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Fibromyalgia: The Fallacy of Its Diagnosis

Albert C. Cuetter

MD, Department of Neurology, Texas Tech University, Health Sciences Center, El Paso, Texas 79905

Abstract: **This report does not intent to discuss fibromyalgia as an entity, but to show that many patients diagnosed as having fibromyalgia have other identifiable pathologic processes that reasonably explain the pain syndromes attributed to fibromyalgia.1,2,3,4**

This is a community-based study of 53 patients with the diagnosis of fibromyalgia made at general medicine outpatient clinics. Most of these patients had been referred to the Department of Neurology for subsequent evaluation intended to reach a plausible and more rational diagnosis than fibromyalgia. A reasonable diagnosis that would explain the symptoms attributed to fibromyalgia was achieved in 31 patients.

The gist of the argument is that a complete physical examination and some laboratory tools are necessary before branding a patient as having fibromyalgia. Many people with the diagnosis of fibromyalgia have medical conditions that can reasonably explain the symptoms diagnosed as fibromyalgia; that is to say, fibromyalgia is not a primary disease but a symptomatic expression of another underlying process.2,4 Therefore the causes of fibromyalgia syndrome should be investigated appropriately.

Keywords: **Fibromyalgia, alternative diagnoses.**

1. INTRODUCTION

This report does not intent to discuss fibromyalgia as an entity, but to show that many patients diagnosed as having fibromyalgia have other identifiable pathologic processes that reasonably explain the pain syndromes attributed to fibromyalgia.1, 2, 3, 4

When reason and science can not give logical, rational, and reasonable response, human mind resorts to faith, metaphysics, and to highly abstruse theoretical systems. This seems to be the issue fibromyalgia, a clinical symptom with accoutrement of a presumptive true clinical entity conceived by the inability to reach an appropriate diagnosis in many individuals with no relevant objective clinical findings in whom requested laboratory tests not fall short of target for correct etiologic diagnosis. In addition, the pharmaceutical companies have managed the issue, have taken over the matter, and have advertised several medications for the symptomatic treatment of fibromyalgia.

About 2 percent of the American population, or about 5 million people, mostly women have the diagnosis of fibromyalgia. Current available publications indicate that the cause is unknown and there is no known cure. It is believed that it may be related to a combination of chemical changes in brain and spinal cord and genetic factors. The condition is also described as a voltage-gated sodium channel dysfunction and that a neuropathic pain perhaps is linked to autonomic nervous system abnormalities associated to a particular SCN9A sodium channel located in dorsal root ganglia.5 Heightened sensitivity in the central pain processing is suggested to occur in fibromyalgia in that the stimulus required to activate pain processing is much lower in individuals with fibromyalgias that in healthy subjects.6,7,8 Based on the hypothesis of central dysfunction there are studies assessing the effect of high frequency repetitive transcranial magnetic stimulation.9

The other aspect of the problem is that many people with the diagnosis of fibromyalgia have an underlying initiating cause for the pain.2 I wish to review the findings in 53 patients with the diagnosis of fibromyalgia. The importance of making a diagnosis of an underlying condition is that many of them have treatment and resolution.

2. METHODS

This is a community-based study of 53 patients in a period of 10 years, with the diagnosis of fibromyalgia made at general medicine outpatient clinics. Most of these patients had been referred to the Department of Neurology for subsequent evaluation intended to reach a plausible and more rational diagnosis than fibromyalgia.

3. RESULTS

Patient Population:

A reasonable diagnosis that would explain the symptoms attributed to fibromyalgia was achieved in 31 patients. The gist of the argument is that a complete physical examination and some laboratory tools are necessary before branding a patient as having fibromyalgia.

The following list includes those chronic medical conditions that were present in individuals with the clinical diagnosis of fibromyalgia.

1. Neuromyelitis optica (NMO) with myeloradiculopathies:

Three patients referred to Neurology for electrodiagnostic studies gave one to two year-history of constant severe paraspinal pain and pain and spams of upper and lower extremities. Their ages ranged from 24 to 26 years. Two were female, and one was male. Previous rheumatologic work up had been negative and they had the diagnosis of fibromyalgia and were taken gabapentin, or pregabalin, and duloxetine. Examination showed no specific motor deficit and one had hyperactive muscle stretch reflexes but no pathologic reflex. No bladder problem. No gait problem or incoordination. MRI of brain was normal. All had more than one segment intra- axial demyelinating lesions in the cervicothoracic cord. One had oligoclonal banding on cerebrospinal fluid. Two patients were sero positive for NMO-IgG antibodies against the aquaporin 4 antigen. A seronegative patient developed left optic neuritis. All these three patients had neuromyelitis optica. The paraspinal pains and extremity pains are explained by irritating effect of demyelinating plaques at the entry zones of the dorsal roots in the cord.10

2. Multiple sclerosis (MS) plaques at the entry zone of cervical and thoracic sensory nerves:

A 32-year-old woman gave a history of bilateral shoulder and interscapular pain. She was taking medications for the diagnosis of fibromyalgia. She had hyperactive MSR, multiple sensory levels to pinprick up to cervical cord and bilateral Hoffmann signs, but no Babinski sign. MRI of the brain and cervical spine showed a diffuse demyelinating process. Cerebrospinal fluid had 7 oligoclonal banding more than in the serum. Visual evoked responses showed delayed P100 waves. The diagnosis was relapsing remitting multiple sclerosis.

3. Cervical spondylosis with multilevel cervical radiculopathies and myelopathy:

Twelve patients had constant periscapular pains, shoulder pains, neck pain and pain in the arms and forearms. Their ages range from 56 to 82 years. Six of these patients had multilevel spondylotic changes, but they and the other six patients were being treated for fibromyalgias. Cervical spine MRIs, somatosensory evoked responses in two patients, and needle electromyography indicated the correct diagnosis of multiple cervical radiculopathies.

An illustrative case is a 76 year-old male physician with significant constant paraspinal and thoracic pain worse in the morning; and pain in both shoulders and upper extremities. At different times since age 56 he suffered recurrent cervical radiculopathies at different level and in both sides. These radiculopathies were punctuated by traumatic events in which he had suffered axial injuries to cervical areas. At one time a rheumatologist suggested that the pain was due to fibromyalgia and recommended gabapentin. Examination showed atrophy of the interosseous and thenar eminence muscles of the right hand (C8-T1), atrophy of the right anconeus, pronator teres and flexor carpi radialis muscles. The MSRs were hyperactive below the C7 level, and there was frequency and urgency in urination. MRI of cervical spine showed old compressive fractures of C7 and T1, significant canal stenosis and multilevel spondylosis. Somatosensory evoked responses were consisted with cervical radiculopathies.

4. Thoracolumbar spondylosis, lumbosacral radiculopathies and stenosis of the lumbar spinal canal:

There were 8 patients with the diagnosis of fibromyalgia who had multi-level paraspinal radiculopathies of the dorsal mixed sensory-motor rami of the spinal nerves. Needle electromyography showed widespread denervation of the

paraspinal thoracolumbar segments. Duloxetine and either gabapentin or pregabalin had been effective for this type of neuropathic pain. There were 2 patients with the diagnosis of fibromyalgia who complained of pains in the trunk, low back pain and cramps in both legs. In both cases MRI of the lumbar spine showed severe spinal stenosis at L3-L4 and L4-L5. Epidural blocks help from 1 to 2 months in both cases. They have been discussing surgical interventions with neurosurgeons.

5. Chronic inflammatory demyelinating neuropathy:

A 56-year-old man had pain in the trunk and in the back. A rheumatologic work up was negative. He was seen by a neurologist at the neurology clinic who found mild muscular weakness, distal sensory deficit to pinprick and absent muscle stretch reflex. Nerve conduction studies showed prolonged latencies, temporal dispersion of compound muscle action potentials, and slow nerve conduction studies ranging from 29 to 32 meters per second. Sensory nerve conduction studies showed absence response or prolonged latencies. F waves (proximal conduction) were prolonged. Needle electromyography showed paucity of fibrillations and a drop out of motor unit action potentials. These findings were indicative of a chronic diffuse demyelinating polyneuropathy. He received intravenous immunoglobulin at 400 mg/kilo/day for 5 days. Slight improvement was noted after repeating treatment every 3 months. The diagnosis was chronic inflammatory demyelinating polyneuropathy. Another patient with paraspinal and extremity muscle pain and paresthesias in the feet was found on electrodiagnostic studies to have a demyelinating neuropathy. She responded to intravenous immunoglobulin.

6. Bilateral cerebral infarcts: A 52-year old woman had constant cervical and thoracolumbar paraspinal pains:

Four years prior to the presentation she suffered recurrent ischemic cerebral infarcts with consequent residual upper motor neuron weakness in both sides of the body and a spastic gait. Extensive evaluation showed that she had antiphospholipid antibody syndrome; and on MRI she had multiple high intensity signals in the cerebrum. She had received daily physical therapy and gait training. The muscle pain in paraspinal muscles and shoulder and pelvis girdle muscles appeared to have been a result of gait imbalance with unequal vectors and consequent unequal resultant of force reeling the body to one side; and as a consequence, the patient would compensate by tilting her body to the other side to prevent falling.

7. Myopathy:

Myopathy was associated with the concomitant use of statin and gemfibrozil. One patient with myalgias taking gemfibrozil and statin was referred to Neurology for electrodiagnostic studies. CK was elevated and needle electromyography showed early full recruitment of short duration abundant small polyphasic potentials compatible with myopathy in some muscles.

8. Giant cell arteritides:

An 81-year-old woman mother of an intensive care unit nurse. Her daughter brought her to clinic because she was concerned about her mother constant neck and shoulder pain and headaches and the nurse thought that her mother could have something different from the fibromyalgia for which she was being treated. No acute visual symptoms. She had nothing significant on neuroimaging of brain and cervical spine. She had abnormal electrocardiographic changes of cardiac ischemia. The sedimentation rate was 59. Temporal artery biopsy was positive for temporal arteritides. The shoulder myalgias were explained by arteritis of the occipital arteries. Her myalgia pains significantly improved on steroids.

4. DISCUSSION

Many patients with neuropathic and non-neuropathic pains of different causes and locations suffer from an amplification of pain process that leads to diffuse pain, allodynia and a chronic dysfunctional pain syndrome. There is a hard core of organicity that generates pain. The hard core is not always “fibromyalgia” as a disease entity, but instead a symptom of another condition. This hard core can be due to multilevel chronic radiculopathies, or irritation of the dorsal roots by spinal stenosis, myeloradiculitis like in NMO, and MS, peripheral neuropathy, muscle ischemia like in giant cell arteritides, and myositis. The problem is more complex when in addition to myalgias there is a clinical consolidated collection of diagnoses that may include muscle tension headache, major depressive disorder, constant low back pain,

paraspinal pains, temporomandibular joint pain, bruxism, sleep apnea, insomnia, chronic fatigue syndrome, irritable bowel syndrome, postural orthostatic tachycardia syndrome, orthostatic intolerance, restless leg syndrome, allodynia, posttraumatic stress disorder and dysmenorrhea.6 These patients should be approached in a manner which gives utmost confidence and the physicians should be sympathetic and never use harsh or inquisitional methods or intimidations. A detailed history and a thorough neurologic investigation and repeated examinations may be necessary.

The Criteria for the Diagnosis of Fibromyalgia11 do not curb clinicians from making the diagnosis of fibromyalgia too frequently in patients with generalized pain, myalgias, insomnia and depression. There are clinicians who have found that more than two-third of patients with diagnosis of fibromyalgia have symptoms related to the cumulative effects of multiple cervical trauma that lead to cervical spondylosis, radiculopathies and myelopathic cord compression.2, 12, 13,14,15,16,17

Denervation of paraspinal muscles in spinal disorders is associated with posterior ramus radiculopathies due to spondylosis, disc herniation, spinal stenosis, or stretching or damage to dorsal primary rami with consequent constant paraspinal pain.18 Radiculopathies produced by stenosis of spinal canal or by spondylosis are the most common pathologic process diagnosed as fibromyalgias. Myeloradiculitis produced by NMO10 without showing overt clinical evidence of myelopathy such as hyperactive reflexes, sensory deficit or pathologic reflexes can be a clinical dilemma. Likewise, posterior ramus radiculopathy18 may be difficult to diagnose unless they have needle electromyography of paraspinal muscles. Paraspinal muscle denervation may be cause by stretching of the dorsal ramus that innervate the paraspinal muscles but not the limb muscles and pain may be localized to paraspinal muscles only.18 In these patients, every effort should be done to extend the neurologic examination with appropriate relevant diagnostic exploration

There is electrodiagnostic and magnetic resonance evidence that patients with fibromyalgia and diffuse limb pains have inflammatory involvement of peripheral nerves due to underlying peripheral nerve pathology.19

Chronic myalgias due to myopathy as a result of statin and gemfibrozil are seen occasionally by physicians who perform electromyography. Also there are reports of adults diagnosed as having fibromyalgias who have specific type of myopathies such as mitochondrial myopathy.1 Treatment of such myopathies may result in resolution of symptoms.

The neurologic examination will guide the diagnostic exploration which in most cases includes neuroimaging and electrodiagnostic studies. In a rare case a temporal artery biopsy showed that the myalgias were the result of muscle ischemia due to vasculitis.

The following list may serve as a guidance to perform further laboratory tests when indicated based on relevant physical complaints and physical findings:

Complete blood count, erythrocyte sedimentation rate, complete metabolic panel, 2 hour-post prandial blood sugar, iron, TSH and free T3; chest x-ray, or CT of the chest, abdomen and pelvis if there is suspicion of malignancy; skeletal survey, serum protein immunofixation electrophoresis, bone scan, bone marrow biopsy, angiotensin-converting enzyme, hepatitis B and C panels, CK, methylmalonic acid and homocysteine when indicated; paraneoplastic panel, fatty acid profile, lactate, amylase, and lipase, serologic tests for syphilis, Lyme disease detection antibodies, human T-lymphocyte virus type I (HTLV1), and human immunodeficiency virus detection antibodies. Fat pad aspirate or rectal and salivary gland biopsy may be necessary in cases of peripheral neuropathy when amyloidosis is suspected;

In rare occasions in cases of suspected hereditary neuropathies when the phenotype is not enough for diagnosis there may be a need for genetic testing

Electrodiagnostic studies (nerve conduction studies, needle electromyography and repetitive nerve stimulation) are essential in cases of suspected motor neuron disease, denervation of paraspinal muscle, spondylosis, radiculopathies, peripheral neuropathies, neuromuscular junction conditions and myopathies.

MRI of spine if there was a suspicion of nerve root enhancement in CIDP other inflammatory processes of nerves and plexus; nerve root clumping in arachnoiditis, nerve enlargement in tumors or cervicothoracic myelopathy and multilevel root compressions; serum and 24-hour urine screening for heavy metals. Rheumatologic workup may include serum ANA, ASO, CRP, RA rheumatoid factor, C reactive protein, proteinase 3, myeloperoxidase, SSA (Ro), Sjögren's syndrome-SSB (LA), antibody (Ab) to SM (Smith) antigen, , Ab to double-stranded DNA. ribonucleoprotein,

complement levels (C3, C4), Schirmer's test, anti-centromere Ab, and Ab to U1-nRNP; circulating Ab (Antineurotrophil cytoplasmic Abs- ANCA), cryoglobulins, NMO-IgG antibodies against the aquaporin-4 antigen;

Multifocal motor neuropathy is associated with IgM serum antibodies against gangliosides mainly GM1 and GM2. Anti MAG may be associated to acquired demyelinating neuropathy. Anti sulfatides antibodies (sensory neuropathies) and anti GD1 antibodies, anti-GQ1b, antibodies and antiGM1 antibodies may be associated with sensory neuropathies. Miller Fisher syndrome may be associated with GQ1b antibody

In deficiency states (including s/p gastric surgery): thiamine level, B12 and folate blood level MRI of the cord, vitamin E, serum copper (excessive zinc intake impairs absorption of copper), antigliadin antibodies, small intestine biopsy. Antigliadin IgG and IgA and transglutaminase when indicated. Sleep studies if indicated by history of snoring and sleep apnea.

5. CONCLUSION

Many people with the diagnosis of fibromyalgia have medical conditions that can reasonably explain the symptoms diagnosed as fibromyalgia; that is to say, fibromyalgia is not a primary disease but a symptomatic expression of another underlying process. 2,4 Therefore the causes of fibromyalgia syndrome should be investigated appropriately. Fibromyalgias are neuropathic and non-neuropathic pains produced by different causes such as multi-level radiculopathy of the dorsal mixed ramus of the spinal nerves, other special types of peripheral neuropathies, myopathies, and intrinsic cord lesions producing irritation of the entry zone of the dorsal root nerve, and generalized osteoarthritis. There is also a need to consider allodynia in depressed patients who exaggerate the response to touch, powered by their knowledge that such a response is necessary to convince the physician that there is something wrong with them. Once the physician makes the diagnosis of fibromyalgia patients appear to treasure such diagnosis because through it "there is a presumptive physical explanation for their symptoms, and a relief to have a word for what plagued them." 20 Further evaluation will show that many of these patients have denervation of paraspinal muscles due to posterior ramus neuropathy; or myeloradiculopathy due to NMO or multiple sclerosis with a well-defined neuropathic pain responsive to medicines that are used to treat neuropathic pain such as duloxetine, gabapentin and pregabalin; and these are the medications that indeed have been advertised to treat "fibromyalgias." All patients with fibromyalgia syndromes must be evaluated as having a secondary symptomatic manifestation of an underlying causative disorder before the diagnosis of fibromyalgia is given.4 This is an important issue since treatment of underlying condition in many cases result in resolution of symptom. Given the absence of clinical findings and laboratory supportive tests there is simply no compelling reason to believe that fibromyalgia is a defined clinical condition.

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