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Diagnosis of Transverse Myelitis:

An Evaluation of the factors leading to Spinal Dysfunction
Including a Review of one patients experience

By
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Abstract

In October, 1965 a 20 year old female presented to hospital with low back pain which eventually ends in paralysis. On review of available medical records over time and personnel interviews with the patient it appears she was given a questionable diagnosis of transverse myelitis (TM). With advancements in medical science over the last 35 years and use of MRI in diagnosis of TM in the last 6 years, would further testing of this now 56-year-old patient be warranted to confirm diagnosis or to at least gain information from her situation. The following will discuss the causes, signs, symptoms and diagnosis of TM. We will then review available information from hospital records and a personal account of the events from the above patient in order to conclude if in fact the diagnosis of TM was correct or if further testing is warranted.

The spinal cord contains all the neural machinery that connects your body with the brain. It contains several types of cells, including groups of neurons, that electrically conduct impulses that convey information about position, sensation, bodily functions, and muscle strength. It is also a conduit of pathways ("tracts") which either ascend to the brain from the extremities, or descend from the brain to the extremities. For example, the dorsal columns refer to tracts of neuron fibers (a bundle of electrical wires) that convey information to the brain about where the legs and arms are in space. They also convey information about what is touching the legs and arms. Other neuron tracts convey information about pain (the spinothalamic tract) and allow you to sense that you've placed your hand on a hot burner (and should remove it quickly!). Still other tracts begin in the brain and carry impulses to various muscle groups. These fibers allow you to move your muscles in response to a command or even involuntarily.

Transverse myelitis is an acute neurologic condition that reflects focal inflammation at one level of the spinal cord. It is characterized by symptoms and signs of neurologic dysfunction in motor and sensory tracts on both sides of the spinal cord. Often this is associated with a clearly defined area of altered sensation on both sides of the body, weakness of both legs and sometimes the arms, and urinary

or bowel dysfunction. The “transverse” reflects dysfunction at a particular level across the spinal cord, manifest as altered function below this level, and normal function above it. Objective diagnostic criteria have been proposed as follows (Berman et al., 1981; Ropper and Poskanzer, 1978; Jeffery et al., 1993):

- 1) acute or subacutely developing motor, sensory and sphincter disturbance
- 2) spinal segmental level of sensory disturbance with a well defined upper limit
- 3) no evidence of spinal cord compression
- 4) absence of other known neurologic disease such as syphilis, previously diagnosed multiple sclerosis, malignant neoplasm, spinal cord arteriovenous malformation, sarcoidosis and HIV infection

Clinical Symptoms

TM symptoms develop rapidly over several hours to several weeks. Approximately 45% of patients worsen maximally within 24 hours (Ibid.). The spinal cord carries motor nerve fibers to the limbs and trunk and sensory fibers from the body back to the brain. Inflammation within the spinal cord interrupts these pathways and causes the common presenting symptoms of TM which include limb weakness, sensory disturbance, bowel and bladder dysfunction, back pain and radicular pain (pain in the distribution of a single spinal nerve).

Almost all patients will develop leg weakness of varying degrees of severity. The arms are involved in a minority of cases and this is dependent upon the level of spinal cord involvement. Sensation is diminished below the level of spinal cord involvement in the majority of patients. Some experience tingling or numbness in the legs. Pain (ascertained as appreciation of pinprick by the neurologist) and temperature sensation are diminished in the majority of patients. Appreciation of vibration (as caused by a tuning fork) and joint position sense may also be decreased or spared. Bladder and bowel sphincter

controls are disturbed in the majority of patients. Many patients with TM report a tight banding or girdle-like sensation around the trunk and that area may be very sensitive to touch.

Recovery may be absent, partial or complete and generally begins within 1 to 3 months. Significant recovery is unlikely, if no improvement occurs by 3 months (Feldman, et. al., 1981). Most patients with TM show good to fair recovery. TM is generally a monophasic illness (one-time occurrence); however, a small percentage of patients may suffer a recurrence, especially if there is a predisposing underlying illness.

Causes of Transverse Myelopathy and Myelitis

Transverse myelitis may occur in isolation or in the setting of another illness. When it occurs without apparent underlying cause, it is referred to as idiopathic. Idiopathic transverse myelitis is assumed to be a result of abnormal activation of the immune system against the spinal cord. A list of illnesses associated with TM includes: (not all of which will be discussed)

- Parainfectious (occurring at the time of and in association with an acute infection or an episode of infection).
- Viral: herpes simplex, herpes zoster, cytomegalovirus, Epstein-Barr virus, enteroviruses (poliomyelitis, Coxsackie virus, echovirus), human T-cell, leukemia virus, human immunodeficiency virus, influenza, rabies
- Bacterial: *Mycoplasma pneumoniae*, Lyme borreliosis, syphilis, tuberculosis
- Postvaccinal (rabies, cowpox)
- Systemic autoimmune disease: Systemic lupus erythematosus, Sjögren's syndrome, Sarcoidosis
- Multiple Sclerosis
- Paraneoplastic syndrome
- Vascular
- Thrombosis of spinal arteries
- Vasculitis secondary to heroin abuse

- Spinal arterio-venous malformation

The cause of idiopathic transverse myelitis is unknown, but most evidence supports an autoimmune process. This means that the patient's own immune system is abnormally stimulated to attack the spinal cord and cause inflammation and tissue damage. Examples of autoimmune diseases that are more common include rheumatoid arthritis, in which the immune system attacks the joints, and multiple sclerosis, in which myelin, the insulating material for nerve cells in the brain, is the target of autoimmune attack.

TM often develops in the setting of viral and bacterial infections, especially those which may be associated with a rash (e.g., rubeola, varicella, variola, rubella, influenza, and mumps). Approximately one third of patients with TM report a febrile illness (flu-like illness with fever) in close temporal relationship to the onset of neurologic symptoms. In some cases, there is evidence that there is a direct invasion and injury to the cord by the infectious agent itself (especially poliomyelitis, herpes zoster, and AIDS). A bacterial abscess can also develop around the spinal cord and injure the cord through compression, bacterial invasion and inflammation.

However, experts believe that in many cases infection causes a derangement of the immune system that leads to an indirect autoimmune attack on the spinal cord, rather than a direct attack by the organism. One theory to explain this abnormal activation of the immune system toward human tissue is termed "molecular mimicry." This theory postulates that an infectious agent may share a molecule, which resembles or "mimics" a molecule in the spinal cord. When the body mounts an immune response to the invading virus or bacterium, it also responds to the spinal cord molecule with which it shares structural characteristics. This leads to inflammation and injury within the spinal cord.

Vaccination is well known to carry a risk of the development of acute disseminated encephalomyelitis (ADEM) which is an acute inflammation of the brain and spinal cord. This was particularly common with the older antirabies vaccine that was grown in animal spinal cord cultures; the

use of the newer antirabies vaccine grown in human tissue culture has almost eradicated this complication. This is also thought to occur as an immune system response.

Transverse myelitis may be a relatively uncommon manifestation of several autoimmune diseases including systemic lupus erythematosus (SLE), Sjogren's syndrome, and sarcoidosis. SLE is an autoimmune disease of unknown cause that affects multiple organs and tissues in the body. Features of this illness include arthralgias (joint pain) and arthritis (joint inflammation), rashes, kidney inflammation, low blood counts (including white and red blood cells, platelets), oral ulcers and the presence of abnormal autoantibodies (antibodies which are directed against the person's own tissues) in the blood. The fully developed syndrome of SLE is easy to recognize; however, this illness may begin with just one or two signs and is then more difficult to diagnose.

Sjogren's disease is another autoimmune disease characterized by invasion and infiltration of the tear and salivary glands by (lymphocytes) white blood cells with resultant decreased production of these fluids. Patients complain of dry mouth and dry eyes. Several tests can support this diagnosis: the presence of a SS-A antibody in the blood, ophthalmologic tests that confirm decreased tear production and the demonstration of lymphocytic infiltration in biopsy specimens of the small salivary glands (a minimally invasive procedure). Neurologic manifestations are unusual in Sjogren's syndrome, but TM can occur.

Sarcoidosis is a multisystem inflammatory disorder of unknown cause manifested by enlarged lymph nodes, lung inflammation, various skin lesions, liver and other organ involvement. In the nervous system, various nerves, as well as the spinal cord, may be involved. Diagnosis is generally confirmed by biopsy demonstrating features of inflammation typical of sarcoidosis.

Acute transverse myelitis is sometimes is a feature of relapsing-remitting multiple sclerosis. Chronic-progressive MS develops as a spinal cord stroke. Multiple sclerosis is an autoimmune disorder of unknown etiology where an immune-mediated attack against the central nervous system causes damage to the nerves and supporting cells within the brain and spinal cord. Most studies have shown that

patients who develop TM usually do not go on to develop MS. In these studies, the range of patients who presented with TM and subsequently developed MS ranges from 0-21% (Jeffery et al., 1993; Ropper and Poskanzer, 1978; Misra et al., 1996; al Deeb et al., 1997). However, other studies have shown a much higher rate of development of MS following acute TM. Ford et al., studied 15 patients with acute partial transverse myelopathy of unknown etiology and found that 12 of them subsequently developed multiple sclerosis (Ford et al., 1992). They also found that TM associated with infections can be distinguished from that associated with MS based on clinical grounds, imaging studies and immunologic evaluations. It should be emphasized that this study excluded those patients with complete myelopathy; subsequent MS is rare in those patients who present with complete TM in the acute phase. Rather, TM as an initial presentation of MS tends to be partial (rather than complete loss of function below the level of injury) and asymmetrical (involving one side of the body more than the other). However, many patients with TM should be screened for MS since treatment strategies may be very different for those patients with likely MS compared to those who do not. This evaluation should include a brain MRI to determine if demyelinating lesions are present elsewhere in the central nervous system and a spinal tap to determine if antibodies are being synthesized within the CNS (oligoclonal bands). It may also include electrical studies called evoked potentials (somatosensory, brainstem and visual evoked potentials)

TM is sometimes a manifestation of systemic lupus erythematosus (SLE, often-called "lupus"). SLE is a multi-system autoimmune disorder that sometimes involves the nervous system. It is most famous for causing a facial rash, inflammation of the linings of the heart and lungs, and kidney disease. However, up to 50 % of patients with SLE develop central nervous system complications. Approximately 1-3% of patients with SLE will develop TM (Wallace and Metzger, 1993), but a higher proportion of acute TM patients will ultimately develop serologic evidence of an autoimmune disorder such as SLE. This is always an important diagnostic consideration because several studies have shown that intravenous steroids and other immunosuppressant regimens seem to improve outcome in this group of patients if given during the acute phase of TM (Goodin, 1991; Miller, 1980; Sebire et al., 1997). However, a recent study has shown that some of the classical features of SLE are absent in those patients with TM, making the diagnosis more difficult (Mok et al., 1998). Only one of ten patients had active renal disease at the time of diagnosis of TM. A blood test highly specific for SLE (dsDNA) was positive

in only 4/10 patients (significantly lower than other patients with SLE), and disease activity in other organ systems was rarely seen at the time of TM diagnosis. To make the diagnosis even more difficult, 44% of spine MRI studies were normal in patients with SLE-associated TM, and 37% of spinal fluid studies were normal. An abnormal circulating antibody has been found in some SLE/TM patients as well as patients who do not meet criteria for SLE. These antibodies confer an increased susceptibility to form blood clots within blood vessels. Formation of a blood clot within an artery supplying the spinal cord may deprive it of oxygen and lead to death of cells in that area causing TM. Prognosis of TM associated with SLE has in the past been considered to be poor, but that may no longer be accurate. In contrast to previous reports (Penn and Rowan, 1968), recent data reveal that with treatment in the acute phase, 70% of patients have a good to excellent recovery (Mok et al., 1998; Inslicht et al., 1998). Some patients have some features of lupus, but do not meet the criteria for this disease. These patients may have another autoimmune process related to lupus.

As indicated above, TM often occurs in the setting of a recent infection. In some cases, TM may be a result of direct infection of the neurons and supporting cells within the spinal cord. More commonly, however, the TM develops after the infectious agent has been cleared by the immune system. In these cases, it is thought that the immune response generated to fight the infection "cross-reacts" with cells within the spinal cord. Thus, it is the patient's own immune system which causes TM. Why this occurs in some patients and not others is not known. Thirty to 60% of TM patients report symptoms of a preceding infection, usually involving the upper respiratory tract, prior to the onset of the TM (Paine and Byers, 1953; Altrocchi, 1963; Lipton and Teasdall, 1973; Ropper and Poskanzer, 1978; Berman et al., 1981). These cases are called 'parainfectious' TM (TM occurring around the time of an infection). In 20-40% of TM cases, the preceding or concurrent infection is a viral illness, including pathogens such as herpes viruses, (herpes simplex 1 and 2; Epstein Barr virus; Cytomegalovirus; varicella-zoster; human herpes virus 6 and 8), measles, rubella, infectious mononucleosis, HIV, HTLV-1, influenza, and various enteroviruses (for excellent table of viruses known to cause TM and references, see Tyler et al., 1986). Several of the hepatitis viruses including hepatitis A, B and C have been shown to be associated with TM. Evaluation of the spinal fluid and blood of patients in both the acute and convalescent phases of TM is the

best way to diagnosis these viral illnesses. Nevertheless, even without samples from the acute phase, we can evaluate a patient's blood and spinal fluid for markers of previous or active infection with these viruses.

Syphilis remains a rare cause of TM, but related spirochetal diseases- namely Lyme borreliosis, leptospirosis, ehrlichiosis and babesiosis, are increasingly common causes of TM today. Lyme disease is caused by a bacterium with a spiral shape (hence the term spirochete) which is carried by ticks and is widely present throughout North America and Europe. TM is a well-known manifestation of Lyme infection, both at the onset of infection and many years after the initial infection (Reik et al., 1979; Pachner and Steere, 1985; Ackermann et al., 1988; Halperin et al., 1991). Often Lyme-associated TM is accompanied by a painful radiculopathy, multiple painful joints, fatigue, changes in personality or thinking, and cranial nerve deficits. The classic triad of neurologic Lyme disease is meningitis (inflammation of the spinal fluid and meninges surrounding the spine and brain), peripheral radiculoneuritis (inflammation of the nerve roots and peripheral nerves), and cranial nerve palsies (inflammation of the nerves that go to the head and mouth). This triad may occur in only 10-20% of patients (Pachner and Steere, 1985). TM is rarely the only manifestation of Lyme disease. It is often, but not always, preceded by a skin rash which is a red, non-painful, spreading rash at the site of the tick bite (with central clearing, resembling a 'bull's-eye' target). The actual tick is usually never seen! Oligoclonal bands are often present in the CSF leading sometimes to an erroneous diagnosis of MS.

Mycoplasma pneumoniae is a rare, but important cause of TM. It is important because antibiotic treatment may lead to improved outcomes (Abele-Horn et al., 1998). This bacterium is one of the few causes of TM that may be due to direct infection of the central nervous system. *Mycoplasma* is a common bacterium that causes pneumonia as well as other systemic infections.

Mycoplasma rarely infects the central nervous system (about 0.1% of patients (Cassell and Cole, 1981)), but it is such a common infection, that a fair number of TM patients that we see are due to antecedent *Mycoplasma* infection. Of those patients with *Mycoplasma* infection and TM, 21% report no antecedent respiratory symptoms, and so this diagnosis may be missed.

Diagnosis in the acute phase is dependent upon novel molecular technologies (PCR based strategies, utilized at Johns Hopkins Hospital) since older methods of detecting Mycoplasma fail to detect the organism in time for effective treatment.

Many cases of TM (up to 20 % in some studies) are due to vascular disease that causes ischemia or bleeding into the spinal cord. In one sense, this can be viewed as a "stroke" involving the spinal cord. Just like in the brain, blood vessels are required to bring oxygen and nutrient, and remove metabolic products from the cells of the spinal cord. When one of these vessels become blocked-- by a clot which has traveled to an artery supplying the spinal cord, or by progressive narrowing of the vessels by atherosclerosis-- that area of the spinal cord may become ischemic. Patients who get TM as a result of vascular causes tend to be older (mean age 51), or have cardiac disease or have recently undergone a chest or abdominal operation. Other patients with TM develop the disease due to an abnormal collection of blood vessels (AVM or AVF) on the surface of the spinal cord which impair blood return away from the spinal cord (increased venous pressure leading to cord ischemia) or cause bleeding into the spinal cord. This possibility is an important consideration in patients with 'recurrent' or 'ascending' TM. The diagnosis of this etiology may be difficult and is often missed. A spinal angiogram or myelogram may be required to make the diagnosis of TM associated with vascular malformations or ischemia. However, this is an important diagnostic step since several procedures exist to halt the symptoms of TM due to vascular disease.

Multiple sclerosis is an inflammatory autoimmune disease of the central nervous system (brain and spinal cord) which results in demyelination or loss of myelin (the insulating material on nerve fibers) with resultant neurologic dysfunction. A definite diagnosis of MS is not given until a patient has had at least two attacks of demyelination (hence, multiple) at two different sites in the central nervous system. The spinal cord is frequently affected in multiple sclerosis and may be the site of involvement of the first attack of MS. This presents the possibility that patients with acute transverse myelitis could later go on to have a second episode of demyelination and receive a diagnosis of MS.

Just what percentage of patients with a first attack of acute transverse myelitis will go on to develop MS is unclear in the medical literature, ranging from 15 to 80%; however, the majority of studies show a low risk. We do know that patients who have abnormal MRI scans of the brain with lesions like those seen in MS, are much more likely to go on to develop MS than those who have normal brain MRIs at the time of their myelitis (between 60 and 90% for those with abnormal brain scans, less than 20% for those with normal scans in one study). It is also suggested in the medical literature that patients with "complete" transverse myelitis (which means severe leg paralysis and sensory loss) are less likely to develop MS than those who had a partial or less severe case. The literature also suggests that patients, who have abnormal antibodies in their spinal fluid, called oligoclonal bands, are at higher risk to develop MS subsequently.

Myelitis related to cancer (called a paraneoplastic syndrome) is uncommon. There are several reports in the medical literature of a severe myelitis occurring in association with a malignancy. In addition, there are a growing number of reports of cases of myelopathy associated with cancer in which the immune system produces an antibody to fight off the cancer and this cross-reacts with the molecules in the spinal cord neurons. It should be emphasized that this is an unusual cause of myelitis.

Vascular causes are listed because they present with the same problems as transverse myelitis; however this is really a distinct problem primarily due to inadequate blood flow to the spinal cord instead of actual inflammation. The blood vessels to the spinal cord can close up with blood clots or atherosclerosis or burst and bleed; this is essentially a "stroke" of the spinal cord.

Diagnosis

The general history and physical examination are first performed, but often do not give clues about the cause of spinal cord injury. The first concern of the physician who evaluates a patient with complaints and examination suggestive of a spinal cord disorder is to rule out a mass-occupying lesion that might be compressing the spinal cord. Potential lesions which might compress the cord include tumor, herniated disc, stenosis (a narrowed canal for the cord), and abscess. This is important because

early surgery to remove the compression may sometimes reverse neurologic injury to the spinal cord. The easiest test to rule out such a compressive lesion is magnetic resonance imaging of the appropriate levels of the cord. However, if MRI is not available or the images are equivocal, myelography must be performed. A myelogram is a set of X-rays taken after a lumbar puncture has been performed either in the neck or in the low back and a contrast agent (dye) is injected into the sac that surrounds the spinal cord. The patient is then tilted up and down to let the dye flow and outline the spinal cord while the X-rays are taken.

This study involves a case in which a female who will be known for the purpose of this study as L.T. who at the age of 20 lost the ability to walk. The significance of this case is that there was no apparent reason for paralysis. This case will look into the history of L.T., exploring findings at the time of the incident from medical records and pt. Interview through to present day. This project presents as a great challenge as this case is 34 years in duration and a complete compilation of all records was not possible due to the time frame. I feel that there is still a need for this study to be done in spite of time line due in fact that there has been significant advances in technology through out the medical paradigm (MRI,C.T.) and research into spinal dysfunction, which was not available 34 years ago. The field of chiropractic has also grown and given new options to those who never got answers, relief from pain, or any help at all from the medical field. As the cause of paralysis was never diagnosed, I feel that it is important to exhaust all possibilities available in order to explain to the pt. why this problem arose. As it is every doctor's responsibility to give the best care possible to every pt. in that what new insights may not relate to the present pt. it may help another.

This study will look at the available data in an attempt to come up with a possible answer to a 34 year old question, of why did this happen, and to perhaps allow insight at some point which could lead to a reversal of paralysis if not for L.T. for the next pt. who may present with similar circumstances. The scope of this study is limited to medical records not destroyed and L.T.'s recall of events.

The following will be a look into the background of the pt. L.T, as it relates to this study. It will incorporate the findings and procedures of the hospital from the time L.T. 's admittance into the hospital in 1965 and include all available history and then explore the patient's point of view and her recall of the events, which took place.

Physician's clinical record from University Hospital Saskatoon, Saskatchewan Oct. 18 1965 – Admission note:

This 20-year-old x-ray technician presents with a history of back pain beginning last Tuesday Oct. 12 with radiation into anterior portion of her thighs. On Thursday Oct. 14 she noticed her urine output had stopped. She was admitted to local hospital the next day she could not sleep due to the severe pain Oct. 16. She also noted that her gait was unsteady. By Saturday morning her legs were paralyzed to sensation and motor function. She was catheterized Oct. 17. She mentioned having a sore throat over a week prior to this occurrence requiring

Oct 18/65 Consultant's report:

History of sore throat 1 week prior to onset of cord symptoms. Onset of low back pain 6 days ago. Sense backache with radiation to leg and inguinal regions 5 days ago. 4 days ago muscle symptoms developed started to stagger-walked on Thursday-completely paralyzed on Friday (3 days ago). Complete paraplegia with flaccid muscles. All reflexes absent below the umbilicus. Sensation to all modalities are absent below the umbilicus

Myelogram

Might be slight swelling of the area of conous medullaris

Transverse myelitis T10 (?)

Oct. 19

This 20-year-old x-ray tech presented with complaints of low back pain beginning while at work last Tuesday. One week prior to this she was being treated for a sore throat. Her local doctor mentioned she had an infected tonsil. The back pain came on suddenly, and was originally described as a constant dull ache. During the evening of the same day the pain became sharp and radiated anteriorly down the front of both thighs. Her thighs were also noticed to tingle and feet numb. On the next day the pain was present in both knees and was associated with a pins and needles sensation in the knees. /the pain in the back had become sharper in nature and had a stabbing quality to it. The pain lasted until Thursday with the patient getting little sleep. On Friday she complained of cramps in her toes along with pins and needles parasthesis. She was hospitalized Friday. A.m. complaining that her back pain had become more severe.

Notes dating from Oct. 22 – Dec. 1

On 10-22:

This patient has flaccid paralysis of both lower limbs as well as loss of cutaneous sensation. There is no evidence of muscle return in lower limbs. She also has a flexion contracture of her left arm along with shoulder weakness (birth trauma injury) no other notes were made in regard to performed tests. 11-05 pt has developed edema in her left leg causing increased circumferential measurements. There is no further mention of tests performed following notes were in reference to L.T. 's progression with the adjustment to her paralysis and classes teaching her to deal with it (dressing, transferring from chair to bed etc.)

Oct 30/65

Surgery:

Vagotomy and antrectomy with Bilroth 11 anastomosis. Treatment for chronic duodenal ulcer with pyloric obstruction

Nov 13/1965

X-ray report:

C/S

The alignment of the vertebral bodies is straight with maintained disc spaces. The bodies are intact, with normal appearing transverse and spinous processes. The pre-vertebral soft tissues show normal width.

Oct. 19/65

Portable chest:

The cardiovascular silhouette is within normal limits. The lungs are clear.

Oct 18/65

Dorsal spine

The alignment is normal and the vertebral bodies and disc spaces are of normal height. The pedicles, transverse processes and posterior spinous processes are intact.

L/S

The alignment is normal with the height of the vertebral bodies and disc spaces within normal limits. The pedicles, transverse processes and posterior spinous processes are intact. Both sacro-iliac joints appear normal.

Panopaque Myelogram

Six cc. Of panopaque was injected under local anesthesia and the contrast allowed to flow through out the lumbar and dorsal areas. No evidence of block was demonstrated the column of contrast material normal throughout.

Feb 1/66

Final diagnosis

Paraplegia due to transverse myelitis

Complications:

Neurogenic bladder

Thrombosis of leg vein

Urinary infection

TX:

Antiseptic, Anti-biotic and sulfa drugs

p.t. and o.t.

Anticoagulant

Bladder training

DISCHARGED!

The following is from the transcribed history as written by pt. L.T.

1944 Born in Nov, 10-lbs. injury was received to left clavicle. Discrepancy as to how injury was received, possibly from birth trauma or nurse carelessness. Fracture was located quite close to the shoulder and failed to properly heal. L.T. is unable to straiten the arm at the elbow and has no extention at the wrist as well as inability to abduct at the shoulder. States she has downward push at the elbow close to normal strength but all other movements are badly lacking.

1954 Appendices removed.

Possibly before 1954 had history of stomach problems. Could not stand tight waistbands. Had the odd bladder infection, which went untreated.

1954-1963 Stomach pains got progressively worse, until at the age of 18 (1963) L.T. had her first major bleed and it was discovered she had a bleeding ulcer. She was treated and needed cut downs in her ankles to set up blood transfusions. Was given Probanine, which was used for 2 years as well as heavy use of "every antacid on the market". Relates possibly to nerves.

Early 1965, Suffered from throat problems, was constantly clearing throat. Treated with 3 different antibiotics- Tetracycline, Chloramycitin, Erythromycine.

Oct 13, 1965

Working as a lab/x-ray tech went to work and concerned with back pain had x-rays taken which she attempted to read herself. Seeing nothing abnormal and not being trained to x-ray reading diagnosis showed them to a doctor at the hospital who laughed at her and said "you have got lumbago go home and put a hot water bottle on it". Upon returning home she paced the floor in pain and by evening could no longer stand the pain. Her husband went to the hospital and got her two Demerol pills but even that didn't come close to reducing the pain.

Oct 14/65

Upon waking L.T. found she was unable to urinate so she went to the hospital, as she was walking up the stairs she noticed that her knees were buckling some. She was admitted to the hospital and was given a shot of Demerol. Once again it had no effect on the pain, and she continued to pace the floor. After examination by the doctor he told her that she was not nearly as paralyzed as they are making you out to be. L.T. was put on bed rest and by 10 p.m. was in a great deal of pain in the kidney area, She finally convinced a nurse to get permission to catheterize her-her bladder was very full. This relieved the pain in the kidney area.

The pain which had started severely in her back left and moved around and down the outside of her thighs were it was just as severe as it was in the low back. The pain then left her thighs and went into her knees. It then left her knees and went to her lower legs.

Oct. 15/65

When I woke up in the morning I could not move from the waist down and I had no feeling. I was then transferred to Saskatoon University hospital.

The tests and instrumentation used back in 1965 was inadequate to provide a diagnosis on the one medical record at discharge it was stated as a diagnosis of transverse myelitis but upon questioning further found out that it wasn't really a diagnosis but a educated guess or a possible working diagnosis. 34 years later we now have access to MRI which would allow for a more accurate diagnosis but when L.T. approached doctors about this she was told that it was too late and there would be no point in running the test, it would be a waste of money.

Conclusion

From the available information reviewed the diagnosis of transverse myelitis given 35 years ago is a correct working diagnosis although it should state it was of idiopathic cause. Due in fact that the patient was discharged and no further testing done, things such as the " possible swelling at the level of the conous medularis were not investigated further. Present day advancements such as the MRI would allow much more knowledge to be gained through further research of this patient. As doctors, is it not our job to eliminate all possibilities? Just because a patient's case is old does that mean it should be forgotten and tossed aside? Is it too late? Well I feel that if the patient is still suffering from the problem then the chances that the cause for the problem still has to be there. Even if that patient has suffered for many years, with the symptoms of an unknown cause, and they may be to old to ever fully recover there may be a chance that someday because somebody was willing and cared enough to believe in helping that

patient to get better and found out why the problem was caused it could save somebody else from all those years of not knowing why? This study is not necessarily about finding the cure of a 34-year idiopathic spinal dysfunction, one may not be found, but to give up and not even try is even more crippling than the dysfunction. With what is known today versus that which was known in 1965 I think that L.T. deserves a chance at knowing. One other aspect to this study is that it will not just be a medical outlook taken into consideration, but one of chiropractic as well. Just the difference in what we see when looking at an x-ray of the spine could hold the answer.

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