

Deadly fluids – hereditary diffuse gastric cancer presenting with ascites

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Aims

Hereditary diffuse gastric carcinoma (HDGC) is a rare and aggressive form of gastric cancer due to a mutation in the *CDH1* gene. Of all ascites cases, 10% relate to malignancy and it may be the only presenting symptom in abdominal malignancy. Our aim was to present the clinical and historical evidence that would warrant a high index of suspicion for HDGC, particularly with ascites as the primary presentation.

Methods

The clinical data of the patient under study was analysed. A review of the available literature was then conducted over a 2-week period using the keywords 'ascites', 'ascites diagnosis', 'theories of ascites development', 'malignancy related ascites', 'gastric cancer' and 'hereditary diffuse gastric cancer'. Articles were eliminated based on relevance to the case and reliability of the source.

Results

A 19-year-old male with no known chronic illness had a 4-week history of progressively increasing abdominal girth and abdominal pain associated with nausea and anorexia. One week prior to presentation he had shortness of breath, dry cough, and pleuritic chest pain. Three days prior to presentation, he passed two to three loose stools daily. He had no other notable symptoms. He had a family history of hypertension, gall bladder cancer and breast cancer and no history of alcohol, smoking, drug use, sexual partners nor tattoos. Vitals were normal. On examination, he was found to have a firm, globally distended abdomen with periumbilical and suprapubic tenderness and fluid thrill. Free fluid in the abdomen and pelvis was confirmed on ultrasound and computed tomography (CT). Ultrasound-guided aspiration of abdomen confirmed mild ascites with normal portal venous flow. Liver span was 16 cm with a normal echo pattern on ultrasound. Blood work was normal except – white blood cell (WBC) count of 13.74 K/ml (neutrophils = 82.5%, lymphocytes = 10%), platelets of $506 \times 10^9/L$, erythrocyte sedimentation

rate (ESR) of 96 mm/hr and lactate dehydrogenase (LDH) of 640 U/L. Urine dipstick was normal. Paracentesis produced a slightly cloudy, dark yellow fluid. The fluid WBC count was 837/ μ L (41% neutrophils, 14% lymphocytes), fluid albumin was 34 g/L, no organisms seen on gram stain and serum-ascites albumin gradient (SAAG) was 0.9 g/dL. Cytology of fluid detected no malignant cells.

On day 2 of admission he had two emetic episodes with significant tenderness and guarding. He presented with tachycardia and a temperature of 38.5°C. Blood and urine cultures were negative. Repeated paracentesis showed a total protein of 53 g/L, LDH of 439 U/L, and glucose of 3 g/dL. Abdominal ultrasound showed no changes. CT abdomen showed no evidence of bowel perforation. Collagen vascular disorder screen was normal. C13, venereal disease research laboratory and Mantoux tests were negative. Blood C-reactive protein was 13 mg/dL. Upper endoscopy showed poorly differentiated diffuse gastric adenocarcinoma, with histology of biopsy specimen suggestive of HDGC. He was treated with Rocephin 2 g, intravenous once daily.

Conclusion

HDGC represents about 1% of gastric cancer cases. It should be suspected more in males younger than 30 years presenting with abdominal symptoms and first or second degree relatives with breast cancer. Sudden onset of fever, vomiting and changes in vitals are suggestive of systemic inflammatory response syndrome which is related to increased mortality in malignancy. Once more common differentials of ascites are excluded, a complete clinical history is important in diagnosing HDGC. Screening of high-risk individuals is vital for early detection. ■

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