ARTHRITIS IN LG

manifestations of lymphomatoid granulomatosis.


overlying the spine makes diagnosis on IVP and angiography difficult. WT in a HSK poses an operative challenge to the surgeon. HSK's are normally situated lower than normal kidneys and have an anomalous blood supply. Generally 4-6 renal arteries supply the HSK; 2 to each hilum and 1-2 to the isthmus. The blood supply to the hilum of the kidney may arise from the renal artery, directly from the aorta, the inferior mesenteric artery or the iliac artery. Preoperative arteriography can thus facilitate surgery. Both ureters take an unusual course due to the abnormal position of the renal pelvis which predisposes them to inadvertent operative injury. The presence of blastemal cells with epithelial cells and stroma allows a diagnosis of WT by FNAC. If the tumor involves one kidney in a HSK, the functional isthmus has to be resected along with the tumor, lest a urinary fistula result. If the tumor arises from the isthmus, isthmusectomy with bilateral lower pole heminephrectomy is needed.

Owing to the position of HSK overlying the spine, it is readily accessible to physical examination. We emphasize a strong clinical suspicion and regular periodic abdominal examination to identify early stage WT in a child with HSK.

REFERENCES


LETTER TO EDITOR

WILMS' TUMOR ARISING IN A HORSESHOE KIDNEY

Sir,

Wilms tumor [WT] is the commonest malignancy in children. It is associated with multiple congenital anomalies and malformations, commonest being aniridia, hemihypertrophy and genitourinary anomalies. Although the overall incidence of malignancies arising in a HSK is not increased, the risk of developing WT increases many folds compared to general population. The association of extrarenal WT and renal carcinoid tumor with HSK is now known.

A two and a half year old male child was brought with history of progressively enlarging abdominal lump for 2 months. Examination revealed a large firm mass predominantly in the right lumbar and hypochondrium. Intravenous pyelography [IVP] was not diagnostic of HSK. CT scan confirmed a HSK with an isthmus at the lower pole and a mass arising from the non functioning right kidney. Fine needle aspiration cytology [FNAC] showed WT. A right radical nephrectomy with isthmusectomy was performed. Histology confirmed stage 1 multilocular cyst variant. Patient was given Actinomycin D and Vincristine for 24 weeks with an uneventful post operative recovery.

HSK was not recognized preoperatively in almost a third of patients in the National Wilms tumor study. Though CT scan is a reliable investigation, the position of HSK in the midline


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CEFTRIAXONE INDUCED CHELOSTASIS IN A NEONATE: A CASE REPORT

Sir,

Ceftriaxone is a third generation cephalosporin frequently used in clinical practice for various infections in all age groups. However, it is associated with life threatening complications such as anaphylaxis and potentially dangerous complications like cholestasis and hypoprothrombinemia. We report a case of Ceftriaxone induced reversible cholestasis in a neonate.

17-days-old male infant was referred to our hospital with direct hyperbilirubinemia after being treated with Ceftriaxone 50 mg/kg/dose twice daily and amikacin 7.5 mg/kg/dose twice daily for 7 days for suspected sepsis on day 9 when presented with lethargy. This neonate was born at term after a normal pregnancy by normal spontaneous vaginal delivery and weighed 2700 grams. He had received 1 mg of vitamin K intramuscularly at birth. The neonate was well until day nine when presented with lethargy and refusal of feeds. All the routine markers of sepsis were negative including blood culture and cerebrospinal fluid analysis. He received a compatible blood transfusion during his hospital stay for blood stained gastric aspirate. Clinically the baby improved after five days of antibiotics although he developed progressive icterus and high coloured urine. He was then referred to our hospital for the management of his progressive jaundice.

On examination, the infant was icteric. There were no bleeding tendencies, no gastric aspirates. Liver was palpable 2 cm below the right costal margin with a span of 6 cms. There was no splenomegaly or any other masses in the abdomen. Urine was high coloured and stools were normal. His haemoglobin was 9.8 gm/dl. Direct Coomb’s Test was negative which ruled out mismatched transfusion. C-reactive protein level was 24 mg/dl (Normal <6 mg/dl). Aspartate transaminase was 118 units (Normal 15-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds.