots should be emphasized. Inquiry into bowel, bladder, and sexual function should be made, and rectal sphincter tone assessed since these functions are subserved by S-2 to S-5 nerve roots. Muscle testing of gluteus maximus and hamstring muscles, best obtained with the patient in the prone position, is too often omitted once anterior leg muscles such as the iliopsoas, quadriceps femoris, and flexors of the foot are found to be strong. In patients complaining of low back pain, sensory testing too often does not include dorsal sacral dermatomes about the buttocks and posterior thighs, the so-called saddle region. In the cauda equina syndrome a normal knee and ankle reflex is misleading unless hamstring stretch reflexes of the lateral biceps femoris and medial semitendinosus and semimembranosus muscles are ascertained.

In the patient reported here a precise diagnosis was not determined because the successful clinical remission experienced by the patient negated need for further diagnostic evaluation. The history suggests that a caudal root neuropathy occurred as a consequence of an anesthetic accident following epidural or inadvertent intraspinal anesthesia. The mechanism is reportedly associated with a detergent or other contaminant of the anesthetic.[27] An epidural hematoma is a less likely explanation in the absence of back pain, anticoagulant treatment, or coagulopathy.

Patient 10. A 69-year-old heavy drinker was admitted to a medical ward following a generalized seizure. Examination showed flat buttocks and thin posterior thigh muscles. The overlying area was anesthetic. Hamstring deep tendon reflexes were absent. Anal sphincter tone was flaccid. Straight leg raising was unremarkable. As his sensorium cleared he stated that 24 years previously fecal incontinenece began. Loss of sacral sensation appeared over several more years. There had been little change in his condition in the ensuing 20-year period. He denied low back pain, although review of his medical records showed that 30 years earlier he complained of lower spinal discomfort for several years. X-rays revealed an increased density of the medial aspect of the iliac bones.

Discussion. Further review of the patient's medical records disclosed the probable cause of his neurologic deficit. During the two-year period of lower spinal pain, he received two myelograms which employed a colloidal solution of thorium dioxide or Thorotrast®. In certain centers this agent was widely used between 1938 and 1945. Its use was discontinued because of recognition that its low-penetrating alpha ray activity with a half life of 14 billion years has a deleterious effect on surrounding tissue and subjects the patient to a risk of subsequent neoplasia. This patient should be added to the reports of Maltby[28] and Tucker et al[29] of patients with a cauda equina syndrome beginning five to fifteen years after their exposure to thorium dioxide. The delayed effect of radiation upon the nervous system which occurred in these cases resembles a similar delayed onset in cases of radiation myelopathy.[30,31] This complication of irradiation therapy is infrequent. Its onset occurs about one year after irradiation is given to the cord or adjacent surrounding areas. Clinically the neurologic deficits are benign and remissive, or chronic and relentlessly progressive. Its etiology is related to the rapidity and total dose of radiation received as well as to individual variations in vascular supply to the portion of the cord which is exposed during therapy. Its diagnosis is problematic since consideration usually is given to a metastatic tumor of the cord.

Patient 11. A 34-year-old drug pusher was shot and fell to the ground. In the emergency room he moved all extremities in response to pain. A bullet entry wound was observed in the left lower abdominal quadrant, and its exit was found in the right posterior flank. At laparotomy several bleeding vessels were ligated. The pancreas was reported to be contused. The patient awoke in an intensive care

ward, complaining bitterly of pain in his legs. He cursed those attempting to examine the lower extremities and demanded that a tent of sheets be constructed over his legs so that nothing could possibly touch them. His stools soiled the bed sheets, and he was incontinent of urine. X-rays of the spine were normal. Lumbar puncture showed normal pressures, and his spinal fluid contained 75 red blood cells, 82 mg% sugar, and 70 mg% protein.

After one month his severe pain diminished. Bowel and bladder function returned. Examination showed moderate bilateral weakness of the glutei and hamstring muscles. He was unable to stand on the toes of his right foot. Hamstring and ankle jerks were absent. There was anesthesia of the buttock and posterior thigh. Mild tactile stimulation of the posterior legs or feet often provoked dysesthetic pain. The rectal sphincter was patulous. Over a two-year period his dysesthetic pain entirely disappeared.

Discussion. This patient presented a difficult situation in the intensive care unit since he would not allow a complete examination, and the true nature of his complaints were not understood. Further difficulty ensued with demands for narcotics to temper either his drug withdrawal or his poorly appreciated causalgic pains. Tomography of the lower spine was in this instance helpful in reaching a plausible explanation of his signs and symptoms. Tomograms of the lumbar spine showed a bullet hole through L-2, L-3 lumbar disc space. Evidently either the concussive shock of the bullet or extruded disc material contused the roots of his cauda equina. Use of the polytomogram is advised in unexplained cases of nerve root injury which occur in the region of the nerve's osseous compartments.

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