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Gastric Signet Ring Cell Carcinoma presenting as Obstructive Uropathy: A Diagnostic Conundrum

Editor : Prof Nandita Kakkar[1]
Chairperson : Prof Subhash C Varma[2]
Pathology Discussant : Dr Balan Louis Gaspar[3]
Clinical Discussant : Prof SK Singh[4]
Radiology Discussant : Dr Anupam Lal[5]
Clinician Incharge: : Prof KL Gupta[6]

This case was discussed on 1st October 2014 as a staff clinicopathological exercise at Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India.

CLINICAL PROTOCOL: PROF SK SINGH

A 70-year-old gentleman, retired clerk and resident of Bihar presented with nausea, vomiting, giddiness, hesitancy and poor urine stream for 3 months. Loose stool, abdominal distension and altered sensorium were present since 10 days. The vomiting was 2 to 3 episodes per day, non-projectile, yellowish, non-foul smelling containing ingested food particles. There was no history of frequency, urgency, dysuria or hematuria. Patient was initially managed conservatively in a local hospital in Bihar as a case of gastroenteritis. His symptoms worsened and he developed altered sensorium. He was referred to another hospital, where his serum creatinine was found to be elevated from 0.9 to 3.6 mg/dl and bilateral hydronephrosis was detected on ultrasonography (USG). A detailed history of treatment was not available. He was then referred to the nephrology services in postgraduate student (PGI). There was no history of diabetes, hypertension or tuberculosis. He was a known case of seizure disorder since childhood and was on regular sodium valproate treatment. The patient had skipped his medication for the previous 2 days. On examination, there was pallor, pedal edema and facial puffiness. Pulse rate was 96/min, BP was 100/70 mm Hg and respiratory rate of 18/min. Respiratory system examination revealed bilateral coarse crepitations. Central nervous system examination revealed a Glasgow coma scale of 15 no. neck stiffness or any focal neurological deficit. Examination of the abdomen revealed increased abdominal girth with free fluid. Cardiovascular system examination was within normal limits. Digital rectal examination revealed grade 1 prostatomegaly. Throughout the course of the hospital stay, polymorphonuclear leukocytosis was seen. Electrolytes were normal. Serum alkaline phosphatase was raised but the other liver function tests were normal. Ascitic fluid serum-ascites allumin gradient (SAAG) was 0.8 and adenosine deaminase (ADA) was 12. The cerebrospinal fluid (CSF) examination was within normal limits. Pleural fluid cytology was suspicious of malignancy. Serum PSA was 3.8 ng/mL. Abdominal fat pad was negative for amyloid. Serum protein electrophoresis did not reveal any M-band. Ultrasound revealed bilateral hydrouteronephrosis with median lobe enlargement of prostate, normal liver and spleen. Contrast enhanced computer tomography (CECT) revealed multiple enlarged hilar lymph nodes, left pleural effusion with massive collapse and multiple sclerotic bony lesions. Non-contrast computerized tomography (NCCT) kidneys, ureters and bladder (KUB) showed globular kidneys with hyperdense contents within the left pelvicalyceal system suggestive of pyonephrosis/infected hydronephrosis. Patient was treated with antibiotics and his diarrhea improved

[1,4]Professor, [2,6]Professor and Head
[3]Senior Resident, [5]Associate Professor

[1,3]Department of Histopathology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[2]Department of Internal Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[4]Department of Urology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[5]Department of Radiodiagnosis, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[6]Department of Nephrology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

Corresponding Author: Nandita Kakkar, Professor, Department of Histopathology, Postgraduate Institute of Medical Education and Research, Chandigarh, India, e-mail: nandita_kakkar@yahoo.com