reason in 10). Ten patients were treated surgically and then given antibiotics; the remaining 96 patients did not receive any antibiotics. Altogether 139 patients with an abscess (89%) were discharged within seven days of their initial treatment.

Of the 119 patients without an abscess, nine with animal bites were given antibiotics when the bacteriology results were available and four with compound digital fractures were given long courses of antibiotics because of suspected bony infection.

Comment

Altogether 38 out of 275 patients (14%) were given antimicrobial treatment in hospital; in only nine cases (3%) was the treatment based on the result of culture and antibiotic sensitivity. In over 60% of cases the patient was discharged before the results of culture were available; in another 25%, despite prolonged follow up, the result was not recorded. This practice is similar to that in other accident and emergency departments, indicating that routine sampling for microbiological testing is unnecessary.

Early and thorough debridement is the most important factor in preventing infection in traumatic wounds. Successful treatment of an established infection depends on early diagnosis and accurate localisation of the sepsis followed by incision and adequate drainage; antibiotics are not then usually necessary. In patients in whom antibiotics are thought to be desirable—for example, those at high risk and those with signs of spreading cellulitis or systemic infection—there are usually sufficient clinical signs to indicate the likely infecting organism and its sensitivity, thus making laboratory confirmation unnecessary.[3 4] Laboratory studies have confirmed that erythromycin and flucloxacillin are active against the most commonly implicated pathogens.[5] Bacteriological examination should be confined to patients with periorbital infection or human and animal bites (particularly near joints) and those in whom drainage alone may not eradicate infection from the surrounding tissue.

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Herpes zoster of second and third segments causing ipsilateral Horner's syndrome

Herpes zoster, an infection of the first sensory neurone and its corresponding area of skin, usually affects ganglia of the dorsal root and the fifth and seventh cranial nerves. It may also cause other neurological complications, such as motor paralysis, myelitis, encephalitis, cranial nerve paralysis, pupillary abnormalities, diaphragmatic paralysis, polyneuritis, and contralateral hemiparesis.[1-3] We describe a case of Horner's syndrome presenting as a complication of herpes zoster of the second and third thoracic segments.

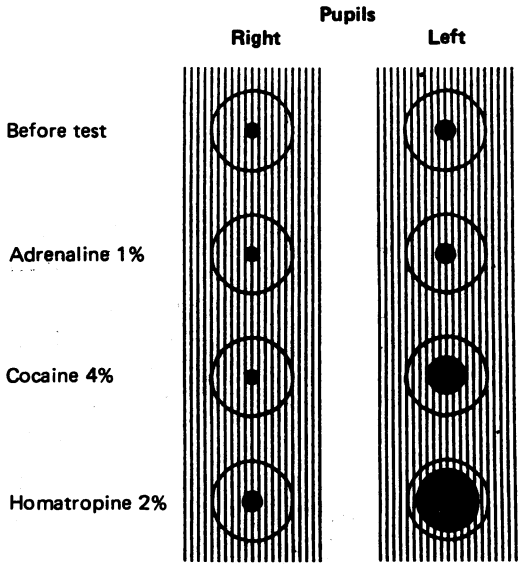
Case report

A previously healthy 46 year old man presented in July 1984 with drooping of the right eyelid. Two weeks before he had developed a painful rash over his right scapula and in his right axilla. This had the typical appearance of shingles. He had also noticed a slight redness of the right eye with drooping of the eyelid. The redness had settled within a few days but the ptosis had persisted. In addition, he had noticed that the right side of his face was warmer and drier than the left.

Examination showed healing scars of herpes zoster over the distribution of the second and third thoracic segments on the back of his chest and in his right axilla running down towards the medial side of his arm. There was partial ptosis, miosis, and apparent enophthalmos of the right eye. The right side of his face felt less moist than the left. There was no finger clubbing or cervical lymphadenopathy or any evidence of pulmonary abnormalities.

Haematological and biochemical investigations yielded normal results. Radiographs of the chest taken in October 1984 and in August 1985 showed no abnormality.

Pupillary reaction was tested with 2% homatropine hydrobromide, 4% cocaine hydrochloride, and 1% adrenaline eye drops (figure).



Approximate pupillary sizes of both eyes during test. (Vertical lines are about 1 mm apart.)

Comment

Horner's syndrome, or ocular sympathetic paresis, occurs when there is interruption of the sympathetic pathway at any point from the hypothalamus, brain stem, spinal cord, sympathetic chain, stellate ganglion, or sympathetic plexus along the internal carotid artery and the ocular sympathetic fibres. It is, therefore, difficult to locate the exact lesion in Horner's syndrome. Studying the pupillary reaction to adrenaline may help to differentiate between preganglionic and postganglionic lesions. In postganglionic lesions adrenaline causes dilatation of the pupil because of denervation hypersensitivity. If the lesion is preganglionic the size of the pupil will not change.[4] Pupillary response to other substances, such as cocaine, which inhibits the uptake of noradrenaline, may help to differentiate a peripheral from a central cause. If the lesion is within the central nervous system the pupil will dilate, whereas if the lesion is peripheral there will be no response.[5]

Our patient had hemifacial anhydrosis, and the results of pupillary tests suggested a preganglionic lesion localised to the second order neurone which stretches from the lateral horn of the upper thoracic segments (T1-T4) to the cervical sympathetic ganglia. He also had a herpetic skin rash in the distribution of the second and third thoracic nerves on the same side as his Horner's syndrome. The combination of these findings leaves little doubt that in this patient the Horner's syndrome was due to herpes zoster affecting the sympathetic fibres at the level of T1-T3.

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