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Case Report

Post-Polio Syndrome: case series and review of the literature

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ABSTRACT

Post-Polio Syndrome (PPS) is a late-onset condition affecting polio survivors, typically decades after the initial infection. PPS is a diagnosis of exclusion, confirmed by clinical history, electromyography (EMG), and the absence of other neurological disorders. Key features include asymmetrical, flaccid paresis without sensory loss, fatigue, muscle atrophy, and pain. PPS involves chronic inflammation and a cycle of denervation and re-innervation in motor neurons, leading to eventual failure of compensatory mechanisms. There is no specific cure. We present three case studies of male patients from South Africa, Mozambique, and Kenya, all of whom experienced a return or worsening of neuromuscular symptoms years after recovering well from childhood polio.

INTRODUCTION

Post-polio syndrome (PPS) is a condition characterized by muscle weakness that appears more than 30 to 40 years after the initial infection with the virus, and tends to progress slowly with periods of stability.(1) The incidence of paralytic polio worldwide has been reduced a 1000-fold with the introduction of the polio vaccine in the mid-1950s and early 1960s. Two of the three naturally occurring (wild type) poliovirus serotypes no longer circulate, and wild type poliovirus (WPV1) disease is now confined to only two countries, Pakistan and Afghanistan. The Independent Africa Region Certification Commission for polio eradication officially declared in August 2022 that the Africa Region was free of wild poliovirus.(2) However, PPS is likely to persist for the next few decades because of the late presentation of the illness. In a study carried out in the US in 1992, the prevalence of PPS was estimated to be around 30% of all paralytic cases of polio, and the risk was higher in patients who sustained substantial permanent impairments after polio.(3)

There are no reports of the prevalence of PPS in developing countries. We describe 3 cases of patients with PPS and review the literature of this extremely rare disorder in terms of its pathophysiology, clinical presentation, diagnosis, and treatment.

CASE I

Mr BM, aged 101 years from Johannesburg, South Africa, was diagnosed at the age of 3 months with infantile poliomyelitis. To our knowledge, he is the oldest survivor of polio in the world. At the age of 75, he presented with weakness of both lower limbs, more marked on the right, and was unable to walk. Initially, this was thought to be due to spinal stenosis, but with neurophysiological investigations, he was diagnosed with PPS.

Clinically, he had weakness in both lower limbs. On the right, this was global and graded at 0/5 (MRC grading) with no power, complete paralysis, and a noticeable foot drop. There was proximal hip flexion and extension weakness, with knee extension and flexion all graded at 0/5. Sensation was normal to all modalities, and reflexes were absent. Coarse fasciculations were present. In the left lower limb, distal weakness was present and graded at 3 to 4 out of 5. In the lower limbs, there was wasting of the tibialis anterior and quadriceps muscles.

Physical examination of the upper limbs revealed wasting of the interossei muscles of both hands but minimal wasting of the forearms, biceps, and triceps muscles. He also had distal and mild proximal weakness in both upper limbs graded at 4/5. His scalene muscles in the neck were marginally wasted, but he had reasonable head control. Nerve conduction tests showed a marked reduction in amplitude with ranges of 0.5 to 1.0 millivolts in the right lower limb.

MRI scan of the spine showed a very marked scoliosis with denervation atrophy of the lower limb muscles. CSF analysis showed a mildly elevated protein of 0.5g/L and was acellular. As PPS is a diagnosis of exclusion, a diagnosis of PPS was reaffirmed. He was given an empiric trial of treatment with intravenous solumedrol (1 g daily

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for 5 days) with minimal benefit. Although still alive with normal higher function, his condition continues to worsen, and he is now bedridden.

CASE 2

Mr JN, aged 69 years from Maputo, Mozambique, presented with weakness in his right lower limb and also with what appeared to be a foot drop. In 1994, he was diagnosed with spinal stenosis, and he underwent treatment for this with discectomy, laminectomy, and fusion.

On further questioning, it became clear that he had developed polio as a child. This was common at that time, and he had forgotten about the polio as he had recovered quite well. He had minimal involvement from the polio during his youth and was able to ride a motorbike, travel extensively, and completely ambulate. On further questioning, he acknowledged a slight shortening of the right leg and needed a heel orthotic attachment to balance that.

Clinically, his main problem was pain in his lumbar spine. This was associated with weakness in both lower limbs. He had no sensory symptoms. His bladder and bowel control were completely normal. Upper limbs were normal. In terms of power, he had 0/5 dorsiflexion weakness in his right lower limb at the ankle, with normal plantar flexion. Eversion and inversion were 0/5. He had 4/5 of hip extension weakness and also had 3/5 knee extension weakness. His hip flexion was 4/5. In the left leg, power was normal. In the upper limbs, he has wasting of the interossei of the muscles. Sensory testing was utterly normal. Coarse fasciculations were noted in the quadriceps and gastrocnemius muscles of the right lower limb. MRI study of the spine showed evidence of fusion and degeneration of discs from L1-L5. No specific spinal cord compression was noted. Lumbar puncture was normal. He was then diagnosed with PPS and treated with IV solumedrol with a reasonable response.

CASE 3

Mr KS, aged 77 years from Nairobi, Kenya, presented with a history that in 1955 he suffered polio infection affecting the right leg. His main complaint was back pain and progressive weakness of both lower limbs. He subsequently sought medical opinions in several countries, but in summary, he was considered to be suffering from either PPS or spinal stenosis.

Clinically, he had marked quadriceps wasting with weakness and fasciculations in both lower limbs. The right lower limb had all the stigmata of the polio infection with weakness, wasting, and a dropped clawed foot. The upper limb examination was normal. Neck extension and flexion were normal. He had deformed pes cavus feet bilaterally, worse in the leg affected by the original polio infection. Sensory examination throughout for all modalities was normal. Nerve conduction studies showed markedly reduced amplitudes in the lower left limb. On the right, these were not elicitable. Review of his MRI scans showed extensive degenerative disease, but with minimal root entrapment. He was therefore diagnosed with post-polio syndrome. He opted not to try any treatments at present.

DISCUSSION

Poliomyelitis affects the anterior horn cells of the spinal cord. Post-polio syndrome is a lower motor neuron disorder. The dorsal roots are not affected. The pathophysiology of PPS involves an ongoing denervation and re-innervation process. After initial infection by the virus, there is a loss of motor neurons. Over time, there is a compensatory increase of up to twenty times in the adjacent motor units of the spinal cord. Eventually, further re-innervation is not possible, and there is subsequent denervation with loss of muscle fibers, atrophy, and loss of muscle strength.(1)

In PPS, the spinal cord demonstrates active chronic inflammation with perivascular lymphoplasmacytic infiltrate in the anterior horns and leptomeninges. There is no cerebral involvement. However, postmortem studies have shown involvement of cerebral structures being affected by poliovirus, including the posterior hypothalamus, thalamus, putamen, caudate, and locus coeruleus. It is postulated that this may account for fatigue and attention deficit disorder of PPS.(4)

PPS patients usually present with new onset weakness, muscle atrophy, myalgia fasciculations and pain in the muscles initially affected by the virus. Other symptoms may include extreme fatigue, dysarthria, and cold intolerance. On examination, the most typical is asymmetrical flaccid paresis with usually no sensory deficit.(5)

PPS is a clinical diagnosis. It is a diagnosis of exclusion of other causes of similar symptoms and signs. A key clinical feature is that the muscle weakness and /or fatigability must have been persistent for at least 1 year. The serum creatinine phosphokinase level is not elevated, and the diagnosis is supported by electrophysiological examination, including electromyography (EMG).(1)

Treatment of PPS involves a multidisciplinary approach, including pharmacological and rehabilitation interventions. High-dose intravenous immunoglobulins significantly reduced pro-inflammatory cytokines in the CSF of patients with PPS, with amelioration of symptoms and signs.(6). Lamotrigine has proven effective in controlling pain, fatigue, and improving the quality of life. Orthoses and assistive devices have improved mobility, reduced pain, and increased functional activity.(6) In this case series, the response to high-dose solumedrol was variable.

The three patients described presented typically with pure motor weakness and a syndrome characterized by progressive weakness with wasting of the muscles and fatiguability. There was an established history of polio in childhood, and all other causes of muscle weakness were excluded. The case reports also highlight that limbs with sub-clinical involvement may develop new muscle

weakness and neuromuscular symptoms later in life. These findings suggest that the muscles affected by the polio virus are vulnerable to later functional impairment, regardless of whether or not they achieved complete recovery after the acute stage of poliomyelitis. According to the literature, risk factors for PPS are female sex and a severe course of initial infection.(3) This was not the case in our patients. They were males who had made a good recovery after initial muscle paralysis in childhood.

CONCLUSION

This case series demonstrates the clinical presentation and pathophysiology of PPS in three patients from Africa seen at our hospital. They were all male patients who initially had mild disease. The diagnosis of PPS was made by exclusion of all other potential aetiologies. Treatment requires a multidisciplinary approach as there is no specific treatment for this condition.

CONSENT

Informed consent was obtained from the patient

AUTHOR CONTRIBUTION STATEMENTS

Modi G: Responsible for drafting, writing, reviewing, revising, and final approval for submission

Schnaid E: Contributed to drafting, writing, and revision of the manuscript.

Ebrahim O: Contributed to writing, review, and revision of the manuscript.

Ebrahim HA: Contributed to the revision of the abstract and references and approval for submission.

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