

Neurologic Signs and Symptoms in Fibromyalgia

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Objective. To determine the type and frequency of neurologic signs and symptoms in individuals with fibromyalgia (FM).

Methods. Persons with FM (n = 166) and pain-free controls (n = 66) underwent systematic neurologic examination by a neurologist blinded to disease status. Neurologic symptoms lasting at least 3 months were assessed with a standard questionnaire. We used logistic regression to evaluate the association of neurologic symptoms and examination findings with FM status. Within the FM group we examined the correlation between self-reported symptoms and physical examination findings.

Results. Age- and sex-adjusted estimates revealed that compared with the control group, the FM group had significantly more neurologic abnormalities in multiple categories, including greater dysfunction in cranial nerves IX and X (42% versus 8%) and more sensory (65% versus 25%), motor (33% versus 3%), and gait (28% versus 7%) abnormalities. Similarly, the FM group had significantly more neurologic symptoms than the control group in 27 of 29 categories, with the greatest differences observed for photophobia (70% versus 6%), poor balance (63% versus 4%), and weakness (58% versus 2%) and tingling (54% versus 4%) in the arms or legs. Poor balance or coordination, tingling or weakness in the arms or legs, and numbness in any part of the body correlated with appropriate neurologic examination findings in the FM group.

Conclusion. This blinded, controlled study demonstrated neurologic physical examination findings in

persons with FM. The FM group had more neurologic symptoms than did the controls, with moderate correlation between symptoms and signs. These findings have implications for the medical evaluation of patients with FM.

Fibromyalgia (FM) is a condition of unknown etiology characterized by widespread muscle pain, sleep disturbances, fatigue, and various neurologic symptoms (1). Despite considerable speculation and research, the etiology of FM remains uncertain. Although a wide range of abnormalities and causes have been proposed (2,3), none have gained widespread acceptance or withstood the rigors of repeated scientific inquiries.

FM patients frequently report an onset of illness following a motor vehicle accident, surgery, or other trauma (4), often in the craniocervical region. Indeed, FM is 13 times more common after neck injuries than after lower extremity injuries (5,6). Neurologic symptoms such as paresthesias, blurred vision, numbness, and weakness are commonly reported by FM patients, with numbness present in up to 84% of individuals (1,4,7–9). These symptoms, along with head and neck pain and difficulty walking (10,11), overlap with symptoms experienced by patients with neuroanatomic abnormalities such as Arnold-Chiari I malformations, spinal canal stenosis, and positional cervical compression (5,12). Although highly controversial, it has been suggested that Arnold-Chiari I malformation and FM are comorbidities, and some practitioners have recommended decompressive craniotomy and cervical laminectomy as treatments for FM (13), particularly in those manifesting signs of cervical myelopathy (14). However, to our knowledge, no blinded, controlled studies have systematically assessed objective neurologic findings in patients with FM.

The goals of this study were to conduct blinded neurologic examinations and assess recent symptoms in FM patients and pain-free controls. We also correlated signs and appropriate symptoms in the FM group. An

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excess of objective findings, in tandem with correlating symptoms, would suggest the need to perform detailed neurologic examinations on all FM patients, and also suggests a possible neuroanatomic origin of FM (5,14).

SUBJECTS AND METHODS

Participants. All individuals in the present study were participating in a study of Arnold-Chiari I malformation and FM. Individuals with FM were identified either through an academic referral clinic devoted to the evaluation of chronic pain and fatigue or through local advertising in the greater Seattle, WA metropolitan area. Subjects in the FM group were required to be age ≥ 18 years, to be nonpregnant if female, and to have FM by self-report or by review of the medical records. A Research Coordinator trained by one of our team (DB) verified the diagnosis of FM according to the American College of Rheumatology 1990 criteria by confirming the presence of chronic, widespread pain and ≥ 11 of 18 tender points on examination (1).

Control participants, recruited through advertising at 3 medical institutions, were required to be age ≥ 18 years, to be nonpregnant if female, and to deny having chronic, widespread pain or chronic fatigue. The Research Coordinator screened controls on the telephone for pain and FM-related symptoms using the validated London Fibromyalgia Epidemiology Study Screening Questionnaire (15). This research was reviewed and approved by the University of Washington Institutional Review Board.

Symptoms. A self-report questionnaire inquired about past and current health status including symptoms characteristic of FM and neurologic functioning (visual, auditory, balance, coordination, motor, sensory, and gait). Subjects were asked to indicate which of these symptoms they had experienced with or without headache for at least 3 months.

Signs. A neurologic examination was performed by a board-certified neurologist (NFW) blinded to participant status. Neurologic findings were recorded on a standardized form indicating the presence or absence of abnormalities. Examination of cranial nerves I–XII assessed smell, visual acuity, extraocular muscle palsy, papilledema, visual field cuts, pupillary shape, symmetry and reactivity, facial sensation, masseter strength, facial symmetry, hearing, nystagmus, gag reflex, hoarseness, shoulder shrug, and tongue bulk and displacement. The cerebellar examination assessed the presence of tremor, dysdiadochokinesia, and dysmetria. To determine sensory deficits, participants were evaluated for analgesia or anesthesia, dissociated sensory loss, and impaired proprioception, vibratory sensation, temperature sensation, or pinprick sensation. Dorsal columns were assessed with the Romberg sign. Motor examination ascertained weakness, impaired fine motor control, decreased or increased tone, and atrophy. Reflex testing evaluated patients for hyper- or hyporeflexia, Babinski sign, clonus, and trophic joint changes. Gait was assessed for ataxia and tested formally with tandem maneuvers, and stance addressed the presence or absence of scoliosis or kyphosis.

Correlation of signs and symptoms. To better understand the relationship of symptoms and signs, specific symptoms were linked a priori with neurologic signs in the FM group as follows: 1) difficulty in swallowing was linked to

abnormal gag reflex; 2) tingling in arms or legs and numbness in any part of the body were each linked to analgesia/anesthesia or impairments in vibration, temperature, or pinprick sensation; 3) weakness in arms or legs was correlated with the presence of weakness or atrophy; and 4) poor balance, poor coordination, or abnormal clumsiness was linked to positive Romberg sign, ataxia, impaired proprioception, or abnormal tandem gait. We also linked poor coordination with dysdiadochokinesia and abnormal clumsiness with impaired fine motor control on examination.

Statistical analysis. Participants missing ≥ 1 key analysis variable (24 [9%] of 256) were excluded from all analyses, leaving 232 subjects for this study. Descriptive statistics were reported as means for continuous variables and percents for categorical variables. We used logistic regression to evaluate the association of neurologic symptoms and examination findings with FM status. A series of models was fit in which symptoms and signs were the outcome variables and the independent variables included an indicator of FM, age, and sex. We present age- and sex-adjusted prevalence estimates and 95% confidence intervals. Wald tests from the age- and sex-adjusted models were used to test for a statistically significant difference between the FM and control groups. For the objective findings, we limited statistical testing to overall abnormality of ≥ 1 condition in each symptom category; however, for completeness, we report prevalence estimates and 95% confidence intervals for each condition. In some instances, odds ratios were not obtainable due to the absence of controls with neurologic symptoms or signs. In this case, statistical testing was performed with Fisher's exact test. We examined the association between self-reported symptoms and signs in the FM group using tetrachoric correlations, which provide an estimate of the underlying correlation when examining the relationship between 2 dichotomous variables (16). Analyses were completed using Stata/SE 10.1 for Windows (2008; Stata, College Station, TX).

RESULTS

Demographics. There were 166 subjects in the FM group and 66 subjects in the control group. Subjects in the FM group were older than those in the control group (mean age 50 years versus 41 years), and many more were women (94% versus 50%). The majority of participants in both groups were white (89% of the FM group and 71% of the control group).

Differences between groups in symptoms. The FM group had significantly more neurologic symptoms than the control group in 27 of 29 categories investigated (Table 1). These symptoms encompassed a large range of neurologic functioning, including the visual and auditory systems, cerebellum, cranial nerves, respiration, and sensory and motor systems. The greatest differences were observed for "bright lights bother eyes" (70% versus 6%; $P < 0.01$), "poor balance" (63% versus 4%; $P < 0.01$), and "weakness" (58% versus 2%; $P < 0.01$) and "tingling" (54% versus 4%; $P < 0.01$) in the "arms or legs."

Table 1. Prevalence of neurologic symptoms in the subjects with and those without FM*

Symptom	FM (n = 166), % (95% CI)	No FM (n = 66), % (95% CI)
Blurred vision	46 (38–54)†	6 (2–15)
Bright lights bother eyes	70 (62–78)†	6 (2–17)
Double vision	15 (10–23)‡	1 (0–8)
Loss of peripheral vision	10 (6–17)‡	1 (0–8)
Floaters, wavy lines, flashing lights	42 (34–50)	25 (15–39)
Dizziness	53 (45–62)†	4 (1–13)
Poor balance	63 (54–71)†	4 (1–13)
ringing in ears	46 (38–55)†	13 (6–25)
Ear pressure	35 (27–44)†	2 (0–8)
Decreased hearing	25 (18–33)‡	7 (2–18)
Vertigo	30 (23–39)†	1 (0–10)
Noises or talking that hurts ears	45 (36–54)†	1 (0–10)
Difficulty swallowing	29 (23–37)†	0
Sleep apnea	32 (24–41)†	3 (1–12)
Tremors	16 (11–23)‡	3 (1–13)
Palpitations	28 (21–36)†	3 (1–12)
Poor coordination	45 (38–53)†	0
Constant throat pain or sore throat	35 (27–43)†	1 (0–9)
Lightheadedness	52 (45–60)†	0
Shortness of breath	39 (31–49)†	1 (0–9)
High blood pressure	23 (16–32)†	4 (1–13)
Tingling in arms or legs	54 (46–63)†	4 (1–14)
Numbness in any part of body	50 (41–58)†	3 (1–10)
Burning feeling in arms, legs, face, or torso	38 (30–47)†	2 (0–11)
Cannot feel hot objects in hands	3 (1–8)	0
Weakness in arms or legs	58 (49–66)†	2 (1–10)
Abnormal clumsiness	38 (31–46)†	0
Loss of muscle mass	13 (9–19)†	0
Incontinence of urine	25 (18–33)‡	7 (3–19)

* Prevalence estimates and *P* values are adjusted for age and sex when possible based on sample composition. Otherwise, estimates are unadjusted and *P* values are by Fisher's exact test. 95% CI = 95% confidence interval.

† *P* < 0.01 versus subjects without fibromyalgia (FM).

‡ *P* < 0.05 versus subjects without FM.

Differences between groups in signs. The detailed neurologic examination revealed multiple differences between the FM group and the pain-free controls. Compared with the control group, the FM group was characterized by more hoarseness, suggesting greater dysfunction in cranial nerves IX and X (42% versus 8%; *P* < 0.01). The FM group also had more sensory findings than controls (65% versus 25%; *P* < 0.01), consisting of diverse abnormalities including pinprick, temperature, and vibratory sensation as well as analgesia/anesthesia. Specific dermatomal distributions were not identified. The FM group also had more abnormal findings on the motor examination than did controls (33% versus 3%; *P* < 0.01), due primarily to weakness on strength testing and impaired fine motor control. Involvement of specific muscle groups was not noted. The FM group also had more gait problems than their pain-free counterparts (28% versus 7%; *P* < 0.01), particularly with tandem gait. Table 2 provides further details, including other

results of the neurologic examination that did not differ between the 2 groups.

Correlations observed between signs and symptoms. Significant correlations were observed between several signs and symptoms in the FM group. Symptoms of both numbness in any location ($\rho = 0.29$, *P* = 0.03) and tingling in arms or legs ($\rho = 0.26$, *P* = 0.05) correlated with corresponding examination findings. Likewise, poor balance ($\rho = 0.33$, *P* = 0.01), poor coordination ($\rho = 0.31$, *P* = 0.01), and weakness in arms or legs ($\rho = 0.31$, *P* = 0.03) were associated with appropriate objective findings. Lesser correlations were observed for the symptom of abnormal clumsiness ($\rho = 0.23$, *P* = 0.08).

DISCUSSION

To our knowledge, this is the first blinded, controlled study to demonstrate objective findings on de-

Table 2. Prevalence of neurologic findings in the subjects with and those without FM*

Cranial nerve or neurologic function, sign	FM (n = 166), % (95% CI)	No FM (n = 66), % (95% CI)
Cranial nerve I		
Impaired sense of smell	2 (1-7)	1 (0-8)
Cranial nerve II, III, IV, VI		
Visual acuity	72 (62-80)	66 (48-79)
Abnormal for ≥ 1 condition below	14 (9-20)	10 (4-21)
Extraocular muscle palsy	3 (1-8)	1 (0-8)
Papilledema	0	0
Field cut	1 (0-4)	3 (0-11)
Pupils equal, round reactive to light/accommodation	11 (7-18)	5 (1-14)
Cranial nerve V		
Abnormal for ≥ 1 condition below	12 (7-18)	2 (0-11)
Facial sensation decreased	11 (7-17)	2 (0-10)
Chewing decreased	1 (0-4)	0
Cranial nerve VII		
Facial musculature asymmetric	1 (0-5)	0
Cranial nerve VIII		
Abnormal for ≥ 1 condition below	7 (3-13)	7 (2-21)
Hearing abnormal	5 (3-11)	7 (2-21)
Nystagmus abnormal	1 (0-5)	0
Cranial nerve IX, X		
Abnormal for ≥ 1 condition below	42 (34-51)†	8 (3-19)
Gag reflex abnormal	6 (3-11)	2 (1-10)
Hoarseness	38 (30-47)	5 (2-16)
Cranial nerve XI		
Shoulder shrug asymmetric	0	0
Cranial nerve XII		
Abnormal for ≥ 1 condition below	1 (0-4)	0
Tongue atrophy	1 (0-4)	0
Tongue displacement	0	0
Cerebellar		
Abnormal for ≥ 1 condition below	16 (10-23)	4 (1-16)
Tremor	7 (4-12)	2 (0-10)
Dysdiadochokinesia	7 (3-13)	2 (0-12)
Dysmetria on finger nose test	1 (0-4)	0
Romberg sign present	7 (4-12)	0
Sensory		
Abnormal for ≥ 1 condition below	65 (56-72)†	25 (14-39)
Analgesia or anesthesia	22 (16-30)	2 (0-8)
Dissociated sensory loss	8 (5-13)	0
Impaired proprioception	4 (2-9)	0
Impaired vibratory sensation	38 (30-47)	20 (11-35)
Impaired temperature sensation	40 (32-49)	6 (2-17)
Impaired pinprick sensation	47 (39-56)	7 (3-18)
Motor		
Abnormal for ≥ 1 condition below	33 (25-41)†	3 (1-11)
Weakness	21 (14-29)	2 (0-13)
Impaired fine motor control	11 (7-17)	1 (0-8)
Decreased tone	0	0
Increased tone	1 (0-5)	0
Atrophy	4 (2-9)	0
Reflexes		
Abnormal for ≥ 1 condition below	57 (49-65)	45 (31-60)
Not symmetric or physiologic	52 (43-60)	35 (22-50)
Hyperreflexia	14 (10-21)	5 (1-13)
Hyporeflexia	39 (31-48)	32 (20-47)
Joint abnormalities-trophic	4 (2-10)	1 (0-11)
Positive Babinski sign	1 (0-4)	0
Clonus	2 (1-6)	2 (0-10)
Stance		
Abnormal for ≥ 1 condition below	18 (12-26)	11 (5-25)
Scoliosis	2 (1-5)	0
Kyphosis	17 (11-25)	11 (5-25)
Gait		
Abnormal for ≥ 1 condition below	28 (21-38)†	7 (3-18)
Tandem abnormal	26 (18-35)	6 (3-18)
Ataxia	6 (3-11)	0

* Prevalence estimates and *P* values are adjusted for age and sex when possible based on sample composition. Otherwise, estimates are unadjusted and *P* values are by Fisher's exact test. Significance testing was only performed for overall abnormality of ≥ 1 sign in each category. See Table 1 for definitions.

† *P* < 0.01 versus subjects without FM.

tailed neurologic examination in FM. Specifically, we found that individuals with FM exhibited abnormalities of cranial nerves IX and X, sensation, strength, and gait as compared with pain-free controls. As expected, symptoms affecting all neurologic systems were more common in the FM group, with correlations observed between many of these symptoms and objective examination findings. These neurologic signs support the possibility of a craniocervical neuroanatomic cause for the FM symptom complex, such as Arnold-Chiari I malformation, spinal canal stenosis, or positional (flexion/extension) cervical compression (5,12,14).

In this regard, our results are consistent with the findings of 2 recent case series that assessed symptoms and performed detailed neurologic examinations and neuroimaging in FM patients (5,12). In one study of 270 patients with FM, results of detailed neurologic examinations were consistent with cervical myelopathy (5). Reported findings included upper thoracic spinothalamic sensory level (83%), hyperreflexia (64%), inversion of the radial periosteal reflex (57%), positive Romberg sign (28%), ankle clonus (25%), positive Hoffman sign (26%), impaired tandem walk (23%), dysmetria (15%), and dysidiadochokinesia (13%). Neuroimaging revealed that 20% of participants had cerebellar tonsillar ectopia >5 mm, and 46% experienced clinically important spinal canal stenosis with the neck positioned in mild extension. In another study (12), 49 FM patients with signs such as positional cervical pain, abnormal grip, positive Romberg sign or gait dysfunction, and symptoms of dizziness and unsteadiness underwent flexion/extension midline sagittal magnetic resonance imaging with transaxial measurement of cervical spinal canal diameter. Details of the neurologic examination were not presented, but almost 4% of these highly selected patients had Arnold-Chiari I malformation. In addition, 71% showed evidence of intermittent cervical spinal cord compression, usually in extension, but neutral sagittal cervical spine views only documented cervical spine abutment in 29%. Taken together, these studies suggest that neurologic findings are common in FM and may, in some cases, have a neuroanatomic basis.

We also found significant correlations between objective neurologic examination findings and symptoms in the FM group across multiple neurologic systems. This observation underscores the need to perform careful neurologic examinations in all FM patients, particularly those with neurologic symptoms. These findings are congruent with possible neuroanatomic causes of FM in some patients (5,14). Of note, no investigators have reported the results of neurologic examinations or radiographic or neuroimaging data that would permit recommendations to be made regarding which patients

should be evaluated for neuroanatomic conditions. Even so, the potential importance of identifying and treating underlying causes of the symptoms of the FM complex was suggested by a recent nonrandomized study of surgical versus nonsurgical treatment of cervical myelopathy (14). The group that underwent surgical treatment experienced reductions in the number of body regions with pain as well as improvements in neurologic signs and physical and mental quality of life (14). Although the nonrandomized nature of the intervention raises the prospect of confounding by indication, it highlights the need for carefully designed, rigorously blinded and controlled studies of craniocervical neuroanatomy in FM.

This study has several limitations. First, there is a concern about subject referral and the highly selected sample of patients with FM. Second, our samples were different with respect to age and sex. We addressed this issue by adjusting for age and sex in our logistic regression analysis whenever possible for the primary examination and symptom end points. In the instances when no participants in the control group experienced a sign or symptom, we could not perform an adjusted analysis. Third, a higher than expected percentage of controls was found to have asymmetric reflexes or hyporeflexia, possibly due to the dichotomous nature of the examination data. Although this could have overwhelmed and obscured any subtle reflex differences between the 2 groups, the fact that the same blinded neurologist performed all examinations obviates any general bias in these estimates. Finally, findings on the neurologic examination can be influenced by factors such as patient effort, pain, and the patient's understanding of the examination, and in some cases findings such as hoarseness may have alternative explanations. In cases in which the effort was variable or the subject appeared to be confused by the examination, the examining neurologist paused to reexplain the examination and reminded the subject to concentrate and give his or her best effort.

In conclusion, we documented that selected abnormalities in cranial nerves and sensory, motor, and gait functions were more common in subjects with FM than in pain-free controls. Neurologic symptoms were also common, and, importantly, correlated with examination findings in many instances. Future investigations of the underlying neuroanatomy of FM could advance our understanding of diagnosis and treatment.

AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. Watson had full access to all of

the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Watson, Buchwald, Goldberg, Ellenbogen.

Acquisition of data. Watson, Ellenbogen.

Analysis and interpretation of data. Watson, Buchwald, Goldberg, Noonan, Ellenbogen.

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Next Editor of *Arthritis & Rheumatism*

At its August 1, 2009 meeting, the American College of Rheumatology Board of Directors approved the recommendation that Joan Bathon, MD be named Editor, *Arthritis & Rheumatism*, July 2010–June 2015 term. Between April 1 and June 30, 2010, Dr. Bathon will work concurrently with the current Editor, Michael Lockshin, MD. Dr. Lockshin and his editorial team will continue to handle manuscripts submitted before April 1 for which a decision has not yet been made, and new manuscripts will be handled by Dr. Bathon and her team. Dr. Bathon is Professor of Medicine at Johns Hopkins University School of Medicine, Deputy Director of the Division of Rheumatology at Johns Hopkins Bayview Medical Center, and Director of the Johns Hopkins Arthritis Center.