Sir,

Paraneoplastic syndromes are disorders seen along with benign or malignant tumors but are not directly related to invasion, mass effect, or metastases. Commonly, paraneoplastic endocrine syndromes are detected in patients after the diagnosis of cancer. In certain cases, they may also be the first sign of an underlying malignancy, and hence, early detection is essential for successful treatment of tumor.[1] Although adenocarcinoma is the most common histological type in lung carcinomas,[2] its presentation with a paraneoplastic syndrome is rare.[3]

We report a rare case of a patient with pulmonary adenocarcinoma who presented with symptoms of ectopic adrenocorticotropic hormone (ACTH) production instead of respiratory symptoms.

A 51-year-old male, manual laborer by occupation, presented with complaints of palpitations, dull aching pain in both legs, and blackish discoloration of both hands and feet for 1 year. There was no history of cough, hemoptysis, chest pain, or dyspnea. He was diagnosed with systemic hypertension 8 years back and type 2 diabetes mellitus 4 months back. He has been a chronic smoker for the past 30 years.

The patient had undergone a chest radiograph posteroanterior view for palpitations 6 months back which revealed a well-defined smoothly marginated opacity in the right lower lobe. Contrast-enhanced computed tomography (CT) scan taken elsewhere revealed a mildly enhancing smoothly marginated soft-tissue density lesion in the right lower lobe without calcification, fat density, or mediastinal lymphadenopathy, and a diagnosis of sequestration was made. Histopathological study was done, but the results were inconclusive.

General physical examination revealed clubbing and hyperpigmentation of both hands and feet. There was Grade 5 power in all the limbs. Laboratory investigations revealed hypokalemia (1.3 meq/L), metabolic alkalosis (pH = 7.52, serum bicarbonate = 39.8 mmol/L), hyperglycemia (fasting blood glucose = 308 mg%), elevated serum cortisol levels (75 mg/dl, normal reference range 7–23 mg/dl), and high serum ACTH (496 pg/ml, normal 0–46 pg/ml).

Skull radiograph lateral view taken to rule out pituitary tumor causing raised ACTH values did not show widening or erosion of sella turcica [Figure 1a]. Contrast-enhanced CT scan of the brain was done, but there was no pituitary mass or sellar erosion [Figure 1b-d]. Therefore, ectopic ACTH production was suspected. Dexamethasone suppression also did not cause any fall in the cortisol levels.

As small cell carcinoma of the lung is the most common source of ectopic ACTH production, a chest radiograph was taken in search of a primary lung malignancy. It revealed a well-defined opacity in the right lower lobe with right paratracheal lymphadenopathy [Figure 2a and b]. Ultrasound scanning of the chest and abdomen was done which showed a well-defined isoechoic lesion measuring 11.3 cm × 8.2 cm in the right lower lobe abutting the liver [Figure 2c]. The right suprarenal gland was enlarged measuring 4.48 cm × 1.87 cm [Figure 2d].

For further characterization, contrast-enhanced CT scan of the chest and abdomen was performed [Figure 3a-e]. A heterogeneously enhancing mass lesion was seen in

Figure 1: X-ray skull lateral view (a) and sagittal reformatted image of computed tomography head (b) showing normal sized sella with no evidence of sellar erosion or ballooning. Contrast-enhanced computed tomography scan of brain coronal (c) and axial (d) images does not reveal pituitary mass lesions
the right lower lobe with invasion of the right descending pulmonary artery, right paratracheal and subcarinal lymphadenopathy, and a mildly enhancing nodule in the left upper lobe suggesting bronchogenic carcinoma Stage IVa (T4 N3 M1a). Both adrenal glands were diffusely enlarged with preservation of normal shape suggestive of bilateral adrenal hyperplasia. Compared to the previous CT scan, the size of the lesion had increased. Now, there is extensive mediastinal lymphadenopathy and an enhancing nodule in contralateral upper lobe. CT-guided biopsy from the right lower lobe mass and histopathological examination identified tumor to be a poorly differentiated adenocarcinoma.

Ectopic Cushing’s Syndrome (ECS) is a paraneoplastic endocrine syndrome manifesting with hypercortisolism, usually seen with carcinoids and small cell lung carcinoma and rarely with nonsmall cell lung carcinoma. Typical cushingoid features include moon face, acne, purple striae, proximal myopathy, peripheral edema, hypertension, hyperglycemia, hypokalemia, and metabolic alkalosis. This classical presentation may be absent in cases of ectopic ACTH production by tumors other than carcinoids because of the sudden onset and aggressive nature. The preferred treatment for ECS includes treatment of the underlying malignancy with inhibition of cortisol secretion. Patients with ECS have poor prognosis and survival rate which is determined by the tumor histology, presence of metastases, and severity of hypercortisolism.

Our patient was started on ketoconazole 200 mg once daily following which he had improvement of symptoms. He also underwent chemotherapy for the primary lung malignancy. While on chemotherapy, he died due to cardiac arrest.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest
There are no conflicts of interest.
Spontaneous rupture of hepatocellular carcinoma with tumor embolism in pulmonary arteries

Sir,

A 68-year-old woman was brought to our emergency department with progressively worsening abdominal pain and abdominal distension followed by sudden onset of shortness of breath. The symptoms started an hour back while she was having her dinner. At presentation, she was diaphoretic with blood pressure of 82/47 mmHg and heart rate of 120. Her abdomen was distended and soft but tender in all quadrants. No past history was available as she was a tourist. Electrocardiography was unremarkable. The chest radiograph revealed bilateral pleural effusion. She was resuscitated with fluids, and an urgent computed tomography (CT) of the abdomen was performed. CT abdomen in the arterial phase revealed a large heterogeneous subcapsular exophytic mass in the right lobe of the liver [Figure 1a-d] actively bleeding into the peritoneal cavity. There was large amount of high-density fluid representing hemoperitoneum. In addition, there were expansive thrombi in the middle hepatic vein and left hepatic vein, extending into the inferior vena cava. Careful evaluation of the partially included images of the lower thorax showed large filling defects in bilateral lower lobe pulmonary artery, consistent with pulmonary emboli. The liver itself was small and shrunken with large splenorenal collateral vessels suggesting the background of cirrhosis with portal hypertension. Based on CT findings, a diagnosis of ruptured hepatocellular carcinoma was made, and the patient was immediately transferred to the interventional radiology department. The right hepatic artery was cannulated and embolized with polyvinyl alcohol particles.

Spontaneous rupture of HCC is a well-known complication. To the best of our knowledge, this is the first case of HCC with spontaneous rupture in the peritoneal cavity and embolization in the pulmonary arteries at the same time. Transarterial embolization is accepted as an effective and lifesaving procedure to achieve hemostasis in cases of ruptured HCC.

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