Postpolio Syndrome: A Review of Lived Experiences of Patients

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Abstract

Postpolio syndrome (PPS) refers to a group of conditions that are present in patients, years after recovery from initial acute paralytic poliomyelitis. About 15%–80% of 20 million polio survivors worldwide will experience exacerbation of symptoms which typically appear 15–30 years after the resolution of initial poliomyelitis. Symptoms include new muscle weakness, fatigue, myalgia, joint pain, dysphagia, and difficulty breathing. Other reported symptoms include cold intolerance, sleep disorder, dysphonia, loss of stamina, musculoskeletal deformities, cardiovascular disorders, psychosocial problems, and restless legs syndrome. These symptoms are attributed to the superimposed neuronal loss of aging with inflammatory mechanisms, but without any convincing evidence of viral reactivation. Risk factors include female gender, respiratory symptoms, normal aging, permanent disability caused by motor neuron damage, muscle overuse and disuse, aging, and immunologic mechanisms. Hypothyroidism-induced myopathy and fibromyalgia are a differential diagnosis for PPS, and exclusion diagnosis is required as confirmatory criteria for PPS. The symptoms of PPS presented determine the course of management.

Keywords: *Fatigue, muscle weakness, myalgia, poliomyelitis, postpolio syndrome*

Introduction

Postpolio syndrome (PPS) is defined as a constellation of symptoms that polio survivors experience years after recovery from initial acute paralytic poliomyelitis.[1] Poliomyelitis is caused by poliovirus, which is transmitted by person-to-person and spreads mainly through fecal–oral route or, less frequently, by a common vehicle (e.g., contaminated water or food).[2] The virus multiplies in the intestine, from where it invades the nervous system and causes paralysis. The poliovirus causes death of the lower motor neuron (LMN) by damaging the cells of the anterior horn of the spinal cord, resulting in muscle weakness, fatigue, and myalgia.[3]

The term PPS was first introduced by Halstead in 1986, where he described the symptoms (muscle weakness, pain, and fatigue) experienced by polio survivors who had stabled functional life before being diagnosed for PPS. The first description of PPS was in 1875 in the French medical literature by Raymond and Charcot. In 1991, Halstead reviewed his criteria for the diagnosis of PPS, and these include new muscle weakness as a required criterion for diagnosis, with other symptoms such as cold intolerance, pain, and fatigue.[4]

The aim and objective of this review are to comprehensively highlight and discuss all the clinical manifestations, prevalence, risk factors, differential diagnosis, and management of PPS.

The exact cause of the development of PPS is unknown. However, studies have shown that active inflammatory process is present in the spinal cord with increased level of cytokines in the cerebrospinal fluid.[5]

Prevalence of Postpolio Syndrome

PPS is the most prevalent progressive motor neuron disease in the USA.[6,7] It was reported in 1987 that 640,000 of 1.6 million polio survivors in the USA presented with PPS.[8] It is estimated that about 15–20 million people are living with polio worldwide and about 20%–80% of them will develop PPS, with 2 million in North America, 700,000 residing in Europe, and 60,000 in France.[9,10] In a survey performed by Takemura *et al.*, a prevalence of 84.3% was recorded among polio survivors living in Kitakyushu, Japan.[11]

Risk Factors

Although polio is a contagious disease, PPS

How to cite this article: Oluwasanmi OJ, Mckenzie DA, Adewole IO, Aluka CO, Iyasse J, Olunu E, *et al.* Postpolio syndrome: A review of lived experiences of patients. Int J App Basic Med Res 2019;9:129-34.

Received: 01 October, 2018.
Accepted: 20 March, 2019.

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Access this article online

Website: www.ijabmr.org

DOI: 10.4103/ijabmr.IJABMR_333_18

Quick Response Code:

cannot be contracted from an affected person. The severity and degree of recovery of the symptoms caused by the initial paralytic poliomyelitis determine the risk of developing PPS. Patients with minimal symptoms are more likely to have mild PPS whereas those who are more acutely affected develop severe PPS symptoms.[12] Various studies have shown that the risk factors that contribute to PPS include female gender, the presence of a permanent disability and severe residual weakness experienced after polio, normal aging, muscle overuse and disuse, and immune reaction.[13]

The female gender and respiratory symptoms are the most important risk factors for developing PPS. The presence of respiratory symptoms in the initial acute poliomyelitis is a major risk factor predisposing to PPS later in life.[14] It occurs as a result of infection of the medulla by the poliovirus, also known as bulbar polio, a rare condition that involves the cranial nerves VII-XII and the muscles they supply, resulting in difficulty in breathing, swallowing, and speaking.[15] Reports have also shown that patients who experience infantile nonparalytic polio (poliovirus infection that does not involve the CNS) are also at risk of developing PPS.[16]

There are only a few studies that establish the genetic basis of developing PPS. A cytogenetic study reported the findings of significant breaks in chromosomes in patients with PPS, predominantly in chromosome 1p36.1.[17] Polymorphism of the poliovirus receptor (PVR) is reported in patients with polio and is regarded as a risk factor for developing acute poliomyelitis. PVR polymorphism is a possible risk factor for developing PPS. However, it is not yet confirmed to be present in patients with PPS.[18] Thus, further research is recommended to be performed to prove the presence of PVR polymorphism as well as to identify any form of gene mutation that can lead to the development of PPS.

Diagnostic Criteria

According to the European Federation of Neurological Societies taskforce on PPS, the universal criteria for diagnosing PPS include (a) confirmed history of LMN denervation using electromyography (EMG) and neurological examination; (b) confirmed partial- or near-complete recovery and functional stability for at least 15 years; (c) new or increased muscle fatigability with or without generalized muscle atrophy or muscle and joint pain; (d) progressive or sudden onset of muscle weakness which persists for at least 1 year; and (e) rule out of any other medical conditions that can cause the aforementioned symptoms.[19]

Clinical Manifestations

The presentations of PPS as reported by patients are as follows: neuromuscular disorders, dysphagia, dysphonia, cold intolerance, respiratory symptoms and sleep disorder, psychological symptoms, cardiovascular symptoms, and restless legs syndrome (RLS). These symptoms are summarized in Table 1.

Neuromuscular disorders

Neuromuscular manifestations are the core cause of symptoms experienced by the patients with PPS. These symptoms occur as a result of the loss of motor neurons in the spinal cord.[20] They include fatigue, muscle weakness, and myalgia.

Muscle weakness

In patients with PPS, it is observed that they have significant muscle weakness that is associated with aging. Reports showed that there is faster muscle deterioration in patients with PPS than normal individuals.[21] The major cause of muscle weakness is the overuse of muscles that compensate for the loss of muscle function in the affected parts of the body, leading to decline in muscle strength. Muscle weakness can also be attributed to disuse as a result of denervation of neurons.[22]

Fatigue

Fatigue is the most common and most disabling complaint of patients with PPS.[23] It is often accompanied by depression, with a prevalence ranging from 48% to 93%.[24] Patients reported that fatigue begins gradually and then progresses into a persistent decrease in endurance, tiredness, and lack of energy. Fatigue in PPS usually occurs daily and progressively increases in severity.[25]

Several theories explain the mechanism of fatigue in PPS, but the most widely accepted is the neural fatigue theory. This theory proposes that the enlargement of motor neuron fibers in patients with PPS causes additional metabolic stress on the cell body of neurons to supply nourishment to the newly added nerve fibers.[26] To compensate for the initial damage to neurons during infantile poliomyelitis, the surviving neurons grow new nerve terminals, resulting in enlargement of motor units. The integrity of these neurons is compromised after many years of use by the persistent metabolic stress, which eventually results in motor unit wasting, creating gaps in the neuromuscular junction, and this is regarded as the main cause of fatigue.[27]

Holmes *et al.* suggested the need to exclude several medical conditions such as infection, malignancy, autoimmune, endocrine, renal, and cardiopulmonary disorders before the diagnosis of fatigue secondary to PPS can be made. However, although the above conditions must certainly be evaluated, they seldom lead to important diagnostic information in the world of polio.[28]

Muscle and joint pain

The joint pain is caused by overuse and poor posture as a result of unbalanced muscle strength; this is called biomechanical pain. Myalgia is caused by muscle cramps, muscle twitching, and overuse, and this is called overuse

Table 1: Symptoms and management of postpolio syndrome

Symptoms	Description	Management
Muscle weakness	Overuse of compensatory muscles and denervation of neurons	Exercise and muscle training
Fatigue	Most common complaint, due to persistent metabolic stress on surviving neurons	Physical training, assistive technology in performing activities of daily living
Myalgia	Due to overuse pain	Pain relievers
Joint pain	Due to biomechanical pain	Analgesics
Dysphagia	Involvement of medulla and CN VII-XII	Review of nutritional habits and swallowing training
Dysphonia	Voice hoarseness associated with cold	Voice therapy
Cold intolerance	Inadequate muscle support for vasoconstriction	Biofeedback, relaxation, or visualization
Respiratory disorder	Respiratory muscles weakness and chest deformities	Inspiratory muscle training
Sleep disorder	Sequelae of respiratory dysfunction characterized by shortness of breath and nocturnal hypoventilation	Assisted ventilation
Psychological disorder	Depressive mood due to the inability to perform activities of daily living	Assessment and treatment to prevent progression to major depressive disorder
Cardiovascular problems	Discomfort on exertion that relives with rest	Medication to relieve specific symptoms
RLS	Irresistible urge to move legs accompanied by unpleasant sensations that worsen at night	Exercising affected muscles, dopamine agonists

CN: Cranial nerve; RLS: Restless legs syndrome

Table 2: Differential diagnosis of postpolio syndrome

Differential diagnosis	PPS	Hypothyroidism-induced myopathy	Fibromyalgia
Fatigue	Generalized or central fatigue	Easy fatigability	Generalized fatigue
Myopathy	Asymmetric	Symmetrical proximal muscle weakness	Generalized muscle weakness
RLS	Specific	Nonspecific	Specific
Nature of disorder	Neuromusculoskeletal disorder	Disorder of thyroid gland	CNS dysfunction
EMG	Fibrillation and fasciculation	Normal	Nonspecific
Hormone therapy	Nonspecific	Specific	Nonspecific

EMG: Electromyography; CNS: Central nervous system; PPS: Postpolio syndrome; RLS: Restless legs syndrome

pain.[29] The pain occurs mostly during the night and is triggered and exacerbated by excessive stress on the affected muscles and exposure to cold.[30]

Dysphagia

Dysphagia is a medical condition that is characterized by difficulty swallowing; this occurs when acute polio infection affects the medulla and the cranial nerves VII–XII (supplying bulbar muscles).[31] Affected muscles include muscles of swallowing and laryngeal muscles. Dysphagia begins to emerge decades after the initial poliomyelitis, but most people only notice mild difficulty in swallowing, while others who deny any difficulty in swallowing actually have the symptoms.[32,33]

The incidence of dysphagia in polio survivors has been estimated to be approximately 18%.[21] Difficulty swallowing could result in retaining of food remnant in the pharynx, posing a risk for aspiration. Nutritional problems and weight loss with tissue damage in the oral cavity occur due to poor nutrition from lack of eating. A careful review of nutrition habits should be undertaken, with patients being advised to chew their food, swallow twice for each bite, tilt their chin down when swallowing, and alternate between soft and hard food.[34]

Dysphonia

Another symptom seen due to the involvement of bulbar muscles is dysphonia. A patient with PPS reported developing long-lasting voice hoarseness that is associated with cold as well as dysphagia which resolved. Voice therapy is typically the treatment for dysphonia. In severe cases, tracheotomy can be used for treating dysphonia.[35]

Cold intolerance

Cold intolerance is the abnormal sensitivity to environments having a low temperature. Most patients with PPS report cold intolerance as a major symptom they experience, with a prevalence ranging from 29% to 56%.[36] It is due to inadequate muscle support for vasoconstriction of blood vessels required to reduce the amount of warm blood flowing to the skin and to conserve body heat. Inadequate muscle support is the consequence of muscle atrophy due to the damage of peripheral nerves caused by the poliovirus in the initial acute poliomyelitis. Techniques such as biofeedback, relaxation, or visualization, taught by psychotherapists and psychologists, can help increase the warm blood in PPS.[37]

Respiratory symptoms and sleep disorder

Respiratory symptoms occur as a result of musculoskeletal deformities such as scoliosis, kyphosis, or degenerative

change in the spinal vertebrae, all of which occur in acute poliomyelitis. Patients with PPS with thoracic cage deformity and weakness of muscles of respiration are at risk of developing respiratory insufficiency, which can lead to sleeping disorders.[38]

A study carried out by Steljes *et al*., where polysomnography was performed on 13 patients with PPS, reported that five patients already required ventilator assistance and these patients demonstrated decreased quality and duration of sleep.[38] To avoid respiratory deterioration during sleep, mechanical ventilation is important to support respiratory muscles. It is also reported that smoking and obesity exacerbate the respiratory insufficiency. Thus, patients are advised to stop smoking and reduce weight.[39]

Psychological symptoms

Most patients with PPS find coping and adapting to new burdensome symptoms, thus resulting in depression. A case study by Esteban J. in 2013 revealed a 57-year-old male polio survivor who reported having to retire early from his job because he gets fatigue easily.[40] The symptoms can lead to deterioration in the day-to-day functioning of patients and affect their quality of life. Most patients report being frustrated because of reduced functionality in activities of daily living, and this ultimately leads to depressive symptoms in such patients.[41] Thorough and regular psychological examination should be performed on individuals suffering from PPS, as this can improve mood and prevent the progression of depressive symptoms to major depressive disorder.

Cardiovascular symptoms

The cardiovascular symptoms reported in patients with PPS include palpitations or irregular heartbeats; chest discomfort which patients describe as pain, pressure, squeezing, and heaviness especially on exertion but relive with rest; shortness of breath with minimal exertion or upon lying down; severe dizziness or loss of consciousness; sudden weakness or paralysis of one part of the body, sudden slurring of speech or loss of vision, and frequent nocturnal urination; unusual and progressive fatigue; and claudication or leg pain discomfort with walking.[42]

Restless legs syndrome

RLS is a neurologic condition that causes an overwhelming, irresistible urge to move the legs, often accompanied by an unpleasant feeling in the legs which improves with moving the legs. Luft and Muller (1947) reported RLS in patients with poliomyelitis. The condition causes an uncomfortable, itchy, pins and needles or creepy crawly feeling in the legs. The sensations are usually worse at night, especially when lying or sitting.[43]

Perdrup (1950) described the case of a patient with PPS who experienced attacks of restless legs accompanied by ulcers. The patient reported that the pains worsen with warm condition and get better with cold. The intensity was so severe that the patient was asked to have the leg amputated. RLS is treated using dopamine agonists.[44]

Differential Diagnosis

Differential diagnosis for PPS includes hypothyroidism-induced myopathy and fibromyalgia. The differential diagnosis of PPS is summarized in Table 2.

Hypothyroidism-induced myopathy

Hypothyroidism is a disorder of the thyroid gland which results in the decrease in the production of thyroid hormone. Symptoms include cold intolerance, decreased sweating, weight gain, easy fatigability, difficulty breathing on exertion, and depression.[45] Neuromuscular signs and symptoms are complications of hypothyroidism. These include sensory signs of sensorimotor ataxic neuropathy and myopathy.[46] Hypothyroidism-induced myopathy can be confused with PPS because its manifestations are similar. Hence, the diagnosis of exclusion is necessary for PPS to be confirmed.

Symmetrical proximal muscle weakness is observed in hypothyroidism with raised serum creatine kinase, which is not associated with the degree of myopathy. Increased serum creatine kinase is also observed in PPS, which occurs as a result of muscle damage and death. Severe muscle weakness is observed in any muscle affected during infantile acute polio. Thus, there is asymmetric muscle weakness in patients with PPS. Patients with hypothyroidism-induced weakness respond to treatment with hormone therapy while muscle weakness persists in patients with PPS. EMG of patients with hypothyroidism-induced myopathy appears normal or with mild neuromuscular pathology in few cases. However, EMG of patients with PPS shows fibrillation and fasciculations.[47]

Fibromyalgia

Fibromyalgia is an idiopathic, chronic condition that causes generalized pain due to the dysfunction in the processing of pain signals in the CNS. Patients with fibromyalgia present with symptoms that mimic PPS which include new muscle weakness and myalgia, generalized fatigue, RLS, difficulty sleeping, and psychological disorder.[48]

It is reported that about 2%–8% of the general population is affected by fibromyalgia. Like PPS, females are significantly more affected with fibromyalgia than males. Patients with PPS have been reported to present with fibromyalgia. Thus, it is imperative to identify distinguishing features of both conditions to make an accurate diagnosis.[49]

The primary problem in fibromyalgia is a CNS dysfunction which results in excessive pain transmission and defective pain interpretation. In other words, patients experience amplified sensitivity to nonpainful stimulation. Due to the involvement of the CNS, patients with fibromyalgia develop cognitive deficits such as memory problems (short and long term) and difficulty in performing multiple tasks.[50]

Management of Postpolio Syndrome

The management of PPS involves identifying the signs and symptoms to determine the best treatment available. Muscle weakness and fatigue are one of the major problems experienced by patients with postpolio; management includes physical training for neuromusculoskeletal disorders, preventing disuse and overuse, and getting rest. Pharmacological drugs are also used in managing PPS where a single high dose of immunoglobulin is given to reduced proinflammatory cytokines present in the cerebrospinal fluid of the spinal cord.[51] Other medications are also provided to help pain and sleeping patterns.

Weakness in laryngeal and esophageal muscles resulting in dysphagia and dysphonia can be treated with swallowing training and voice therapy. In the case of hypoventilation, assisted ventilation would be required along with inspiratory muscle training to increase endurance. Lifestyle changes are also important to help in PPS management where persons can use assistive devices when necessary, enroll in weight-controlled programs, group therapy, and occupational interventions.[52] Management of PPS is summarized in Table 1.

Authors' Opinion

Although the administration of polio vaccine worldwide has effectively reduced the incidence of poliomyelitis, there are still millions of polio survivors who will still experience PPS. The significance of nonparalytic polio in the development of PPS should always be considered when patients presenting with PPS symptoms with no history of paralysis are encountered. Further genetic and clinical studies should be performed to analyze the relationship between PPS and fibromyalgia and their connection with increased incidence in females compared to males.

Conclusion

PPS is a sequela of acute poliomyelitis which occurs decades after resolution of the initial paralytic or nonparalytic polio. Fatigue, muscle weakness, myalgia, and joint pain are the major symptoms of PPS. Diagnosis of PPS is made based on the exclusion of other conditions that can cause similar symptoms. The quality of life of patients with PPS is significantly affected by the reoccurrence of the constellations of symptoms they experience. Proper assessment and treatment should be performed to prevent severe impairment of function in patients.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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