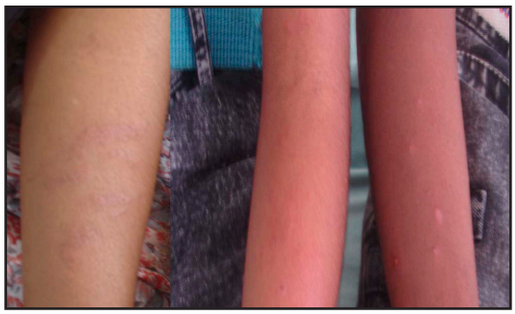
SPOT DIAGNOSIS (IMAGE GALLERY)



Pruritis in all family members

Kalyani Pillai, Sandhya Acharya
Department of Pediatrics, Department of Dermatology, Amala Institute of Medical Sciences, Thrissur, Kerala, India

Address for Correspondence: Dr Kalyani Pillai, Santhwanam, Mallika Garden, Chittilappilly P O, Thrissur, Kerala 680551. India. Email: pillaiskpillai@dataone.in

A 7 year old boy was brought to the pediatric dermatology clinic with intense pruritic lesions over forearm, neck, legs {exposed areas} since 1 week. There is history of similar lesions in mother and sibling. The lesions were linear, vesicular, oozing. On enquiry, all of them were involved in stacking up of firewood one week back.

What is the diagnosis?

Phytodermatosis. It is an allergic contact dermatitis commonly missed and rarely diagnosed. Plants contain some oily chemical resin that is absorbed quickly into the skin. The eruption is characterized by redness, papules, vesicles and linear streaking. The eruption usually appears within two days but may occur within eight hours and rarely is delayed longer than ten days. The rash will frequently break out in stages depending on the length of exposure and the amount of resin on the skin. Treatment depends on how severe the reaction is and is usually symptomatic

Authors contribution: KP researched the article and drafted and wrote the manuscript. SA provided the clinical material and reviewed the manuscript.

Acknowledgement

The authors acknowledge the help rendered by Dr S Criton, Professor of Dermatology and Dr V K Parvathy, Professor of Pediatrics in diagnosis of the case and in publishing this article.

E-published: April 2010 . **Art**#25

Abdominal defect

Sanjeev Kumar Digra, Niraj Kumar, Pankaj Kumar Gupta, Varun Kaul
Department of Pediatrics and Department of Anesthesia, Government Medical College, Jammu

Address for Correspondence: Sanjeev Kumar Digra, Aayu Shree Child Care Centre, Pragati Nagar, Upper Barnai, Jammu, {JandK} 181124, India. Email: sanjeevahsaas@yahoo.co.in

What is the diagnosis?

Gastroschisis. This is a low birth weight neonate with gastrochisis who was born with herniation of small intestine from a small defect on the right side of umbilical cord with no covering membrane. Gastroschisis is a Greek term which stands for belly cleft. It is the herniation of abdominal contents through an abdominal wall defect, usually occurring on the right side of a normally positioned umbilical cord {1}. The defect is most often less than 4cm and as with the other abdominal wall defects, the rectus muscles are normally formed {2}. The prevalence of gastroschisis is about 1, 7000 total births and both sexes are equally involved {3,4}. Young maternal age {median age 21 years} is consistently associated with this condition and the neonates are typically of low birth weight {median weight 2.3kg} {3,4}. This defect is commonly believed to be a result of a vascular accident that leads to intrauterine interruption of omphalomesenteric artery {1}. This anomaly occurs early in gestation and bowel is left in contact with the amniotic fluid, which produces an intense inflammatory response or "Peel" causing poor intestinal motility {1,5}. Unlike exomphalos, there is no covering membrane and the extent of evisceration is variable which may include stomach, small bowel, colon, and ovary and fallopian tubes or testes.

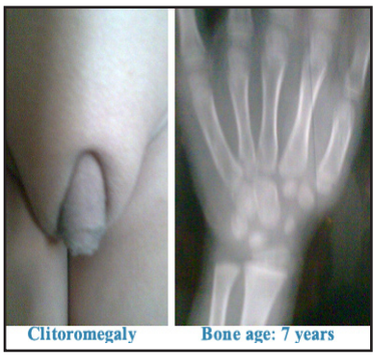
SPOT DIAGNOSIS (IMAGE GALLERY)

Liver is rarely involved. The incidence of associated anomalies in patients with gastroschisis is relatively infrequent. Intestinal atresia may occur in 10-15 percent of cases {1,4}. Maternal serum alpha- fetoprotein {AFP} is used as screening test although there is a 40 percent rate of false positive results. It is elevated in case of neural tube defects, abdominal wall defects, or atresia of duodenum or oesophagus. Analysis of amniotic AFP and acetylcholinestrase-pseudocholinesterase can be sensitive in detecting abdominal wall defects, especially Gastroschisis. The ultrasonography is helpful beyond 14 weeks {because midgut normally is herniated in 1st trimester} {1}. Unlike with Omphalocele, primary closure is possible in 90 percent patients, but larger defects may require staged repair. Mortality rates have decreased to 5 percent to 10 percent. Enteral feedings may not be established until 2 months after operation {1}.

REFERENCES:

1. Berseth CL, Poenaru D. Abdominal wall problems. In : Taeusch HW, Ballard RA, Gleason CA {eds}. Avery's Diseases of the Newborn. 8th Edn. Elsevier, Saunders, Philadelphia; 2005: 1113-1117.
2. Klein MD. Congenital Abdominal wall Defects. In: Ashcraft KW, Holcomb GW, Murphy JP {eds}. Pediatric Surgery. 4th Edn. Elsevier, Saunders, Philadelphia, 2005: 665-669.
3. Curry JI, McKinney P, Thornton JG, Stringer MD. The aetiology of gastroschisis. BJOG. 2000: 107: 1339-1346.
4. Stringer MD, Sugarman I, Smyth AG. Congenital Defects And Surgical Problems. In: Rennie MJ {editor}. Roberton's Text Book of Neonatology. 4th Edn. Elsevier, Churchill Livingstone, 2005: 733-737.

E-published: May 2010. **Art**#31



Four Year Old Child with Clitoromegaly

Vaswani ND, Lekhwani S***
**Department of Pediatrics and **Biochemistry, PGIMS, Rohtak, India*

Address for Correspondence: Dr N D Vaswani, 55, 9J medical campus PGIMS ROHTAK, India. Email: dr_vaswani@yahoo.co.in

A four year old female child born from non consanguineous marriage (weight 15 kg and height 102 cm with upper segment, lower segment ratio 1:1) presented with clitoral enlargement in pediatric out-patient department of tertiary teaching care hospital in Haryana. According to parents, problem was present since birth but has increased from past one year. No other physical abnormality was detected in that child. On that basis diagnosis of clitoromegaly was suspected. Blood investigations revealed raised levels of testosterone (1.58 ng, ml), and 17 a-hydroxy progesterone (223.10 ng, ml) with serum electrolytes within normal limits. FSH was raised (3.44 mIU, ml) while LH was normal. Thyroid function tests were normal. Karyotyping revealed female chromosomal complement with no numeric or structural anomalies (46 XX). Magnetic resonance imaging of pelvis showed a small uterus and hypoplastic vagina. No definite testicular tissue was identified. There was prominence of ischiocavernosus and bulbospongiosus muscles forming a penile like structure with urethral meatus at the base of structure. Bilateral adrenal prominence was noted. X-ray wrist revealed increased bone age (7 years).

What is the diagnosis?

Clitoromegaly, otherwise a rare condition, if presents in childhood usually is attributed to congenital adrenal hyperplasia (CAH) in which adrenal gland produces additional androgens and 21 a-hydroxylase deficiency accounts for 95 percent of diagnosed cases. (1) This was a case of simple virilizing CAH with some residual activity of 21-hydroxylase, (2) hence mineralocorticoid deficiency was insignificant and salt wasting did not occur. Adrenal testosterone production was suppressed by glucocorticoid like hydrocortisone.

References

1. Nimkarn S, New MI. Congenital adrenal hyperplasia due to 21-hydroxylase deficiency: A paradigm for prenatal diagnosis and treatment. Ann N Y Acad Sci. 2010; 1192: 5-11
2. Nimkarn S, Lin-Su K, New MI. Steroid 21 hydroxylase deficiency congenital adrenal hyperplasia. Endocrinol Metab Clin North Am. 2009. 38: 699-718

E-published: June 2010. **Art**#37