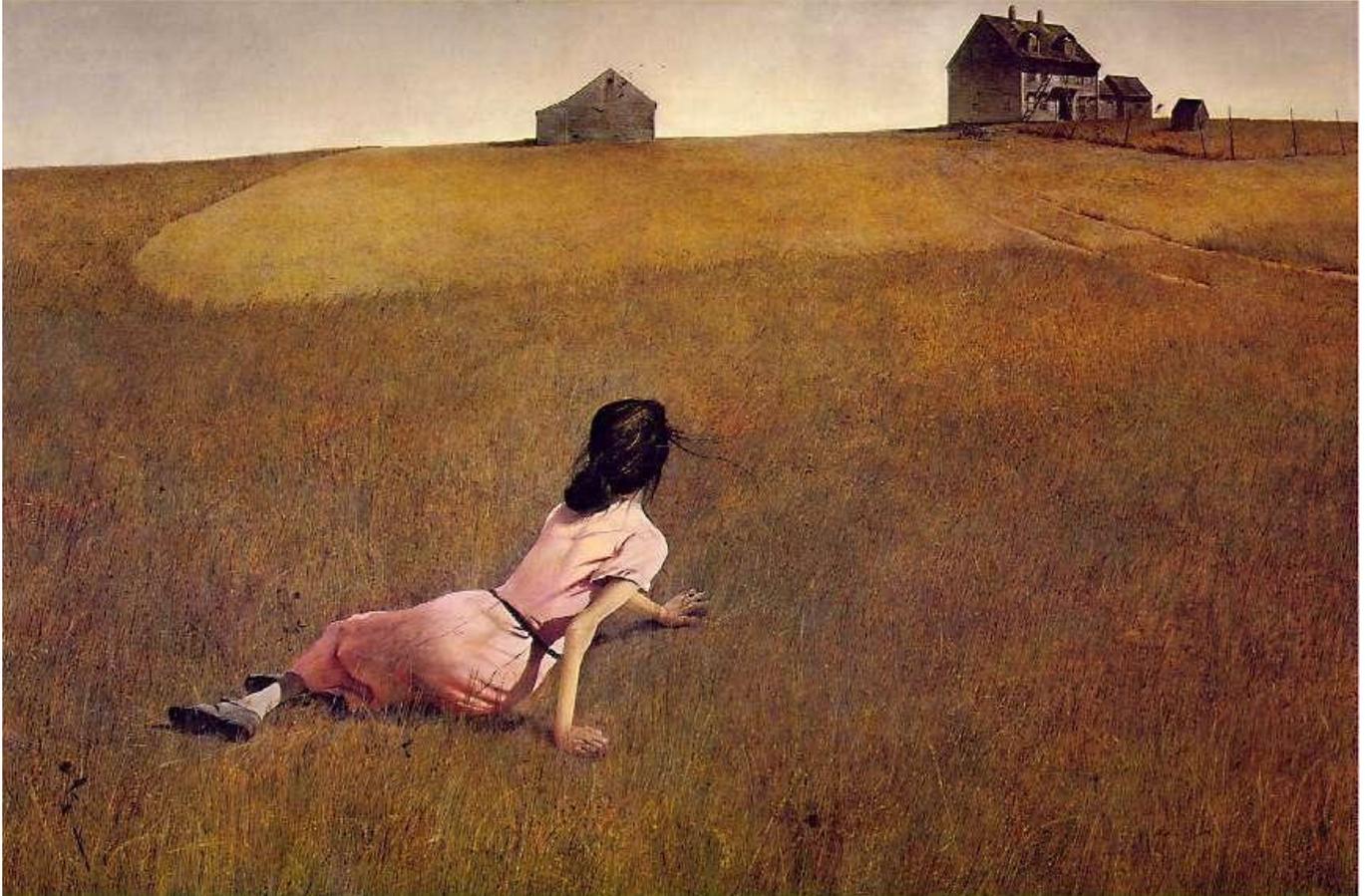


POLIOMYELITIS



“Christina’s World” tempera on gessoed panel, Andrew Wyeth, 1948, Museum of Modern Art, New York City.

Andrew Wyeth’s “Christina’s World”, was one of the late 1940s most iconic popular images. On a hot summer afternoon in 1948, the artist happened to glance from his open window to be greeted by the sight of his neighbor, a woman who had been struck down at an early age by poliomyelitis and who had lost partial use of her legs, sitting in the corn field. She had been out in sun when she had collapsed from exhaustion. He watched her crawling inch by inch, pushing herself with her thin arms, across a vast field, stopping now again to catch her breath and look upwards to gauge her progress to the distant farmhouse where she lived.

Wyeth became fascinated by the sheer endurance and determination of the courageous woman, never once flagging or calling out for help to anyone. Later on he used his wife, Betsy, as a model to recreate the scene in his painting. The image became an instant hit in

the popular culture of post-war America. For many it became a poignant symbol of the struggle of their own lives during the recently ended Second World War. It became an icon for the determination of Americans, and indeed many others around the world to continue the long and difficult fight of rebuilding their own worlds shattered by the global conflict. The woman who inspired Wyeth's work was Anna Christina Olson (1893-1968). She was a lifelong resident of the Cushing, Maine farm pictured in "Christina's World".

The fight to finally eradicate the devastating disease of childhood, poliomyelitis, remains one of humanity's most noble endeavors. Wyeth's image will forever remain a poignant reminder of its devastating consequences.



"Puddle", woodcut, M.C Escher, 1952.

"In childhood a useless leg does not bring with it a sense of shame; it is only when one learns to interpret the glance of people unable to hide their feelings that one experiences a desire to avoid them".

Alan Marshall, "I can Jump Puddles", 1955.

POLIOMYELITIS

Introduction

Poliomyelitis appeared in the early Nineteenth century (though it probably existed before this time) and continued to increasingly ravage humanity until the 1950s, when it was one of the most feared infectious diseases.

A killed injectable polio vaccine was developed in 1955 by Jonas Salk and in 1962 an oral attenuated polio vaccine was developed by Albert Sabin. Following these effective vaccines there was a dramatic decrease in poliomyelitis cases. Vaccination now brings the hope of eventual global eradication of this disease.

It is very rarely encountered in Australia today, nonetheless sporadic cases have been documented in high risk groups which include overseas immigrants from regions where the disease has not been fully eradicated and where immunization of the population is incomplete or non-existent.

Epidemiology

Prior to vaccination programs polio occurred worldwide.

Since the Global Polio Eradication Initiative was launched in 1988, three WHO regions have been certified polio-free:

- The Americas, 1994.
- Western Pacific (including Australia), 2000
- Europe, 2002.

Polio cases have dropped from an estimated 350,000 cases in 125 countries in 1988 to just 480 reported cases in only ten polio-endemic countries in 2001.

In endemic areas, cases of polio still occur both sporadically and in epidemics.

In countries where polio has been eradicated, importation from non-vaccinated individuals remains a threat.

Pathology

Organism

- Poliovirus, an RNA enterovirus of the picornavirus family
- Serotypes 1, 2 and 3 are responsible for clinical disease.

Pathophysiology

Acute paralytic poliomyelitis is a disease of the **anterior horn motor neurons** of the **spinal cord and brain stem** caused by the poliovirus.

Reservoir

- Humans.

Mode of transmission

- Poliomyelitis virus is highly infectious. The infection rate in households with susceptible young children can reach 100%.
- Wild poliovirus is spread primarily through faecal-oral spread and is an important consideration where sanitation is poor.
- Less commonly it can be spread via saliva.
- Live oral polio vaccine (OPV) virus can be shed in the faeces for six weeks and may lead to infection in unvaccinated contacts. Unvaccinated household contacts of a case should therefore be vaccinated at the same time. Stressing the importance of hand washing for parents following nappy changing and disposal is important.

Incubation period

- The incubation period ranges from three to 35 days
- It is most commonly seven to 14 days for paralytic cases.

Period of Communicability

- The risk of transmission of infection is greatest for the seven to ten days prior to and following the onset of symptoms.
- The virus persists in the pharynx for approximately one week and in the faeces for up to six weeks but these times may be longer in immunosuppressed patients.
- Transmission of the virus is possible for as long as the virus is being excreted.

Susceptibility and Resistance

- Any non-immune person is susceptible to infection.
- After infection from both clinically recognizable and inapparent infections, *type specific* lifelong immunity occurs. Reinfection is rare but can occur if infected with poliovirus of a *different type*.

- Vaccine efficacy of OPV and Inactivated Polio Vaccine (IPV) after a primary course is 95% and thought to be lifelong. Both vaccines give protection against all three types of poliovirus.
- Infants born of immune mothers have transient passive immunity.

Clinical features



Child showing the long-term effects of spinal polio, (WHO Website October 2008)

Polio remains a predominantly childhood illness with 80% to 90% of cases occurring in children less than five years old.

It is an acute illness following gastrointestinal infection by one of the 3 types of poliovirus.

The majority of polio infections are in fact either subclinical or present as a non-specific febrile illness.

Flaccid paralysis fortunately occurs in less than 1% of poliovirus infections.

High Risk Groups:

Polio cases in Australia are very rare unless the patient belongs to a high risk group.

High risk groups include:

- Those who have not been or have been incompletely vaccinated.
- Immigrants from regions where polio has not been eradicated.
- Travellers to regions, where endemic polio still exists.

Non-paralytic poliomyelitis:

Symptoms of minor illness include self-limiting non-specific constitutional symptoms only, such as fever, lethargy, malaise, headache, nausea, anorexia and vomiting.

The illness may manifest as an URTI, gastroenteritis or simply a non-specific viral-type picture. These syndromes therefore are clinically indistinguishable from other viral illnesses.

Occasionally a non-paralytic “aseptic” type meningitis may occur.

Paralytic poliomyelitis:

If the disease progresses to major paralytic illness, the hallmark features will include:

1. Severe muscle pain.
2. Stiffness of the neck and back.
3. Development of flaccid paralysis.

Characteristics of the major illness paralysis include:

- Fever and muscle pain are generally present at onset.
- Polio paralysis characteristically has an **asymmetric** distribution, which affects some muscle groups while sparing others.
- The maximum extent of paralysis usually reached within three to four days.
- Progression of paralysis almost tends to halt when the patient becomes afebrile.
- The site of paralysis depends upon the location of nerve cell destruction in the spinal cord or brain stem.
 - ♥ Proximal muscles of the extremities tend to be more involved than distal.
 - ♥ The legs are more often affected than the arms.

- ♥ Paralysis of the respiratory and swallowing muscles is life threatening.
 - After 60 days the degree of existing paralysis is likely to be permanent.
 - Sensory loss is very **rare** and its occurrence should strongly suggest some other diagnosis such as Guillain-Barre syndrome.
4. Many persons with paralytic poliomyelitis recover completely and, in most, muscle function returns to some degree.
- Weakness or paralysis still present 12 months after onset however is usually permanent.

Paralytic poliomyelitis has been classified into 3 types, based on the region of the spinal cord predominantly affected:

1. Spinal polio:
 - This is the most common type (79 %) ² It is characterized by asymmetric paralysis that most often involves the legs.
2. Bulbar polio:
 - This leads to weakness of muscles innervated by cranial nerves and is uncommon, (2 %)
3. Bulbospinal polio:
 - This is a combination of bulbar and spinal paralysis, and is the second most common form, (19 %).

Post-polio syndrome

Post-polio syndrome is an infrequent recurrence of muscle weakness that may occur many years after initial infection.

It is thought to be due to progressive dysfunction and loss of motor neurons that compensated for the neurons lost during the original infection, not to persistent or reactivated poliovirus infection.

Vaccine-associated paralytic poliomyelitis

Vaccine-associated paralytic poliomyelitis (VAPP) is a very rare complication in recipients of oral polio vaccine or their contacts, with approximately one case per 2.4 million doses of vaccine.

The risk is greater for the first dose than subsequent doses and is slightly greater for adults than children.

Investigations

Viral Isolation:

Diagnosis can be made by isolation of the virus from:

1. Faecal specimens.
 - Two separate faecal specimens taken at least 24 hours apart and within 14 days of onset of symptoms give the best chance of diagnosis.
2. Oropharyngeal secretions.
3. Cerebrospinal fluid (CSF).
 - CSF usually reveals a mild elevation in protein and a lymphocytosis.

The Health Department requires that all suspected cases of polio have appropriate faecal specimens sent for analysis by the National Poliovirus Reference Laboratory (NPRL), managed by the Victorian Infectious Diseases Reference Laboratory. The NPRL can also differentiate between “wild-type” and vaccine-associated strains.

The Department coordinates with clinicians and the NPRL to ensure that appropriate infection control procedures are followed in the collection, transfer and analysis of all clinical specimens from patients with suspected polio.

PCR:

Polymerase chain reaction testing is also available and is routinely used to differentiate wild-type strains from vaccine strains.

Electromyography:

This may be used to confirm early nerve dysfunction.

MRI:

MRI may show increased signal density lesions within the anterior regions of the spinal cord consistent with anterior horn cell motor neuron damage.

MRI may be performed for acute neurological findings in the limbs, before a diagnosis of polio is even suspected.

In the great polio epidemics of the Nineteenth and early Twentieth century this modality of course was not available. Further, in those third world regions where polio still exists today MRI is not available.

The sensitivity and specificity of MRI in the diagnosis of polio therefore remains largely unknown, but the modality remains an important first step in investigation, for both ruling out possible *alternative diagnoses* such as space occupying spinal cord lesions and as a *possible* indicator of the disease, before the definitive results of PCR tests become available.

Management

Once paralytic polio develops, treatment is supportive.

Physiotherapy will form an important part of ongoing management.

Vaccination:

Universal vaccination in early childhood is the most effective means of preventing and eradicating poliomyelitis.

Catch-up immunization is also recommended for unimmunized or partially immunized adults at risk of exposure such as those travelling overseas and health care workers in possible contact with polio cases.

Immunization can be given as an *intramuscular inactivated polio vaccine (IPV)*, or as an *oral live attenuated polio vaccine (OPV)*.

Under the National Immunization Program, polio immunization consists of a primary course of OPV given as two drops by mouth at 2, 4 and 6 months of age with a booster at four years of age.

IPV is given for individuals with immunosuppression from disease or chemotherapy and for their siblings and household contacts.

See latest edition of Australian Immunization Handbook for full prescribing details.

Notification:

Poliomyelitis (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion:

School exclusion is applicable for at least 14 days from onset. Return after receiving a medical certificate of recovery.

References

1. Poliomyelitis, The Bluebook Website.
2. Australian Immunization Handbook 9th ed 2008
3. The Pink Book, Vaccination Handbook, USA.

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