CASE REPORT

Herpes zoster with cutaneous dissemination: a rare presentation of an uncommon pathology in children

Catarina Faria,[1] Ana Antunes,[1] Henedina Antunes,[1,2] Susana Carvalho[1]

[1]Department of Pediatrics, Hospital de Braga, Braga, Portugal
[2]Life and Health Sciences Research Institute (ICVS), School of Health Sciences; ICVS/3B's—PT Government Associate Laboratory, Universidade do Minho, Braga/Guimarães, Portugal

Correspondence to
Dr Catarina Faria, catmagalhaesfaria@gmail.com

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SUMMARY

Herpes zoster, caused by varicella zoster virus (VZV) reactivation, affects mainly the adult population, although it can occur in children. This happens when primary infection (varicella) has occurred at a very young age or in immunocompromised patients. Complications are rare in healthy individuals. They include VZV cutaneous dissemination, which affects 2%–10% of immunocompromised patients.

We present a previously healthy child, with history of varicella during her first month of life, which presented at age 8 with a severe case of herpes zoster, complicated with cutaneous dissemination. Immunity study was unremarkable. Causes, management and follow-up are discussed.

BACKGROUND

Herpes zoster, or shingles, is a viral cutaneous infection caused by varicella zoster virus (VZV) reactivation, manifested by painful vesicular lesions grouped together along an area corresponding to one or, less frequently, more dermatomes.[1 2] Usually it is a benign, self-limited and well-tolerated pathology.[3 4]

While chickenpox is considered a childhood disease, herpes zoster mainly affects adults. From age 0 to 14 years the incidence is low (0.45 cases per 1000 individuals/year), especially in immunocompetent children. It is more frequently described when VZV primary infection (varicella) occurred in utero or during the first year of life.[2]

Cutaneous dissemination is a complication that affects 2%–10% of immunocompromised patients, being rare in the immunocompetent, especially at paediatric age.[5] This complication manifests 3 to 4 days after the onset of dermatome-associated lesions. Cutaneous dissemination occurs due to transitory viraemia during viral reactivation.[6 7] Other complications of herpes zoster must be kept in mind if cutaneous dissemination occurs, as viraemia can lead to the infection of other organs such has the brain, kidneys, lungs and liver,[8] which can be life threatening, especially in severely immunocompromised patients.[6]

CASE PRESENTATION

An 8-year-old girl was admitted in our unit for evaluation and treatment of an extensive papulovesicular rash.

Her symptoms had started 9 days prior to hospitalisation when she complained of electric shock-like pain in the area corresponding to the right cervical plexus (between the jaw and clavicle). Three days later, she developed an itchy erythematous papular rash over the right postauricular region which extended to adjacent scalp, cervical region and shoulder and subsequently became vesicular. She was evaluated by her paediatrician who diagnosed herpes zoster and started acetaminophen. After 3 days, the rash progressed, extending eventually to her thorax, abdomen and limbs, affecting palms and soles. She also developed fever (maximum 38.0°C every 3 hours).

Of note, her medical history was remarkable for varicella infection during her first month of life. She was born full term, following an uncomplicated pregnancy and had no history suggestive of immunocompromising conditions. Her growth and development were normal. She also had history of cutaneous reaction to amoxicillin at the age of 3, atopic dermatitis and allergic rhinitis (pollen). Her vaccination schedule did not include varicella vaccine, which is optional in our country; as so, antibodies to varicella were not checked after the infection. Family history was irrelevant, except for parental consanguinity (second degree cousins).

At admission, she had normal vital signs and was febrile. She presented with exuberant lesions, mainly vesicles and scabs (and some pustules), most of them in coalescence, distributed over the areas described above—corresponding to cervical plexus and C6 dermatomes (figure 1). Subjacent tissue presented erythema and oedema. Erythematous papules and vesicles were present through thorax (figure 2), abdomen and limbs. Remaining physical examination was unremarkable.

INVESTIGATIONS

Complete blood count was normal and C reactive protein was 8.91 mg/L. Renal function, ionogram and liver transaminases were normal. Blood VZV DNA screening was positive. Blood culture was negative. Given the extension of the affected area and the presence of disseminated lesions, HIV serologies, flow cytometry and quantitative immunoglobulins test were performed—results were unremarkable.

TREATMENT

The child was admitted to our Unit and started acyclovir 1500 mg/m^2/day intravenously. Given the concern about secondary infection antibiotics were

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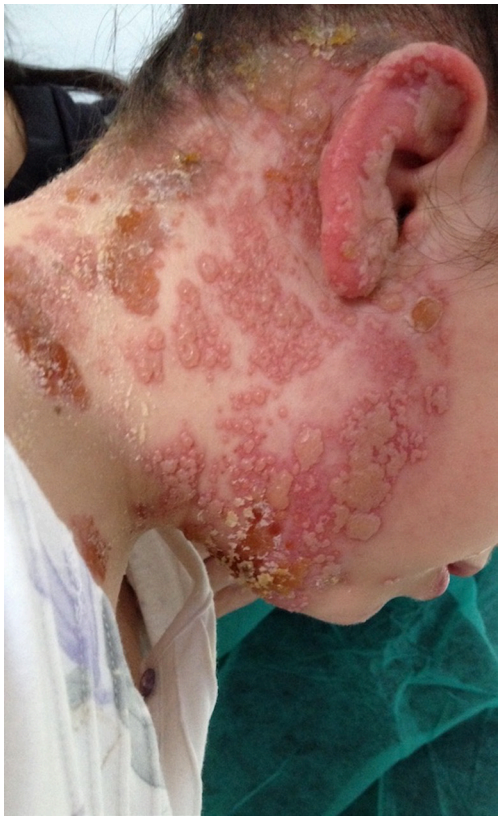

Figure 1 Zoster lesions in coalescence (vesicles, scabs and some pustules), at admission. Subjacent tissue with erythema and oedema, more pronounced on the ear.

added (flucloxacillin 150 mg/kg/day intravenously and clindamycin 30 mg/kg/day intravenously).

OUTCOME AND FOLLOW-UP

Fever subsided 3 days later. After an initial flare up, the localised and disseminated lesions began to improve. At day 8 of medication, she developed a generalised macular-papular rash affecting palms and soles, leading to flucloxacillin and clindamycin suspension. Cytomegalovirus, Epstein-Barr virus, parvovirus B19 and *Mycoplasma pneumoniae* blood serologies were negative. At day 10 of acyclovir, the child re-initiated electric shock-like pain in the affected cervical area, requiring tramadol prescription to control pain during that day. She completed 14 days of acyclovir. She was discharged after 15 days, with symptomatic treatment for residual pain and cutaneous care of remaining lesions. The pain subsided a few days after the discharge. During the following year, she was followed up in paediatric consultation—immunological screening was repeated and came out normal. There were no relapses or complications.

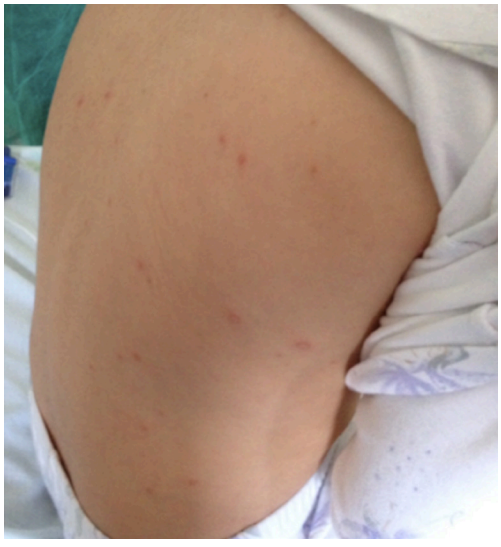

Figure 2 Disseminated cutaneous lesions (papules and vesicles) on thorax, at admission.

DISCUSSION

Herpes zoster involvement of multiple dermatomes and cutaneous dissemination is rare in immunocompetent children.[2] A study about herpes zoster complications concluded that cutaneous dissemination was the only complication with statistically significant difference between immunocompetent and immunocompromised children.[9] Therefore, children who present with this complication should be tested for immunodeficiency and malignancy. Ideally, tests should be done 1 month after the disease presentation. In our case, given the severity of manifestations, we chose to do it in the acute phase of the disease and to repeat it later, during follow-up in paediatric consultation. In this child, whose immunity test were unremarkable, the extension of the affected area and cutaneous dissemination were probably related to the fact that the she had varicella during her first month of life—in this period, due to immune system immaturity and presence of maternal antibodies, an effective immunity against this virus is difficult to obtain.[10] Universal varicella vaccination is not implemented in some countries, including ours. One of the main reasons for that was the concern that the widespread use of varicella vaccine would lead to an increase of herpes zoster cases in the adult population who had natural infection in the past.[11] The circulation of the wild-type virus was thought to be important for mantaining long-term immunity and prevent VZV reactivation. However, recent studies do not support this hypothesis.[12] Although the attenuated vaccine virus can reactivate and cause herpes zoster, it does it less frequently than the wild-type virus. Therefore, universal immunisation would ultimately lead to a global reduction in the incidence of herpes zoster.[6]

Acyclovir (orally: 40–60 mg/kg/day; intravenously: 30 mg/kg/day or 1500 mg/m^2/day if <12 years) for 7–10 days is the treatment of choice for herpes zoster and should be initiated in the first 72 hours,[13] to decrease the duration and severity of the disease.[7] Treatment can be extended if new vesicles continue to arise (until 2 days after the last ones appear) or while complications persist.[13] Once our patient had complicated herpes zoster and re-initiated pain in day 10 of acyclovir, we decided to extend treatment to 14 days.

Usually, herpes zoster resolves in 1–3 weeks.[13] Sequels such as postherpetic neuralgia (pain that lasts >4 months since the arise of skin lesions) are rare in children—nevertheless, it is important to follow-up these patients during enough time to exclude its development.[9] In our patient, no sequelae were documented.

Learning points

- Herpes zoster is rare in children but can happen when varicella occurred in the first year of life or in utero.
- Rare complications of herpes zoster should prompt a thorough screening for immunodeficiency and malignancy.
- Children should be followed-up for postherpetic neuralgia, a rare but important sequel.

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