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ORIGINAL CONTRIBUTIONS

CUTANEOUS INVOLVEMENT IN ACUTE MENINGOCOCCEMIA (Study of an epidemic in Delhi)

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Thirty (88.2%) out of 34 children with skin lesions were observed during the recent epidemic of meningococcus group A infection in Delhi. Purpuric lesions were the commonest (60%), maculo-papular in 26.67% and faint pink macules in 13.33%. Conjunctivae were affected in 3. These eruptions appeared within 6 hours to 4 days of onset of the disease. Cutaneous ulcers and gangrene appeared after 7 days in one child. Severe vascular damage was seen on histopathology of the ulcers as compared to the early lesions. Gangrene of the extremities developed during the recovery phase when all other signs of the disease had subsided.

Key words : Acute meningococcemia, Skin lesions.

During the winter of 1984 there was an outbreak of meningococcal infection in and around Delhi. The numbers increased in peak winter and by mid-1985 more than 1900 cases had been recorded with a fatality of 13%.[1] A similar situation prevailed in 1985-86 during which many patients, particularly children, were referred to our hospital. The present article deals with the skin lesions seen in these children.

Materials and Methods

Thirty four children between 6 months and 11 years of age, having acute meningococcemia were observed. The diagnosis had been confirmed by recovery of *Neisseria meningitidis* group A on culture of blood in chocolate agar. Complete hemogram was done in all. Tests of clotting, bleeding time (Duke method) and coagulation time (Lee-White) were done in 10 children. Skin biopsy was done in 3 patients.

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Results

Thirty (88.2%) patients had skin lesions which appeared between 6 hours to 4 days after the start of the illness. Purpuric eruptions were the predominant lesion in 18 (60%) and maculo-papular in 8(26.7%). Faint pink macules were the only lesion seen in 4(13.3%) while in 7(23.3%) these were seen in association with papular and/or purpuric lesions mentioned above. These macules could be made out only on careful examination of the skin surface. Though in many cases, macular, papular and purpuric lesions could be demonstrated in the same patient, only one type predominated. The eruption was generalised in 50% with sparing of the face and involvement of the palms and soles in 6, while in the rest it was limited to the lower limbs and abdomen. Three children had conjunctival petechiae and 7(20.6%) had herpes labialis. Twenty nine patients became afebrile in 48-72 hours after parenteral therapy with crystalline penicillin and gentamicin in adequate dosages, and the rash disappeared completely within 7-10 days leaving normal skin. Two children had late cutaneous mani-

festations. An 11-year-old male developed dull red plaques over the lower limbs on the 9th day which resulted in multiple deep ulcers of varying sizes over the thighs and legs, necessitating skin grafting. Another patient had gangrene of a finger tip on the 10th day and dry gangrene of all the toes on the 25th day.

One child succumbed to shock and had a platelet count of 80,000/ mm^3 and prolongation of both bleeding and coagulation times. Tests of clotting in the remaining 9 were normal. The patient with gangrene of all the toes had platelet count of 150,000/mm^3, while the rest had counts ranging between 180,000/mm^3 and 220,000/mm^3.

Biopsies were done in 3 patients : 2 with maculo-papular lesions and one from the child with ulcers. The former revealed dilatation and swelling of the endothelial cells of the blood vessels in the upper and mid-dermis, their lumina being partially occluded by eosinophilic material. A perivascular infiltrate of neutrophils and lymphocytes was seen. Section from the ulcer showed necrosis of the vessel walls in the lower third of the dermis. An infiltrate composed of mainly mononuclear cells, a few lymphocytes, neutrophils and nuclear dust was seen within and around the damaged blood vessels. Gram stain revealed no organisms.

Comments

Cutaneous lesions are seen in 61%-100% of the patients with acute meningococcal infection, the higher incidence being attributed to increased awareness.[2] Based on the clinical observations of other workers,[2-7] the cutaneous manifestations of acute meningococcemia can be broadly described under three groups : (1) early lesions, occurring between a few hours to 7 days of the illness, which constitute purpuric lesions including petechiae, suggillations, ecchymoses, purpura fulminans and purpura topped by a vesicle or pustule; maculo-papular, papular, urticarial eruptions, and plaque-like lesions, (2) late lesions, appearing 7-10 days after the onset, which clinically manifest as nodules and ulcers showing severe vasculitis on histopathology, episcleritis and peripheral gangrene, and (3) associated infections, herpes simplex being frequently seen, and herpes zoster rarely. These studies also revealed that purpuric lesions were seen in 70% of the cases and were generally distributed over the trunk and extremities, sometimes over the palms and soles, while the face was usually spared. The conjunctivae and oral mucous membrane may be involved[3] and occasionally large areas of haemorrhage may be seen.[4] Though purpuric lesions initially evolve through a macular stage,[2] the macules may be so faint as to escape detection, unless carefully examined. Appearance of a rash after prodromal symptoms should alert the physician in making a clinical diagnosis prior to meningeal localisation.[5] The macules appear in crops and may change to papules, purpura and other variants. On the whole the eruptions are polymorphic, though only one type may predominate in the course of time. Depending upon the morphology and distribution the lesions may mimic erythema multiforme,[4] rose spots of typhoid,[6] erythema nodosum[6] and Osler's nodes.[6]

Two mechanisms are involved in the pathogenesis of acute meningococcemia—Shwartzmann phenomenon and immune complex reaction. Accordingly the early lesions predominantly show a picture of non-leucocytoclastic vasculitis and the late lesions which are mediated mainly by immune complexes show a picture of leucocytoclastic vasculitis.[6,8,9] The patients we biopsied conformed to this description, but diplococci could not be demonstrated in the sections. It has been observed that smears from purpuric lesions often revealed the organisms, whereas those from macular and late lesions are negative for organisms.[6,10] Other studies have shown the presence of diplococci within the thrombosed vessels, leucocytes, endothelial cells and extravascular space.[9-12]

Vesicles, when present, can be located either subepidermally or in the epidermis.[2,13]

Fulminant infections can interfere with the clotting mechanisms leading to disseminated intravascular coagulation[14] with a high mortality rate. Gangrene is an uncommon complication. The patient with gangrene of all the toes had a platelet count in the lower limit of normal, probably due to their consumption in the thrombotic process. Since, gangrene can manifest during the later period when the patient has clinically recovered, they must be kept under follow-up for at least a month from the date of onset of the illness.

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