Sir,

A 33-year-old female presented with right-sided pleuritic chest pain and expectoration of mucopurulent, foul-smelling, and salty sputum for one year. There was a history of close contact with a pet dog for last two years. General survey and systemic examination revealed no abnormality. Absolute eosinophil count was 800/cmm. Sputum microscopy for acid fast bacilli was negative. A homogenous opacity in right lower lobe was seen in chest radiogram [Figure 1]. Contrast enhanced computed tomography (CECT) scan of thorax showed two rounded, heterogeneous opacities (1 × 1 cm and 1.5 × 2 cm, respectively) with presence of air pockets within the lesions—the “air – bubble sign” [Figure 2]. Ultrasonography (USG) of thorax revealed loculated collection of fluid with echogenic organization inside. USG-guided aspiration of the lesion revealed viscid material. Cytology of the smear showed fragmented membranous structures with lamination along with aggregates of degenerated inflammatory cells in a background of proteinaceous fluid [Figure 3]. Fiberoptic bronchoscopy did not reveal any endobronchial lesion. USG of whole abdomen did not reveal cyst in intra-abdominal organs. Serum anti-echinococcal IgG level was 26.41 U/ml (normal value: <8 U/ml, ELISA method). A diagnosis of infected hydatid cyst in right lung was made and albendazole tablet, 400 mg twice daily, was started. The patient was referred to the department of cardiothoracic surgery for further management and follow up.

“Air – Bubble” Sign – An Uncommon Presentation of a Common Disease

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Echinococcosis, a zoonosis caused by *Echinococcus granulosus* is endemic in India, mostly involving liver and lungs. Most pulmonary cysts are asymptomatic and become clinically evident as a result of complications like cyst rupture and secondary infection. Pulmonary cysts are characteristically solitary and involve single lobe, mainly the lower lobe, and more common on the right.\(^1\) Structurally, the cysts consist of pericyst, exocyst, and endocyst from outside inwards.

Echinococcal cysts carry high risk of rupture with subsequent seeding to the other viscera\(^2\) and secondary infection. The rupture may lead to anaphylactic reaction.\(^3\) Most common respiratory symptoms of ruptured hydatid cyst are cough with fetid expectoration.\(^4\) CECT scan of thorax is the diagnostic modality of choice for pulmonary hydatid cyst. Most of intact pulmonary cysts present as solid lesions of fluid density on CECT thorax. But ruptured cysts almost always present with a variety of radiological appearances due to different combinations of collapsed membrane, air, and fluid, making its diagnosis difficult. Sometimes, secondary bacterial infection supervenes on the ruptured cyst with subsequent increase in attenuation numbers in CECT thorax.\(^5\) Because of solid density of the cyst, the differentiation from a pulmonary neoplasm is usually impossible. Serological test like anti-echinococcal IgG is helpful in most patients for final diagnosis, but measurable immunological response may not develop in few patients.

Pulmonary hydatid cyst may rupture through pericyst only or through pericyst, exocyst, and endocyst with expulsion intracystic contents within airways. A number of radiological signs of ruptured cysts are described which include crescent sign, water lily sign, daughter cyst, double arch sign, ring within a ring sign, serpent or snake sign, and spin or whirl sign.\(^6\) When there is dissection of air between the pericyst and parasitic membrane, due to erosion of a bronchiole by an expanding cyst, “airbubble” sign is seen. The “air bubble” sign which is a relatively newly recognized radiological sign is reported to be very sensitive and specific (85.7% sensitivity and 96.6% specificity) in establishing diagnosis of ruptured, infected hydatid cyst.\(^6\) Air bubble sign is best seen in mediastinal window as single or multiple small, rounded radiolucent areas with sharp margins within the periphery of a solid mass lesion.

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Letter to Editor

Sir,

Auto urine therapy (AUT) is practiced worldwide either as prophylaxis or as a therapy in complementary and alternative medicine[1] for many diseases. Though many cases of AUT are known, urine-induced sepsis is not reported yet to our knowledge. Herein, a case of near fatal polymicrobial sepsis with acute respiratory distress syndrome due to self injection of urine is reported for its rarity and to create awareness among physicians working in emergency care.

A 38-year-old livestock inspector was brought to the emergency room (ER) in an unconscious state following two episodes of a witnessed new-onset generalized tonic-clonic seizure (GTCS) within a period of 30 minutes. There was a history of fever associated with chills and rigors since 12 hours. The patient had no previous history of seizures, head injuries, or any other illnesses and was not on any regular medications or illicit drugs. Family history was not contributory.

On admission, he was unconscious with a Glasgow coma scale (GCS) of 8/15 and the pupils were 4 mm dilated and sluggishly reacting to light. There were no meningeal signs or rashes. He was febrile (104 F) and had frothy blood stained secretion in the oropharynx. He was tachycardic (pulse 128/minute) with supine BP 90/60 mmHg, SaO2 80%, and FiO2 0.9. He had shallow respiration (rate 11/minute) with bilateral coarse rales extending up to apices of both lungs. The rest of the clinical examination was unremarkable.

Arterial blood gas (ABG) revealed wide anion gap with metabolic acidosis and hypoxia. The total WBC was 36.0×10³/mm³ with 41% band forms and neutrophils 25.4×10³/mm³. Hemoglobin, packed cell volume (PCV), platelet count, electrolytes, liver function test, coagulation profile, capillary blood sugar and CSF were normal. His HIV status and peripheral smear for malaria were negative. Blood and urine were negative for toxicology or for any drugs. His ultrasound abdomen, CT brain, and ECG were unremarkable. The chest X-ray showed extensive bilateral alveolointerstitial infiltrate compatible with acute respiratory distress syndrome (ARDS). He was started on anticonvulsants and intravenous broad-spectrum antibiotics in Intensive care unit (ICU). Inotropic support was started to maintain blood pressure. He was stabilized in the following 48 hours. His blood cultures grew K.pneumoniae, E. coli, and Proteus, which were sensitive to imipenem. Antibiotics were changed as per culture and sensitivity pattern. The sepsis started subsiding gradually and patient got discharged on day 12.

On day 11, he confessed that he collected his urine in a container and self injected about 10 ml of his urine intravenously to maximize his vitality and potency, as he had developed nausea and vomiting twice after drinking his urine orally. This might have led to polymicrobial sepsis, toxic encephalopathy, and septic shock with multi organ dysfunction. Psychological assessment did not reveal any abnormalities.

Drinking one’s own urine in the morning (“amaroli”) is a traditional practice in yoga as well as “Siddha medicine” a branch of complementary and alternative medicine taught officially at some Indian medical universities[2]. It recommends drinking one’s own urine for prophylactic and therapeutic purposes. Oral urea and methylglyoxal, both derivatives of urine have anticancer properties[3,4]. Although urine therapy might have its own benefits, its practitioners and followers should exhibit caution. Also, when a clinician is confronted with challenges of polymicrobial infection, in an immunocompetent individual, history of an unconventional cause either deliberate (Munchausen’s) or as unconventional methods needs to be elicited.