tem symptoms which reversed once BP was controlled, a label of hypertensive encephalopathy can be given to the case. The absence of papilledema or hemorrhages precludes the diagnosis of malignant hypertension.

The mechanisms of hypertension in Wilm's tumor include compression of renal vasculature or parenchyma and renin production by the tumor itself(4). To know the pathogenesis of hypertension is very important to decide the antihypertensive agents. Early surgical removal of the tumor is advocated.

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Turner's Syndrome in a Neonate

Turner's syndrome, though relatively not so rare, is very uncommonly diagnosed in the early neonatal period. This report described one baby diagnosed as Turner's syndrome clinically at birth.

A full term female baby, a product of non-consanguinous marriage, weighing 2750 g, was born normally. Examination revealed edema of the dorsum of both upper and both lower extremities extending up to the base of the terminal phalanges (*Fig. 1*), webbing of the neck with loose posterior nuchal skin folds (*Fig. 2*); triangular face with micrognathia; large abnormal auricles, high arched palate, widely spaced nipples and hypoplastic nails.

There was no evidence of coarctation of aorta or hypertension. A skeletal survey and ultrasonography of the abdomen did not reveal any abnormality. Karyotyping revealed a genotype of 45 XO . The baby's FSH, LH and TSH levels were 160.4 MIU/ml (5-15 mIu/ml), 86.0 MIU/ml (5-15 mIu/ml) and 10.7 MIU/ml (0-9 mIu/ml), respectively.

Turner's syndrome was first described by Turner in 1938(1) and is caused by complete or partial monosomy of the short arm of 'X' chromosome, the incidence being 1 : 2500 female births. Almost 95% of all Turner's conceptuses are aborted. Fifty-seven per cent of all Turner's syndrome have 45 XO. Other chromosomal anomalies are--mosaicism (45X/46XX, 45X/47XXX, 45X/46XY) and isochromosomes(2). Patients presenting during the neonatal period are usually 45XO, as mosaics present later in life.

Investigations include karyotyping, FSH levels in blood and in urine, diabetes

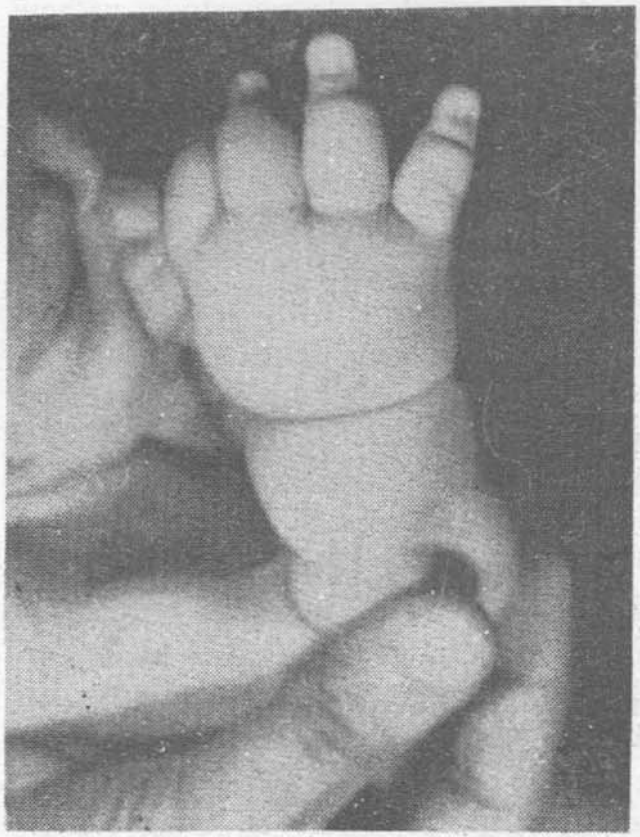

Fig. 1. Clinical photograph showing edema of the dorsum of extremity.

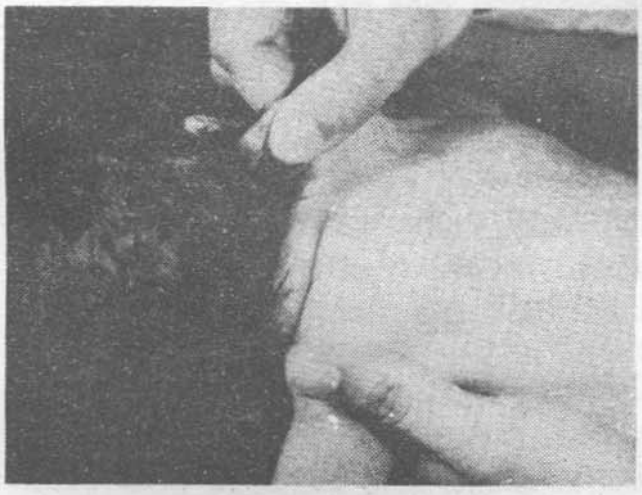

Fig. 2. Clinical photograph showing webbing of the neck with loose posterior nuchal skin folds.

and antithyroid antibodies. Urinary estrogen and estriol levels are low. Skeletal survey is required to rule out anomalies like retardation of bone age, short 3rd, 4th and 5th metacarpals, metatarsals with ballooned ends and premature fusion and spur at tibial metaphyses(3). Abdominal ultrasonography is necessary to rule out renal anomalies.

Treatment of the patient consists of psychotherapy, hormonal therapy(4) and treatment of associated conditions. After 12 years of age ethinyl estradiol 10 μg/day can be given from day 0-20 followed by norethisterone 5 mg from day 20-30. Subcutaneous hormonal implants and a combination of anabolic steroids (oxandrolone) and growth hormone have been tried(4). If the patient is 45X/46XY mosaic, the gonads must be removed to prevent the risk of gonadal neoplasia(5).

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Congenital Lymphangiomatosis of Greater Omentum

Omental cysts have been reported occasionally in the Western literature and sporadically from India, but congenital lymphangiomatosis of greater omentum is a distinct rarity(1-6). We wish to report a case having interesting clinical course and presenting as a case of intractable loculated ascites.

A 3-year-old male child had progressive enlargement of abdomen since birth. His father had open pulmonary tuberculosis. At the age of 15 months he was taken to a family physician who noted a cystic mass occupying mainly the right side of abdomen. After abdominal paracentesis, he was diagnosed to have loculated tuberculous ascites. He was put on isoniazid, rifampicin and ethambutol for one year. While on treatment, he had abdominal paracentesis twice.

However, the mass reappeared again and parents took him to an ESIS hospital. He was put on second course of antitubercular drugs for about 10 months with intermittent diuretics and aspirations of loculated abdominal mass repeatedly without apparent relief. he had also received all kinds of antimalarials and antibiotics and for associated fever with rigors. He was referred to several General Surgeons who preferred to treat him conservatively.

The protein content of the aspirated fluid varied between 14 to 22 g/L in initial samples and between 30 to 60 g/L in subsequent samples. Cytological examination had shown persistent RBCs and polymorphonuclear cell counts which varied from 50 to 720 cells/cumm.

He developed persistent pyrexia and features of subacute intestinal obstruction and was, therefore, referred to us for further management. On examination, he was malnourished, anemic, cachexic and had high grade fever. The abdomen was protruberant containing tender, lobulated, cystic, ill defined, relatively fixed masses occupying nearly whole of the abdomen with fullness of the flanks and marked widening of upper abdomen. These multiple irregular intra-abdominal lumps of various sizes were dull on percussion.

Laboratory investigations showed anemia, hypoproteinemia, polymorphonuclear leucocytosis, high ESR and negative Mantoux test. Chest *X*-ray showed marked elevation of both domes of diaphragm. Plain *X*-ray abdomen showed ground glass appearance with a few loops of intestine in left upper abdomen and gross widening of upper abdomen. Ultrasound showed multiple cystic lesions, some of them were clearly echoluscent especially near both flexures of the colon and others showed mixed echogenicity (*Fig.*) The cysts were situated just beneath the anterior abdominal wall pushing the intestine posteriorly. This was confirmed by lateral films of *X*-ray abdomen and barium meal study which was essentially normal.