Postpoliomyelitis Syndrome: Assessment of Behavioral Features

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Summary: Postpoliomyelitis syndrome (PPS) is an increasingly recognized phenomenon characterized by late-onset weakness, pain, and fatigue. Psychiatric and cognitive disturbances have been noted in postpoliomyelitis patients, but the relationship of these symptoms to PPS is unknown. We examined postpoliomyelitis patients with and without PPS using objective neuropsychological and neuropsychiatric procedures. Our results suggest that disturbances of mood were common and that subtle cognitive deficits also occured in postpoliomyelitis patients. However, patients with PPS did not have greater depression or cognitive deficits compared to postpoliomyelitis patients without PPS. **Key Words:** Postpoliomyelitis syndrome, Mood disturbances, Cognitive deficits. **NNBN 2:272-281, 1989**

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Recent reports have described the emergence of late-onset symptoms in patients who had previously had acute poliomyelitis *(Dalakas et al., 1986; Cashman et al., 1987; Dalakas, 1988; Eulbert et el., 1988; Klingman et al., 1988)*. The new onset of lower motor neuron symptoms, including pain, weakness, and fatigue, has been referred to as postpoliomyelitis syndrome (PPS) *(Cashman et al., 1987)*. Other symptoms, possibly unrelated to lower motor neuron involvement associated with delayed onset, have also been described, including parkinsonian symptoms *(Vincent and Meyers, 1978)*, memory and concentration difficulties *(Lane et al., 1974)*, mood and emotional disturbances *(Lane et al., 1974; Kohl, 1987)*, pain alone *(Smith et al., 1987)*, and central and peripheral sleep apnea *(Fischer, 1987)*. Lane et al. *(1974)* suggested that complaints of late-onset depression and memory difficulty are associated with late-onset ventilatory insufficiency.

The association of nonmotor symptoms, including mood and cognitive changes, and PPS is unknown. In

this study, clinical and objective procedures were used to examine possible neuropsychiatric and neuropsychological differences between postpoliomyelitis patients with and without PPS.

METHODS

Subjects

Thirty consecutive patients from a large orthopedic outpatient poliomyelitis clinic were examined after obtaining informed consent. All patients had acquired poliomyelitis between 1923 and 1958, except for one patient who, having failed to be vaccinated, acquired the illness in 1965. The diagnosis of poliomyelitis was confirmed by neurological examination and by review of the history. Patients were considered to have had poliomyelitis if they had sustained an acute onset of fever and paralysis with the physical findings of asymmetrical weakness, hyporeflexia, muscle atrophy, lack of sensory involvement, and a prolonged stable course after convalescence. None of the patients had a history of familial amyotrophy, and none had a history of psychiatric disease prior to the onset of poliomyelitis. Five potential study candidates were excluded: two for failing to adequately complete the examinations, one due to a history of a basilar skull fracture, and two for failing to meet criteria for confirmation of paralytic poliomyelitis. Of the 25 patients included in the study, 20 were women and 5 were men.

Clinical Examination

Complete history and neurological examinations were done by the same neurologist, who was experienced in the evaluation of neuromuscular disease and neuropsychiatric disorders. Specific data concerning recent onset of symptoms, including fatigue, pain, weakness, and loss of function were elicited. Patients were considered to have PPS if they had: (1) a credible history of poliomyelitis; (2) initial partial recovery; (3) at least 10 years of stability; (4) subsequent development of progressive weakness, pain, and fatigue; (5) a change in function as a result of increasing pain, weakness, and fatigue; (6) weakness on exam in areas corresponding to symptomatic complaints; and (7) no other cause for progressive weakness and pain, such as arthritic changes or nerve entrapment. The Karnofsky scale was used to rate current disability *(Grieco and Long, 1984)*.

Psychiatric history was obtained by a semistructured clinical interview, concentrating on symptoms related to mood, psychosis, and personality disorders using Diagnostic and Statistical Manual of Mental Disorders criteria (third edition, revised)*(APA, 1987)*.

Pulmonary Function Tests

Assessment of pulmonary function included measurement of forced vital capacity (FVC) via a Collins Vitalometer. Percent FVC was determined by the method proposed by *Comroe et al. (1955)*. P_aCO_2 determinations were made on a Beckman LBI Medical Gas Analyzer utilizing rebreathing techniques. P_VCO_2 was calculated by multiplying the P_VCO_2 value by 0.8 *(Powels, 1978)*.

Neuropsychometric Tests

Attention and Psychometer Speed

The Digit Span subtest of the Wechsler Adult Intelligence Test --- Revised *(Wechsler, 1981)* was used to measure verbal attentional abilities, and the Trail-Making Test Part A *(Reitan, 1958)* was used to assess attention, visual scanning, and psychometer speed.

Mental Set Function

The Stroop Color Name Test *(Stroop, 1935)*, and the Trail Making Test Part B *(Reitan, 1958)* were used to measure response inhibition and ability to shift mental set, respectively. The Symbol Digit Test (oral presentation) was administered to assess mental set function as well as psychomotor speed and novel problem solving ability *(Smith, 1968)*.

Memory

Recent verbal memory was evaluated using the Rey Auditory Verbal Learning Test (RAVLT) *(Taylor, 1959)* and recall of the Rey Osterreith Complex Figure (ROCF) *(Lezak, 1983)* following a 3-min delay was used to assess nonverbal memory.

Word List Generation

The Controlled Word Association Test (FAS) *(Benton, 1973)* was used to assess speed of retrieval from long-term lexical memory.

Visuospatial Ability

Copy of the ROCF was used to assess constructional skills *(Lezak, 1983)*.

Mood and Personality

The Beck Depression Inventory (BDI) *(Beck et al., 1961)* and Minnesota Multiphasic Personality Inventory (MMPI) *(Dahlstrom et al., 1975)* were used in addition to the clinical psychiatric interview to examine mood and personality.

RESULTS

There was a strikingly high incidence of mood disorders in the postpoliomyelitis patients. Forty-six percent of those with PPS and 50% of those without PPS had evidence of depression (adjustment disorder, depressive episode, recurrent depression, bipolar episode), and 8% of the patients in each group had attempted suicide. Even when those with adjustment disorders are eliminated, the rate of mood disorder is substantial (39% in PPS patients, 33% in patients without PPS). Mean BDI scores were moderately elevated (11.5 and 12.1 in the PPS and no PPS groups, respectively). The most commonly endorsed items on the BDI in the group as a whole were related to somatic features, including fatigue (84%), difficulty working (72%), and concerns related to physical appearance (64%). Irritability (60%) and difficulty with sleep (64%) were also common. In contrast, feelings of guilt (12%) or of being punished (12%), as well as symptoms of anorexia (16%)and weight loss (20%), were least common. It is possible that somatic features were most common due to intrinsic features of the disease: however, there was no significant correlation between BDI scores and physical disability (r = -0.28, n.s.), suggesting that mood impairment was not simply a reaction to disability. Additionally, patients with positive psychiatric histories on clinical interview had higher average BDI scores (14.9) compared to those without positive histories (8.4), but this failed to reach statistical significance, $t(23) = 2.02$, $p < 0.06$.

The group as a whole performed within the average range on the majority of neuropsychological measures compared to existing normative data. There was, however, borderline impairment on nonverbal recall (8th percentile) *(Lezak, 1983)* and low average performance on a timid color-naming task (Stroop Part B)(10th percentile) *(Nehemkis and Lewinsohn, 1972)*.

As illustrated in Table 1, when the patient group was divided into those with (n = 13) and those without PPS (n = 12), there was no significant difference between the two patient groups with respect to age, education, age at onset of acute illness, overall disability, and gender distribution. Clinical history data related to the proportion of patients with current complaints of memory or concentration difficulties were also not significantly different between the two groups. Pulmonary function studies indicated that percent FVC was not different between the two groups. However, patients with PPS had significantly elevated P_aCO_2 levels compared to patients without PPS. In the group as a whole, P_aCO_2 of patients with complaints of decreased memory [$t(20) = 2.06$] or concentration [$t(20) = 1.29$] did not differ from those without such complaints.

TABLE 1. Demographic, clinical, and pulmonary data in patients with and without PPS

	PPS (n = 13)	No PPS (n = 12)	Significance
Demographics			
Age	49.6 ± 3.2	45.4 ± 2.8	$t = 1.0$, NS
Education	14.9 ± 1.0	13.3 ± 0.9	$t = 1.2$, NS
Age at onset	8.4 ± 2.1	8.6 ± 1.7	$t = -0.1$, NS
Disability	70.0 ± 2.5	64.2 ± 4.2	$t = 1.2$, NS
Male/Female	10/3	10/2	$x^2 = 0.2$, NS
Clincal			

DSMIII(R)			
Adjustment disorder with depressed mood	15	17	z = -0.06, NS
Major depressive episode - single	23	25	z = -0.02, NS
Major depressive episode - recurrent	08	08	NA*
Bipolar disorder	08	00	z = 1.10, NS
Suicide attempts	08	08	NA*
Cognitive complaints			
Memory	54	42	z = 0.60, NS
Concentration	46	33	z = 0.70, NS
Pulmonary			
P_aCO_2	37.7 ± 1.3	33.7 ± 0.7	t = 2.7, p < 0.01
FVC	99.1 ± 4.2	106.2 ± 4.2	t = -1.2, NS

* NA, not analyzed.

Demographic and pulmonary data are presneted as means ± SEM and clinical data as the percentage of patients with positive symptoms.

Primary data analysis involved comparisons of the two patient groups on the objective neuropsychological and neuropsychiatric measures. Sidak's procedure was used to adjust the level of statistical significance for multiple pair-wise comparisons. A *p*-value of 0.002 defined the corrected level of statistical significance. Due to the large number of comparisons and the lack of power associated with the small sample sizes, any differences between the groups may most accurately be interpreted as trends.

As seen in Table 2, none of the scores from the neuropsychiatric or neuropsychological measures were significantly different between the two groups. There was, however, a trend for patients with PPS to perform better on both the Symbol-Digit and Trails B tasks. These procedures are related to attention, speed of mental processing, and ability to shift mental set. There was a trend for patients with PPS to have higher scores on the MMPI scale related to introversion and lower scores on the scale related to symptoms of hypomania. Despite failing to meet statistical significance, the fact that these two neuropsychological and the two neuropsychiatric procedures measure similar functions enhances the reliability of the findings.

TABLE 2. Comparison of neuropsychological and neuropsychiatric measures between patients with

and without PPS.

Measures	PPS (n = 13)	No PPS (n = 12)	*t* Test
Neuropsychological			
Attention/psychomotor speed			
Digits Forward	6.9 ± 0.4	6.8 ± 0.5	0.14, NS
Digits backward	4.9 ± 0.3	4.5 ± 0.4	0.87, NS
Stroop			
A	44.5 ± 2.1	50.4 ± 3.5	-1.47, NS
B	58.3 ± 1.7	62.8 ± 2.5	-1.53, NS
Trail Making A	29.6 ± 2.2	31.9 ± 2.6	-0.68, NS
Mental set functions			
Trail making B	67.8 ± 4.2	92.0 ± 7.2	-2.98, p<0.007
Symbol digit	52.7 ± 1.4	44.8 ± 2.4	2.90, p<0.008
Stroop C	114.8 ± 4.5	116.8 ± 8.2	-0.23, NS
Memory			
Verbal memory			
RAVLT			
Trial 1	7.6 ± 0.5	7.0 ± 0.8	0.7, NS
Trial 5	12.6 ± 0.7	12.1 ± 0.9	0.5, NS
Recall	10.6 ± 0.8	9.8 ± 1.0	0.68, NS
Recognition	14.1 ± 0.3	13.8 ± 0.3	0.57, NS
False positives	1.0 ± 0.3	1.2 ± 0.5	-0.43, NS
Visual memory			
ROCF	14.5 ± 1.4	15.9 ± 1.9	-0.59, NS
Word list generation			
FAS	39.2 ± 3.0	37.8 ± 3.7	0.29, NS
Visuospatial ability			
ROCF (copy)	31.5 ± 0.6	32.6 ± 0.9	-1.04, NS

Neuropsychiatric				
	BDI	11.5 ± 2.7	12.1 ± 2.2	-0.16, NS
	MMPI			
		1	72.3 ± 2.6	64.9 ± 2.7	1.97, NS
		2	72.5 ± 3.9	60.7 ± 4.4	2.01, NS
		3	70.4 ± 2.4	68.6 ± 3.4	0.43, NS
		4	61.2 ± 3.7	61.5 ± 3.2	-0.07, NS
		5	48.8 ± 1.9	52.1 ± 4.6	-0.66, NS
		6	60.6 ± 2.7	58.3 ± 3.5	0.53, NS
		7	63.0 ± 3.5	59.0 ± 3.2	0.84, NS
		8	65.5 ± 3.3	63.2 ± 2.6	0.56, NS
		9	50.9 ± 2.4	61.7 ± 3.2	-2.72, $p<0.01$
		10	63.1 ± 3.8	50.8 ± 2.2	2.74, $p<0.01$
Data are presented as means ± SEM.				

DISCUSSION

Similar to a recent study by *Conrady et al. (1989)*. who noted more psychiatric distress in women with postpoliomyelitis than in those with metastatic breast cancer, our postpoliomyelitis patients, when examined as a single group, had a high prevalence of mood disturbance, including somatic concerns and

feelings of irritability. Performance on cognitive tasks appeared to be normal, except for borderline impairment for nonverbal memory and speed of color naming.

However, when comparing patients with and without PPS, complaints of memory and concentration difficulties, as well as psychiatric histories, failed to differentiate the two patient groups. Additionally, no significant differences between the two groups were seen on any of the objective neuropsychological and neuropsychiatric measures. Despite the late onset of increasing weakness and pain, patients with PPS failed to have signiticantly higher scores of depression than those without PPS. In less clearly defined subgroups of postpoliomyelitis patients, *Conrady et al. (1989)* also reported no significant difference of psychiatric symptoms in those patients seeking assistance for physical disability and those in support groups presumed to be more physically stable.

Lane et al. (1974), however, previously reported late-onset mental changes in postpoliomyelitis patients. They found that 21 of 55 patients had a syndrome of fatigue often accompanied by headache, depression, and decreased ability to concentrate. Exertional dyspnea and abnormal sensitivity to cold were also noted in their patients. Fourteen of these 21 patients had mildly elevated P_aCO_2 levels, with associated reduction in vital capacity. The authors suggested that the emergence of this syndrome may be secondary to late-onset ventilatory insufficiency. Although their study was completed prior to the more recent criteria for

PPS, their patients may have had this disorder.

In the present study, patients with PPS had significantly higher P_aCO_2 levels, but did not have increased memory or concentration difficulties assessed either by interview or by objective neuropsychological procedures. In addition, when the entire sample was considered, P_aCO_2 levels were not in the clinically elevated range and no differences in P_aCO_2 levels between patients with and without complaints of decreased memory or concentration occurred. Thus, our study fails to confirm the findings of *Lane et al. (1974)*; however, more advanced degrees of ventilatory insufficiency in their patients may have contributed to these differences.

The cause of mood disturbances and cognitive complaints in postpoliomyelitis patients is unknown. Ventilatory changes *(Lane et al., 1974)*, sleep disturbances *(Fischer, 1987)*, and emotional reactions to disability *(Kohl, 1987)*, previously reported, need to be further evaluated. Additionally, these symptoms may relate to the pathological changes of the brain that are associated with poliomyelitis. With the acute infection, these pathological changes were associated with feelings of apprehension, anxiety, and fear, as well as symptoms of hypomania *(Noran, 1968)*. Residual psychic disturbances have also been observed *(Bodian, 1947)*. Acute histopathology *(Bodian, 1947, 1949; Matzke and Baker, 1951)* and viral immunofluorescence studies *(Torack and Morris, 1988)* have shown involvement of the thalamus, hypothalamus, and brainstem reticular formation -- structures that contain important elements of the limbic and reticular activating systems. In addition to the behavioral changes noted in acute poliomyelitis, other forms of pathology involving these areas have produced alterations of attention and concentration as well as psychiatric symptoms *(Trimble and Cummings, 1981; Cummings, 1988[sic]; Torack and Morris, 1988)*. Recently, *Pezeshkpour and Dalakas (1988)* examined the pathology of postpoliomyelitis patients with and without PPS. They reported the presence of active gliosis, inflammatory cells, and neuronal atrophy in the spinal cords of both groups of postpoliomyelitis patients, but suggested that an ongoing neuronal reaction continues for many years and may contribute to the late-onset weakness associated with PPS. It remains unclear, however, whether this inflammation is causative or reflects secondary effects of cell death associated with aging. Although only the spinal cords were evaluated in this study, the same slow inflammatory reaction may also be present in the diencephalon and nonmotor areas of the brainstem and might be expected to contribute to nonmotor symptoms. The high incidence of depression noted in the entire group of postpoliomyelitis patients might be attributable to brainstem changes sustained at the time of the original infection or to on-going cell loss. Depression has been reported previously in patients with lesions involving this region *(Cummings, 1986)*, and source nuclei for serotonin and norepinephrine, transmitters implicated in the mediation of depression *(van Praag, 1982)*, are located in the same area. The trend noted in this study for PPS patients to perform better than those without PPS on two procedures evaluating concentration and the ability to shift mental set may reflect less intense original injury in patients now beginning to develop PPS. A similar mechanism has been suggested to explain less intense, although new onset, weakness in PPS patients *(Klingman et al., 1988)*.

Our results suggest that PPS is not consistently associated with specific cognitive or personality disturbances. These findings suggest that either nonmotor structures in survivors of acute poliomyelitis were originally less severely involved, compensated more than motor areas, and/or have greater functional reserve, permitting relative sparing of nonmotor behavior with the onset of PPS. Delayed inflammatory changes, if associated with PPS, may essentially be restricted to lower motor neurons despite evidence for more widespread pathological involvement of the central nervous system noted acutely. It should be pointed out, however, that other explanations of these data exist. As half the fibers of a muscle need to be affected before clinical weakness occurs *(Klingman et al., 1988)*, the non-PPS patient group may have included patients with subclinical escalating weakness who are destined to develop PPS. Our patients were examined on only one occasion, so that progressive weakness and atrophy could not be documented.

Longitudinal studies are required to adequately address these issues.

The delayed onset of new symptoms in this large population of patients is an important medical problem, and systematic investigation of nonmotor involvement has clinical and pathogenetic implications.

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