CASE REPORT

A 2 year old previously healthy girl first presented to ED with vomiting for 2 days. The vomiting was non projectile with no blood or bile, no diarrhea, without preceding trauma or fever. She was diagnosed as acute gastroenteritis and discharged home. 12 hours later, she presented again with lethargy and rapid breathing. On further questioning, she had increase in fluid consumption and polyuria since the course of the illness. Initial assessment revealed drowsiness, lethargy, pale, Kussmaul's breathing, moderate dehydration with estimated fluid deficit of 7.5%. She was normotensive with BP 110/60 mmHg (MAP 70), tachycardic (HR 150/minute), SpO2 96% and normothermic (temperature 37.2°C). Her blood glucose level was 37 mmol/L with severe acidosis (venous pH 6.8, HCO3 too low, lactate 2.6, BE -28) and serum ketone 4.7. Full blood count shows leukocytosis with white cell counts of 17.3 (lymphocytes 50%, neutrophils 43%), platelets of 590 and hemoglobin of 12.9 g/dL. Blood urea was high (6.6), however liver enzymes and electrolytes were all within normal limit. Initial saline bolus of 100 ml over the first hour was started, followed by 7.5% correction using 16 ml/hour of saline over 48 hours and maintenance using half saline mixed with 1g KCL at a rate of 40ml/hour. Insulin infusion was started at a rate of 0.1 unit/kg/hour and was transferred to PICU.

DISCUSSION & CONCLUSION

Diagnosing DKA in pediatric patient can be challenging, especially when the staff is unfamiliar with such case as they usually present with vague symptoms without known history of diabetes. This unfortunate case demonstrates the importance of high level of suspicion and considering glucose as the sixth vital sign in sick child.

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SPONTANEOUS PNEUMOPERICARDIUM, CASE REPORT

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INTRODUCTION

Pneumopericardium is defined as collection of air or gas in the pericardial cavity. It is a rare complication of tuberculosis and HIV. Pneumopericardium most commonly results from trauma, approximately 60%, other causes can be due to iatrogenic and noniatrogenic. Development of spontaneous pneumopericardium is a very rare complication of tuberculosis with coexisting HIV infection.

CASE REPORT

22 year old male man with no known medical illness, and not obese. P presented to Emergency Department due to with on and off pleuritic chest pain for the past 2 weeks, associated with mild dyspnea. Pain is was relieved by rest and leaning forward. On examination patient was alert, pink and not tacypneic. Vital signs was were stable, the lungs were clear, equeal air entry, no hyperesonance, CVS no heart murmur and no pericardial rub heard, per and the abdomen was soft and non tender. Chest xray erect done showed lucencies around the right and left heart border suggestive of pneumopericardium, with no pneumothorax or mediastinal mass. The ECG, showed sinus rhythm with no

acute ischaemic changes. Patient remained stable and was admitted to the ward for further investigation.

DISCUSSION AND CONCLUSION

Spontaneous pneumopericardium is a very case rare. Cases that have been reported usually are related to tuberculosis and immunocompromised patients, which is not in this case. The pathogenesis of pneumopericardium is increase in intra-alveolar pressure with alveolar overdistention that results in rupture of alveolar walls, allowing air to travel through the pulmonary interstitium along perivascular sheaths to the lung hilum and mediastinum and to the pericardial reflection. Pericardial connective tissue is discontinuous at the reflection of parietal onto visceral pericardium near the ostia of the pulmonary veins so that there is a site of potential weakness where a microscopic dissection of air into the pericardial sac is possible. It is potential that in this case it is related to vasalva maneuver with forced expiration against a closed glottis in exertional activity.

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SUBGALEAL ABSCESS: A CASE OF UNFORTUNATE INMATE

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Almost all causes of scalp abscess is are due to direct introduction of microbes into the subgaleal space after disruption of skin barrier (laceration, or puncture wounds). Infected scalp hematoma without interruption of skin barrier is rarely reported. Mr S, 35-year-old IVDU was brought by the prison officer with a complaint of worsening right sided scalp expanding hematoma for more than a month. He was assaulted with a blunt object prior to that and did not seek any medical treatment up until the current visit. He denied of any external wound or bleeding from the site during the attack. Other than the pain over the scalp, he did not have fever, loss of appetite or change in behavior. Computer tomographic scan showed right subgaleal collection with extradural extension and right temporal bone fracture. He was referred to neurosurgical team and subsequently a craniectomy, exploration and evacuation of abscess was done. Intra operatively, the temporalis muscle was unhealthy with sloughy dura. He was started on antibiotic and discharged home well.

The diagnosis and treatment of subgaleal abscess are often not complicated. Nevertheless, this condition is usually affiliated with extended morbidities such as seizure or sepsis and diagnosis might be missed initially. High level of suspicion of an abscess is especially suspected in the immunocompromised with non resolving and expanding hematoma. Identification of this condition is usually made with history of prolonged swelling, increased inflammatory markers and a computer tomography scan that showed subgaleal collection. The recommended treatment is surgical incision and careful debridement followed by appropriate systemic antimicrobial therapy for a week with continuation of oral therapy for another week.