

Infectious Disease Eradication: Poliomyelitis as a Lesson in Why “Close” is Not Good Enough

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The current polio epidemic in Indonesia is a reminder that infectious agents know no political or race boundaries. Like most of the world, this country was polio-free and prior to this outbreak, there were only 6 countries with endemic polio: Nigeria, India, Pakistan, Afghanistan, Niger and Egypt.¹ The outbreak in Indonesia began in March 2005 and to date, in excess of 225 children have been paralysed due to imported poliovirus.² The infections were initially in 2 provinces on Java but have spread to involve Jakarta, as well as Sumatra and Central Java.

The earliest recorded description of poliomyelitis is the funeral stele of a 19th Egyptian dynasty priest showing Ruma with a wasted and crippled leg.³ Charcot and Joffroy are credited with identifying the spinal anterior horn cell as the site of damage, but it was Jacob von Heine who recognised poliomyelitis as a disease entity and termed it infantile spinal paralysis.⁴

The poliovirus is an enterovirus with 3 antigenic serotypes. Poliovirus 1 has been implicated in nearly all epidemics of paralytic poliomyelitis in the United States.⁵ Transmission is usually fecal-oral, but droplet transmission from sneezing or coughing can occur. The virus then replicates in the oropharyngeal or intestinal mucosa before reaching the submucosal lymphoid tissue via microfold cells⁶ and the blood stream causing a primary viraemia. After this phase, a secondary viraemia occurs which may result in central nervous system (CNS) involvement.

Poliovirus has an incubation period of 1 to 2 weeks. The initial pre-CNS phase of 1 to 3 days consists of gastrointestinal complaints and systemic manifestations including fever, headache, malaise and sore throat. The subsequent spectrum of CNS disease includes aseptic meningitis or non-paralytic polio, polio-encephalitis, bulbar polio and paralytic poliomyelitis. Combinations of these conditions can occur and aseptic meningitis is common prior to the onset of paralytic poliomyelitis. Paralysis is preceded by myalgia, cramps, fasciculations, and radicular pain which occur about 1 to 2 days before weakness onset.

There are 3 clinical subtypes of paralytic poliomyelitis – spinal, bulbar, and bulbospinal – of which the spinal form is most common.⁷ The infection tends to affect the lumbar

more frequently than the cervical region. Paralysis is typically flaccid, asymmetric and patchy. Weakness involves the proximal musculature more than distal muscles, and as the disease progresses, the tendon reflexes are lost. Bulbar poliomyelitis from brainstem involvement can also occur in up to 15% of cases.

Routine laboratory tests are usually non-specific. The total white count may be elevated and cerebrospinal fluid (CSF) obtained from lumbar puncture usually shows increased cells with a normal or mildly elevated protein level. The CSF cell picture is initially a polymorphonuclear leukocytosis shifting to mononuclear cells with cell counts in the hundreds. Magnetic resonance imaging can demonstrate inflammation to the spinal cord anterior horns.⁸ Patients who developed paralysis tended to have higher cell counts in the CSF and a higher protein concentration than patients who did not.⁹

Virus isolation and serologic testing are necessary for diagnosis. Poliovirus can be isolated from the oropharynx for several weeks and from the stool for several months, but is almost never found in the CSF. A 4-fold or greater rise in serum antibody titre between the acute and convalescent samples is diagnostic. The CSF IgM antipoliovirus response¹⁰ as well as CSF to serum antibody ratio may also be diagnostic.¹¹ More recently, polymerase chain reaction (PCR) is also available for detection of poliovirus infections.¹²

There is no specific treatment for poliomyelitis. Management hinges on good supportive care, followed by passive, then active physiotherapy and possibly orthopaedic attention. During the preparalytic stage, bed rest should be enforced as physical exercise can worsen the severity of paralysis.¹³

Mortality of 5% to 10% of paralysed patients results from respiratory and cardiovascular compromise secondary to bulbar involvement.¹⁴ Patients who survive acute paralytic poliomyelitis usually have significant recovery of motor function, although permanent and severe paralysis of 1 or 2 limbs are often the sequelae. Most of the recovery will occur within the first 6 months.

Acute complications of immobility include urinary tract

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and respiratory infections, contractures and pressure ulcers. In the medium and longer term, other concerns include osteoporosis and skeletal deformities including scoliosis and reduced limb development especially in children. The post-polio syndrome is a long-term complication of progressive muscular atrophy that can occur decades after the acute episode.¹⁵ Long-term mortality amongst poliomyelitis survivors is also increased, especially amongst those who contracted severe paralytic poliomyelitis at a young age.¹⁶

Since the advent of initially the inactivated (killed) poliovirus vaccine by Salk¹⁷ and then the live attenuated oral vaccine by Sabin,¹⁸ the worldwide incidence of paralytic polio has fallen, especially where vaccination is available. Poliomyelitis is a notifiable disease in Singapore and all cases should be referred to the Communicable Disease Centre at Tan Tock Seng Hospital for isolation and management.¹⁹ In Singapore, the childhood immunisation programme commenced in the early 1960s and close to 95% of children are now immunised. There has been a corresponding precipitous fall in the incidence of poliomyelitis.

In an assault on global poliomyelitis in 1974, the Expanded Programme for Immunisation (EPI) was established by the World Health Organization (WHO). During the 1980s, global elimination of polio became a realistic aspect of the world health agenda. During 1988, there were 350,000 recorded cases of polio, but this year (up to 6 September 2005), just 1163 cases have been identified worldwide. India is the only Asian country with endemic polio but has seen an amazing reduction in cases from 25,253 in 1988 to 225 cases in 2003. Other countries in the region have been polio-free for at least 3 years. Strategies have included routine childhood vaccination, National Immunization Days (NIDs), upgrading of laboratory skills and surveillance efforts, as well as rapid response to epidemics such as that currently being undertaken in Indonesia. Regrettably, like Indonesia, 9 other countries in the last 12 months have been re-infected – Somalia, Yemen, Angola, Ethiopia, Chad, Sudan, Mali, Eritrea and Cameroon. As per WHO recommendations, NIDs are being undertaken now in Indonesia's affected areas as part of an appropriate rapid response.

Quite simply, global polio eradication requires a period where no one in the world has the disease. Until that time, for any country, the only means of protection is a high level of vaccination. Before a country can safely wind back vaccination efforts, there must be no endemic disease as well as no threat of imported disease. Infectious diseases remind us that as a world we are one. To assist one's neighbour is to assist one's self with "upstream efforts" in infection control essential in any such national public health goals.

Global eradication of polio remains an achievable goal but it requires vigilance which is unrelenting until that point. Public health authorities must behave as one global community and not accept that "near enough is good enough". Until the declaration of eradication is made, each region is vulnerable and must maintain vaccine policy and surveillance irrespective of local incidence.

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