intoxication of neonates(7). However, our experience shows that it does not have any therapeutic usefulness. This is quiet understandable because pharmacologically aminophylline has no specific action against diazepam.

With a view to avoid the adverse effects of diazepam on the fetus and neonate, it is suggested that alternative drugs like flurazepam and lorazepam be used. These benzodiazepines have shorter half life, are not converted to active metabolites and at the same time give adequate sedation in eclampsia.

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Oral Steroids in the Treatment of Periorbital Hemangioma

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Hemangiomas are common tumors of infancy(1). Nearly 90% of them resolve without complications or significant cosmetic deformity(2). Eventhough most of the hemangiomas resolve spontaneously, those occurring over special locations like eyelids, subglottic area, need immediate treatment. Various treatment modalities are available for treatment of hemangiomas. The commonly used methods include, surgical resection when small, intralesional steriod injection and in recent years, pulsed dye laser in early superficial lesions(1). Non-availability of pulsed dye laser, morbid fear of surgery, anesthesia and injections near vital organs like the eye make these modalities of treatment unsuitable for most of our patients.

Systemic steroids are suggested for treatment of visual or respiratory obstruction caused by a rapidly enlargng hemangioma or to treat the complications of Kasabach-Merrit Syndrome(3). We report a one-month-old infant with large

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hemangioma completely occluding the right palpebral fissure who responded remarkably with oral steroids. Such therapy, we believe, is simple, acceptable by most of our patients and can be practised even in remote areas.

Case Report

A one-month-old infant was brought to us with a large hemangioma completely occluding the right palpebral fissure (*Fig. 1*) which was present right from the birth. The child was given oral prednisolone in a dose of 2 mg/kg/day for a period of 4 weeks. We observed that there was a remarkable regression of the hemangioma and the child was able to open the eye (*Fig. 2*).

Discussion

The incidence of hemangioma in term infants is 1.1–2.6%(1). However, 10-12% of children develop hemangiomas and most of them are noticed at about 1 month of age(1).

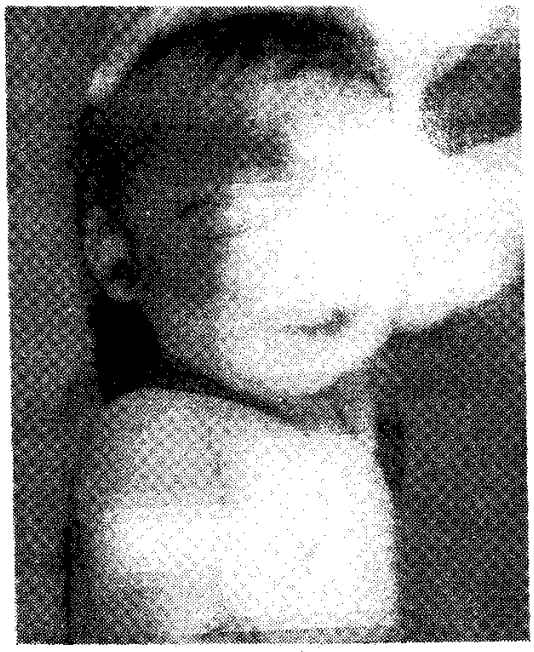

Fig. 1. Hemangioma occluding the right palpebral fissure

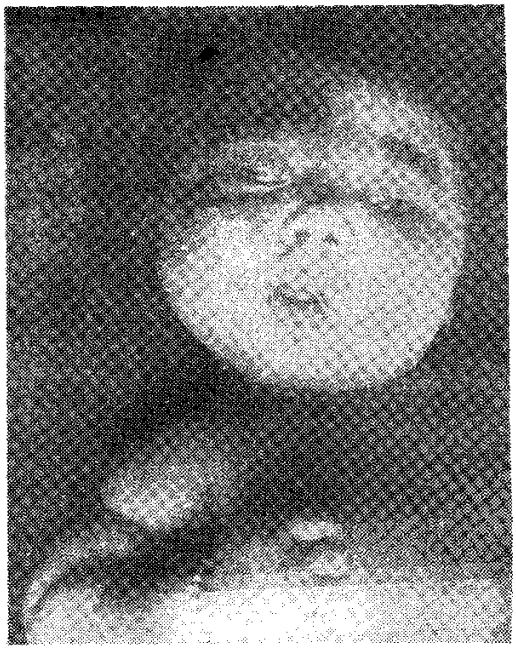

Fig. 2. Regression of the hemangioma.

The most superficial forms of hemangiomas are the capillary or strawberry hemangiomas which involve the dermal vessels. When the deeper vasculature is involved the hemangioma is called cavernous, which can extend into subcutaneous tissue and even deeper into the musculature. If both the dermal and subcutaneous vessels are involved, the lesion is called a mixed capillary cavernous hemangioma.

Hemangiomas have a well defined course consisting of an early rapid growth phase, followed by slow involution in most cases(2). Most clinical series report complete or near complete regression of cutaneous hemangiomas by the age 5 to 7 years, with most of this process occurring within the first few years of life(3). Nearly 10% of patients will develop problems like ulceration, infection, localized hemorrhage, Kasabach-Merritt syndrome, disfigurement from rapid growth, occlusion or

compression of a vital structure or orifice and high output cardiac failure(1).

Hemangiomas over the eyelids may produce permanent visual damage by occlusion of the pupil, compression of the globe or extension into the retronbulbar space. Periorbital hemangiomas if untreated may go in for significant complications like amblyopia, astigmatism, strabismus, anisometropia, ptosis, proptosis, exposure keratitis, and optic nerve atrophy(1).

Several decades ago radiotherapy was used for treatment of hemangiomas and has been given up because of its longterm sequelae. Sclerosing agents and cryotherapy tried for this purpose cause significant scarring and so became unacceptable in most cases. Surgical resection is possible only in small hemangiomas(4). The larger ones are difficult to excise, because they usually extend deep into the tissues and are fed by large number of small vessels. The resulting scar is another disadvantage. Compression therapy has shown to encourage regression and reduce the size of stable masses in selected patients. Since interferons have antiproliferative effects, synthetic interferon alfa 2a has been used successfully in certain type of hemangiomas(1). Laser has been used in recent years for surgical excision of hemangiomas. Pulsed dye laser is a new therapeutic tool useful in early superficial lesions and treatment option for ulcerated hemangiomas(2).

In few cases systemic steroids have been used for the treatment of visual or respiratory obstruction caused by rapidly enlarging hemangiomas(3). Intralesional corticosteroids is another treatment option. High local concentrations of corticosteroids from intralesional therapy produce more rapid results than orally administered agents. But this needs repeated injections and carries significant complications apart from the necessity of general anesthesia for each injection because of pain from the procedure. The complications of local injections in periorbital hemangiomas include central retinal artery occlusion, full thickness eyelid necrosis, linear subcutaneous atrophy and localized lipoatrophy(1).

Systemic steroids are recommended because of the problems of intralesional therapy. This treatment is well accepted by our patients. But before giving steroids one should ensure that the local infection is brought under control with appropriate antibiotics. Prednisolone in a dose of 2-3 mg per kg per day usually causes shrinking of the hemangioma in 3 days to 3 weeks(3). Hemangiomas in infants less than 6 months of age tend to be more steroid responsive. Alternate day steroids are advocated by some authors. Rebound growth may occur sometimes when steroids are discontinued. If this happens a second or third course of steroids will usually be effective(5). A 7 year follow-up study of patients treated with systemic steroids showed no recurrence of the lesion(5).

Similarly, systemic steroid therapy could be tried in cases of facial hemangiomas and those involving the nasal tip. Facial hemangiomas may produce facial asymetry from a significant subcutaneous component or from overgrowth of underlying bone, presumably due to increased periosteal circulation(3). Hemangiomas involving the nasal tip is a serious cosmetic problem and should be appreciated by the physician. The mechanism of action of steroids on enlarging hemangiomas is not clear. It is postulated that corticosteroids increase vascular sensitivity to circulating vasoconstrictive agents(5).

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Radial Immunodiffusion versus Serum Protein Electrophoresis as a Tool for Diagnosis of Alpha-1-antitrypsin Deficiency

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Alpha-1-antitrypsin (AAT) derived its name from the identification of an alpha-1-globulin on serum protein electrophoresis whose activity was measured by inhibition of trypsin action. Since AAT is actually capable of interfering with the action of a variety of proteolytic enzymes, it is also known as alpha-1-protease inhibitor. In 1963 Laurell and Eriksson first reported a genetically determined deficiency of a major serum protease inhibitor which has strong association with early onset of severe emphysema(1). In 1968 Sharp noted a serum electrophoretic pattern without an a_1-globulin peak and found it belonged, not to an adult with emphysema, but to a child with cirrhosis(2). Studies have shown that in those affected with AAT-deficiency, conjugated hyperbilirubinemia usually occurs within first 3-4 months of life(3-5). Elevated hepatocellular enzyme levels which remain so even after the jaundice has cleared and bile-duct hypoplasia have been reported(6,7). Recently, we encountered AAT-deficiency in a 2-month-old female Libyan patient, which we believe, is the first case of AAT-deficiency reported from Libya.

Case Report

The reference child was born to a non-consanguinous parents and was admitted with diarrhea of 10 days and jaundice of 2 days duration and lethargy. The child was born 3rd in order at 35-36 weeks of gestation and weighed 2200 g at birth. There was no family history of jaundice or any history suggestive of liver disorder. The child was breast fed for 5 days and thereafter received formula feeding.

On general examination, the child was sick looking, moderately jaundiced,

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