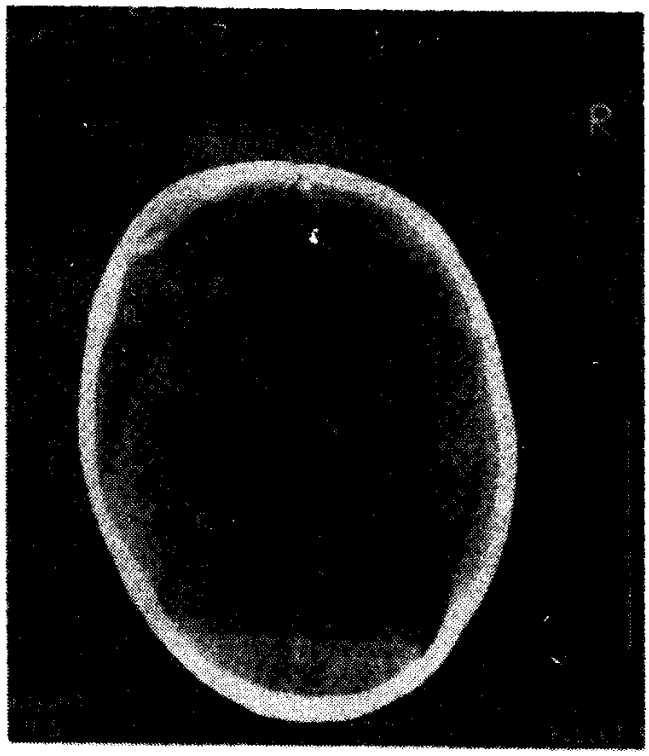

Fig. A hypodense lesion between the inner table of the skull and the brain parenchyma in the left frontoparietal region causing collapse of the ventricles on the same side and shift of the brain to the right. B&C. Areas of decreased attenuation in the left cerebral hemisphere suggestive of cerebral edema.

irritability, failure to thrive, anemia and unexplained fever as documented by others (1,2) were all present in the case under review. However, vomiting which is almost invariably present(1) was conspicuous by its absence in our patient. Retinal or subhyaloid bleed that are pathognomonic of chronic subdural hematoma was also not seen in this infant. Ipsilateral 3rd cranial nerve palsy as noted in the patient under review has not been reported earlier. Though head injury is the usual initiating factor it may be so insignificant as to have been forgotten or disregarded. The parents may come out with the history only on repeated questioning as seen in this case.

Summing up, the possibility of chronic subdural hematoma should be considered in an infant with this clinical profile, specially if the head size is rapidly increasing despite decongestive therapy. Ultrasound or CT Scan of the head should be done to rule out the possibility.

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Visceral Leishmaniasis Treated with Rifampicin and Cotrimoxazole

Visceral leishmaniasis is a parasitic disease treated conventionally by stibogluconate or, in stibogluconate resistant cases, pentamidine. The more toxic amphotericin B is recommended in cases not responsive to these drugs. Other drugs including rifampicin, INH and cotrimoxazole have been used in therapy of visceral leishmaniasis with inconsistent results(1). We

treated 13 cases of visceral leishmaniasis with rifampicin and cotrimoxazole and showed clinical and hematological improvement in 9 cases (69%). The first case did not respond to both stibogluconate and pentamidine given in adequate dosage and duration. He was then treated with oral rifampicin and cotrimoxazole and showed satisfactory response. In all subsequent cases rifampicin and cotrimoxazole were used as first therapy.

All the patients were between 3 and 11 years of age; 8 were girls and of the 13 cases, 9 were residents of Bihar and 4 of West Bengal. The diagnosis was made in presence of fever, hepatosplenomegaly, positive aldehyde test and demonstration of Leishman Donovan (LD) bodies in bone marrow or splenic aspirate.

All cases were treated with oral rifampicin 15 mg/kg/day and cotrimoxazole 5 ml (40 mg trimethoprim and 200 mg sulfamethoxazole) twice daily for those up to 6 years of age and one tablet cotrimoxazole (80 mg trimethoprin and 400 mg sulfamethoxazole) twice daily for those above 6 years of age. Four out of 13 cases, with a hemoglobin level of less than 6g/dl, required initial blood transfusion. The drug therapy was continued for 8 weeks for the 9 patients who responded. The remaining 4 cases, who did not show any response to this therapy within 2 weeks, were treated with parenteral stibogluconate with satisfactory results.

All the 9 cases who responded to this therapy became aferbile within 4-7 days. But even on the fourteenth day all patients, except one, had LD bodies demonstrable in bone marrow. By the end of 4th week of therapy no LD bodies could be demonstrated in any of these cases, but regression of spleen was not clinically detectable in seven out of nine cases. At the end of 8 week therapy a definite regression of spleen was noted in all cases and LD bodies were not demonstrable.

All the 9 cases who responded were reviewed every month for the next 6 months for fever and hepatosplenomegaly, and bone marrow examinations for the presence of LD bodies 3 monthly. Till date 3 cases are followed up beyond 6 months, 4 cases are under supervision between 3 to 6 months and 2 cases are followed up for less than 3 months. None of them has yet shown any feature of relapse, except one, who relapsed within 2 weeks after stoppage of therapy.

Visceral leishmaniasis is called cured when no evidence of clinical or hematological relapse is seen within 6 months after therapy(1). The first case of this series was cured by this therapy but did not respond previously to two courses of stibogluconate and two courses of pentamidine tried over a span of more than five months. This patient became afebrile within 5 days of rifampicin and cotrimoxazole therapy. Encouraged by this prompt response and cure we treated all subsequent 12 cases in this series with rifampicin and cotrimoxazole at diagnosis but we got response in 9 out of 13 cases (69%). After stoppage of therapy, 3 cases had no relapse even after 6 months, one relapsed within 2 weeks and rest 5 cases have not yet shown any feature of relapse though 6 months follow up is not yet complete. The preliminary results indicate that rifampicin and cotrimoxazole could be evaluated for the treatment of visceral leishmaniasis for the convenience of oral administration and prompt response, accepting lack of cent per cent response and occasional relapse.

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P-Floxacin Induced Arthropathy

Resistant Gram-negative infections especially multidrug resistant typhoid fever in children necessitates therapy with fluoroquinolones, *i.e.,* ciprofloxacin, P-floxacin or O-floxacin. Systemic use of fluoroquinolones results in a number of side effects even when administered in recommended therapeutic doses. The common side effects are fever, drug rash, gastrointestinal complications, benign raised intracranial tension, seizures, arthralgia and rarely bone marrow suppression. Toxic crisis following treatment of typhoid fever with P-floxacin as well as hydroarthrosis, thrombocytopenia, skin and musculoskeletal complications, and reputure of Achilles tendon have been reported(1,2). Destruction of the growing epiphysis in animal models cautioned pediatricians all over the world against the routine use of fluoroquinolones, though recent studies in human beings do not conclusively prove this hypothesis(3,4).

Here we report P-floxacin induced arthropathy in an adolescent girl treated for enteric fever.

A 12-year-old female treated with P-floxacin for typhoid fever (Widal positive) by a private practitioner presented to us with joint pains and difficulty in getting up from bed of one week duration and persistence of fever even after 20 days of administration of P-floxacin (400 mg orally twice daily). For joint pains she had received ibuprofen-paracetamol combination and mefenamic acid without any relief.

General examination revealed only a palpable spleen. Investigations revealed sterile blood, urine and stool cultures, negative Widal test and normal hemogram. ASLO titre, rheumatoid factor, LE cell phenomenon, antinuclear antibody test and Mantoux test were negative. *X*-ray of knee joints and surrounding long bones were normal.

All the drugs were discontinued and the child was kept under observation. The fever subsided within 48 hours. The difficulty in getting up from bed disappeared. The joint pains gradually disappeared over a week's time in all the joints except the left knee joint which took almost 3 months to be completely pain free. Now the child is leading a completely normal life. Movements around all joints are normal with no skeletal deformity. Thus the routine use of P-floxacin in childhood enteric fever should be advocated with a word of caution.

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