PRIMEVIEW

VARICELLA ZOSTER VIRUS INFECTION

nature REVIEWS DISEASE PRIMERS

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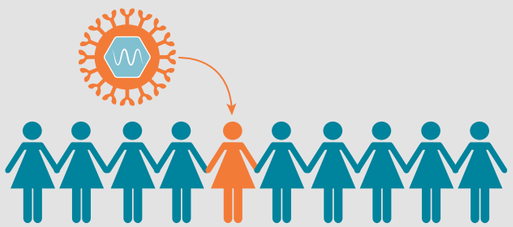
Varicella zoster virus (VZV) is a human herpesvirus that infects lymphocytes, skin cells and neurons. Primary infection causes varicella (chickenpox) and its characteristic rash of itchy blisters involves the whole body. Reactivation of the virus later in life causes zoster (shingles), for which the rash is usually contained to one or a few areas.

EPIDEMIOLOGY

Varicella occurs primarily in children and is a self-limiting, usually harmless disease. VZV is highly contagious; >90% of people become infected before they reach adulthood in countries without general varicella vaccination programmes. Age-related decline in immune function is one of the causes underlying the reactivation of VZV replication and the development of zoster. More than half of all 85-year-old people will have experienced at least one episode of zoster.



DIAGNOSIS

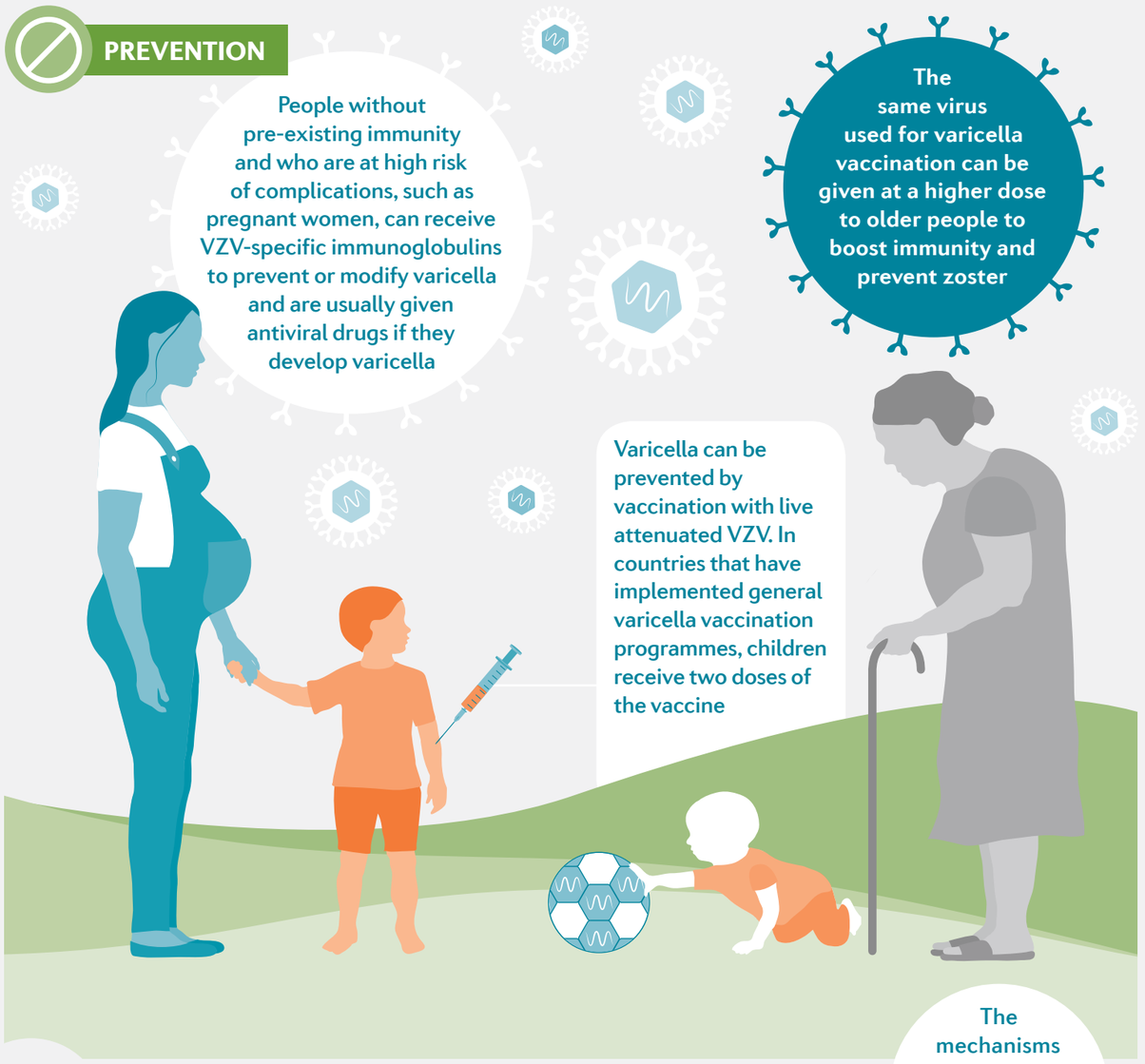
In most cases, varicella and zoster can be diagnosed on the basis of clinical presentation (its characteristic rash). Sometimes, however, the diagnosis needs to be confirmed using PCR for viral DNA, for example, when a patient presents with an atypical rash (such as zoster covering the whole body) or only with symptoms of possible complications (such as facial palsy or meningitis) without a rash.

PREVENTION

People without pre-existing immunity and who are at high risk of complications, such as pregnant women, can receive VZV-specific immunoglobulins to prevent or modify varicella and are usually given antiviral drugs if they develop varicella

The same virus used for varicella vaccination can be given at a higher dose to older people to boost immunity and prevent zoster

Varicella can be prevented by vaccination with live attenuated VZV. In countries that have implemented general varicella vaccination programmes, children receive two doses of the vaccine



MECHANISMS

The mechanisms that underlie the neuron-restricted latency of VZV are unclear

VZV can cause either lytic or latent infections. During lytic infection, the virus completes the full viral life cycle and new virus particles are produced. This damages the host cell, which, in skin cells, leads to the formation of blisters. Latent infection stops before new virus particles are produced and cells often show no obvious signs of infection. VZV establishes latency in neurons, and when viral replication is reactivated — often years later — the virus is transported along the nerve fibres to the area of skin or other tissues innervated by these neurons.

MANAGEMENT

In most patients, VZV infection requires no treatment. Only if a patient is severely ill or at risk of complications, for example, owing to compromised immunity, is antiviral treatment given. Nucleoside analogues are the most commonly used antivirals for VZV infection. Antiviral treatment of zoster treats acute pain from the disease but does not prevent or treat postherpetic neuralgia — a chronic pain syndrome and the most common complication of zoster. Postherpetic neuralgia is difficult to treat and can have a substantial impact on the quality of life of affected patients.

OUTLOOK

The varicella and zoster vaccines have greatly reduced the disease burden caused by VZV infections. However, only some countries have introduced general vaccination programmes and some people, including immunocompromised individuals and pregnant women, cannot receive the vaccines. This is particularly worrying because these people have the highest risk of complications.

Recently, a subunit vaccine containing recombinant VZV glycoprotein E was shown to be highly effective at preventing zoster without having the drawbacks of the live attenuated zoster vaccine

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