

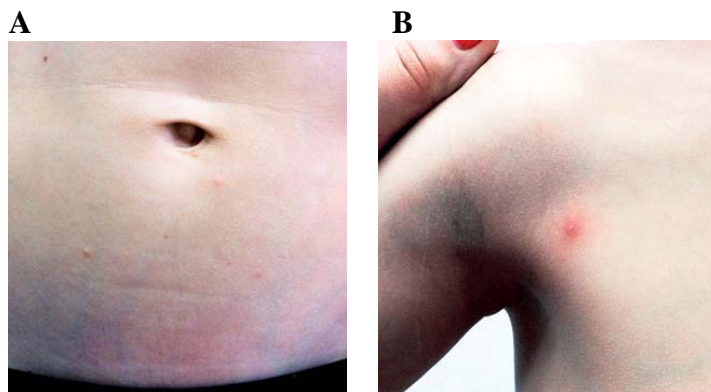
Vesicular rash after an exotic trip

A pediatrician examines a 9-year-old boy complaining of groin swelling and body rash. The first red bulging element developed on the groin skin 8 days ago in the setting of sub-pyretic fever; this element later developed into a sore with raised edges under dark hemorrhagic crust (pic. 1); groin lymph nodes have become bigger. The child had been examined by a surgeon, who established diagnosis “Acute lymphadenitis” and prescribed amoxicillin/clavulanate and chloramphenicol-based salve dressings. Several days later the boy started to develop vesicular rashes on body skin, which have persisted until now (pic. 2 A, B). The child feels well; body temperature normalized 2 days ago. Medical history also shows that a week ago the boy, his 20-year-old brother and father returned from the Republic of South Africa, where they had been taking a vacation for 10 days and participated in bushmeat hunting. The father has no disease. 2 days ago, the older brother found a tick on his body and removed it himself. According to the mother, a red bulging element is now observed in the sting site.

Pic. 1. Hemorrhagic crust with hyperemic aureola in the right groin (and regional lymphadenitis)



Pic. 2. Papulovesicular rash in the child



Probable diagnosis?

1. Chickenpox.
2. Pityriasis lichenoides et varioliformis acuta.
3. Pyoderma with local lymphadenitis and drug toxicoderma.
4. Tick-borne spotted fever.
5. Lyme disease.
6. Generalized herpes.

Correct answer: 4. Tick-borne spotted fever.

Chickenpox (etiology – virus *Varicella zoster*) is one of the most frequent causes of vesicular rash in children. However, vesicles usually develop on the face and the scalp first and then spread to the body and the limbs in the event of chickenpox. Life duration of vesicles in the event of chickenpox is 8-12 hours. Our patient is characterized by scanty vesicular rash without erosions and scabs persisting for several days [1].

Pityriasis lichenoides et varioliformis acuta is a rare skin pathology of unknown pathology. It manifests itself with acutely developing papules, which rapidly develop into pseudovesicles characterized by necrosis in the central part of the elements. It is sometimes accompanied by fever and disturbed state of health. In most cases skin rashes subside within several weeks or months spontaneously; however, rashes may spontaneously develop and disappear within months and years after the first episode [2]. It is a noncontagious disease. In our patient, rash duration is only around 1 week. Neither anamnesis morbi featuring a visit to Africa nor a probable tick sting count in favor of pityriasis lichenoides et varioliformis acuta.

Pyodermas may develop in the event of a slightest skin damage and manifest themselves with impetigo (superficial skin infection characterized by formation of a vesicle of a pustule, lancing of which results in erosions and scabs), ecthymas (deep skin infection characterized by formation of a scabbed ulcer), abscesses, furuncles and carbuncles. They are usually accompanied by regional lymphadenitis. Amoxicillin/clavulanate is a drug of choice for treating pyodermas, as the most frequent causative agents of pyodermas are group A streptococcus and *Staphylococcus aureus*, which are sensitive to this antibiotic. In our patient, bacterial infection as etiology of the first element (groin) is unlikely given absence of positive dynamics in the setting of amoxicillin/clavulanate intake. Vesicular non-pruritic rash is uncharacteristic of allergic reactions, including drug toxicoderma. Typical clinical manifestations of allergic reactions to pharmaceuticals are drug (morbiliiform) rash, urticaria, Quincke's edema, fixed toxicoderma, bullous rash, Stevens-Johnson syndrome, Lyell's syndrome, vasculitis (hemorrhagic rash), lichen ruber planus-like rash, photoallergic reactions [1]. No such manifestations were observed in our patient.

Causative agent of Lyme disease – spirocheta *Borrelia burgdorferi* – is carried by ixodidae. Chronic migratory erythema in the form of a papule or a macule which develop into an annular erythema with bright red outer boundary and fading in the center within several days is the first manifestation of Lyme disease (Lyme borreliosis). Size of chronic migratory erythemas varies from 3 to tens of centimeters. A vesicle, thickening or necrosis may develop in the center of the erythema. Secondary erythemas may develop aside the primary element; they are smaller and do not feature thickening in the center. Pyretic or low-grade fever may be observed beside the rash; neurological complications, such as meningitis, encephalitis, cranial neurites and radiculopathies may develop several days/months after the rash [3]. Vesicular rash is uncharacteristic of Lyme disease [1]. It should also be mentioned that, although ixodidae inhabit Africa, Lyme disease is not observed in that region.

Generalized herpes (causative agent – *Herpes simplex virus*) is a severe disease observed at immunodeficient diseases, particularly in AIDS patients. Herpetic vesicles develop in groups in the virus penetration site in people without immune disorders. In the event of transmission by direct contact, vesicles may develop on various parts of the body; however, the most affected areas are lips, circumoral skin, cheeks, nose tip and distal maniphalanxes. Herpetic vesicles feature umbilication in the center; they are surrounded by hyperemic areola; vesicles burst spontaneously, and scabs form instead of them.

According to the epidemiological anamnesis (visit to Africa, tick sting and similar clinical pattern in the brother) and characteristic clinical manifestations ("black spot" and papulovesicular rash), tick-borne spotted fever is the most likely diagnosis for the child.

Tick-borne spotted fevers are carried by gamasoidae and ixodidae and may take vary in course from mild ailment and rash to severe, life-threatening condition. Old World tick-borne spotted

fever (Marseilles fever) and vesicular rickettsiosis are observed in Africa [4, 5]. The “black spot” is characteristic of both diseases. A papule develops in the tick sting site; after 3-7 days, it develops into a “black spot”. “Black spot” is a small ulcer with dark brown scab in the setting of hyperemia (looks like a cigarette burn; see pic. 1). Papulovesicular rash is especially characteristic of vesicular rickettsiosis (causative agent – *Rickettsia akari*). Several days after the disease onset, papules start to develop on body skin, in the center whereof vesicles develop. Vesicular rickettsiosis is usually characterized by mild course. Diagnosis of rickettsiosis may be confirmed by polymerase chain reaction (detection of rickettsia DNA in rash elements and blood) and serologically (enzyme immunoassay for IgM-antibodies). Doxycycline is a drug of choice for treating tick-borne spotted fevers; it decreases severity and duration of the disease. The patient had been taking doxycycline in the dose of 100 mg/day for 14 days and ultimately recovered in that setting.

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