

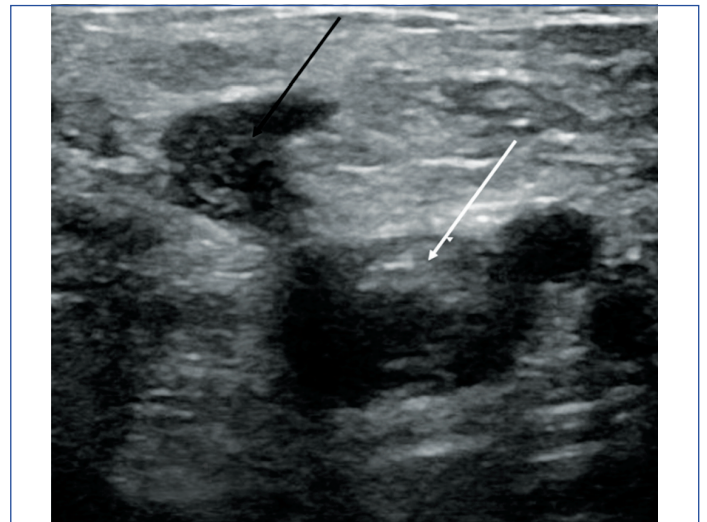
# Imaging Findings of Macronodular Adrenal Hyperplasia

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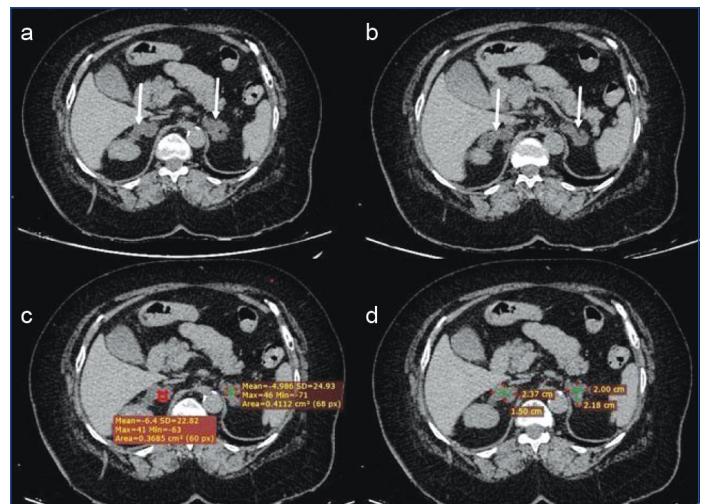
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A 67-year-old female presented with complaints of pain and difficulty while walking for three days. There was no history of trauma, fever, chest pain, cough, breathlessness, abdominal pain, nausea or vomiting. Family history and personal history were unremarkable. On examination, the patient was conscious and oriented. No pallor, icterus, cyanosis, clubbing or significant lymph node enlargement was noted. Vitals: temperature: afebrile, Pulse rate: 70/min, Blood Pressure (BP): 140/80 mmHg, and SpO<sub>2</sub> of 98% on room air. Systemic examination was regular. On local examination, diffuse non-pitting oedema was present extending from the left foot to the knee joint. Calf tenderness, erythema and local rise of temperature of left lower limb was present. Laboratory investigations revealed a haemoglobin value of 10.6 g/dL and a total leukocyte count of 10,400/cu.mm, (neutrophils: 67.8%, lymphocytes: 7%), serum creatinine was 1.2 mg/dL. Thyroid and liver function tests were within normal limits. The patient underwent left lower limb venous Doppler, which showed evidence of deep vein thrombosis in left common femoral vein extending across saphenofemoral junction into the great saphenous vein [Table/Fig-1]. Diffuse subcutaneous oedema and thickening of fat planes were noted in the left lower limb. The right lower limb Doppler was normal. CT pulmonary angiogram was done which showed incidental finding of enlarged bilateral adrenal glands in the form of large distinct nodules demonstrates a mean attenuation of -4 to 7 Hounsfield Units (HU) on CT, with no evidence of calcification. Right adrenal gland measured 2.3x1.5 cm and left adrenal gland measured 2x2.8 cm. Both adrenal gland shows homogenous postcontrast enhancement [Table/Fig-2a-d,3a]. There was evidence of small filling defect involving the subsegmental branch of lateral segment of right lower lobe- suggestive of pulmonary thromboembolism [Table/Fig-3b]. Blood investigations including serum cortisol 11 mcg/dL (6-23 mcg/dL), and plasma Adrenocorticotropic Hormone (ACTH) level 20 pg/mL (6-76 pg/mL) was normal. Following the investigations, patient was diagnosed as a case of femoral deep vein thrombosis with incidentally detected bilateral adrenal macronodular hyperplasia. The patient was treated with intravenous anticoagulants and planned for catheter-directed thrombolysis; however, the patient expired after 48 hours due to cardiac arrest. There was no relationship between adrenal macronodular hyperplasia and cardiac arrest, since the hormonal levels were normal and the cause for cardiac arrest was pulmonary thromboembolism.

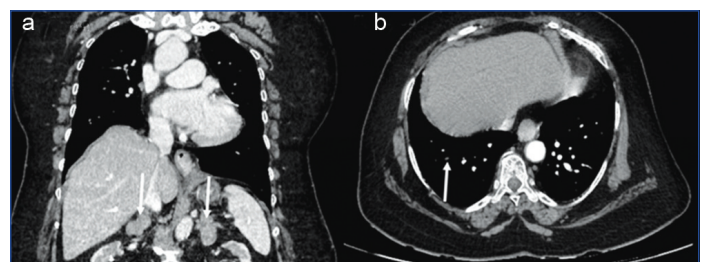
Bilateral macronodular adrenal hyperplasia can have a divergent manifestation ranging from asymptomatic cases, subclinical Cushing syndrome to overt Cushing syndrome with severe complications [1]. The differential diagnoses for bilateral adrenal masses can be metastases congenital adrenal hyperplasia, pheochromocytoma, lymphoma, cysts, infectious causes and haemorrhage [2-5]. In the present case, the masses maintained the shape of the adrenal gland, which is uncommon for metastasis, and no primary malignancy was identified. Congenital adrenal hyperplasia is an autosomal recessive condition characterised by reduced cortisol production due to enzymatic defects in the steroidogenic pathway.



**[Table/Fig-1a,b]:** USG venous Doppler shows echogenic contents within common femoral vein (white arrow) extending across saphenofemoral junction into great saphenous vein (black arrow)- suggestive of deep vein thrombosis.



**[Table/Fig-2a-d]:** CT axial sections demonstrate enlarged bilateral adrenal glands in the form of large distinct nodules (white arrows). The CT attenuation value and size of the adrenal gland are mentioned in [Table/Fig-2c,d].



**[Table/Fig-3]:** a) Contrast CT chest and upper abdomen coronal sections showing bilateral enlarged homogeneously enhancing adrenal glands in the form of large nodules (white arrows); b) CT pulmonary angiogram showing minor filling defect in the right lower lobe lateral basal subsegmental pulmonary artery suggestive of thromboembolism (white arrow).

In the absence of feedback, excessive ACTH secretion leads to increased adrenal androgen production. This condition is most often diagnosed and managed in childhood; thus, it is uncommon as a presentation in adults with newly diagnosed adrenal lesions [6]. Pheochromocytomas, which secrete catecholamines, may present with hypertension, arrhythmia, anxiety, headache, pallor, diaphoresis, and tremor. The classic triad comprised headache, episodic sudden perspiration and tachycardia. Imaging of pheochromocytoma with Computed Tomography (CT) differs from that of adrenal adenomas: unlike adrenal adenomas, pheochromocytomas typically have increased attenuation (mean 35 HU) [6]. Attenuation values and imaging features did not correlate with lymphoma, cysts, infection, or haemorrhage [6]. The present case had no symptoms of Cushing syndrome. Most patients are identified incidentally during imaging studies, such as CT or MRI, for unrelated conditions, as in the present case. The diagnosis of bilateral macronodular adrenal hyperplasia in this case was established incidentally on CT Pulmonary angiogram. The management of bilateral macronodular adrenal hyperplasia involves a series of laboratory investigations-serum cortisol level, blood adrenocorticotrophic hormone, 1 mg overnight Dexamethasone suppression test and urine free cortisol [2]. An unenhanced CT density of 10 HU or less can effectively rule out malignancy, as in the present case. If the hormonal studies are normal, any comorbidities, if present, are medically managed. In patients with an asymptomatic, nonfunctioning bilateral adrenal mass and obvious benign features on imaging studies, no surgery is indicated [3]. Follow-up is indicated unless new clinical signs of endocrine activity appear or there is worsening of comorbidities like

hypertension and type 2 diabetes. Sweeney AT et al., described a similar case of a 35-year-old woman who had bilateral macronodular adrenal hyperplasia found during evaluation for kidney stones [4]. Carlson AL et al., reported a case of a 34-year-old, Caucasian male who was referred to the endocrinology clinic for the assessment of resistant hypertension. A renal artery duplex was done to evaluate for renovascular disease due to patient's young age and hypertension. The duplex demonstrated bilateral adrenal gland enlargement, and MRI of the abdomen showed features of bilateral macronodular adrenal hyperplasia [5]. Bilateral macronodular adrenal hyperplasia, also called adrenal incidentalomas, can be incidentally detected and need to be investigated for evidence of hormonal hypersecretion and/or malignancy.

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