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Case Report

Unexplained Mediastinal Widening and Dyspnoea Revealing Mediastinal Lipomatosis in a Patient with Exogenous Cushing Syndrome: A Case Report

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Introduction

Mediastinal lipomatosis is characterized by accumulation of adipose tissue within the mediastinum in excessive amounts. It is a benign condition and often discovered when a patient presents with unexplained shortness of breath. The common aetiologies are obesity, exogenous steroid abuse, alcoholism and endogenous Cushing syndrome and it rarely can be idiopathic [1].

Cushing syndrome results from excessive circulating glucocorticoids which could be due to excessive adrenal synthesis or exogenous administration leading to systemic effects. Long standing hypercortisolism promotes lipogenesis and adipose tissue accumulation, not only in the subcutaneous tissue but also in visceral compartments [2].

We present a case of dyspnoea and mediastinal widening due to mediastinal lipomatosis in the background of prolonged steroid use leading to exogenous Cushing syndrome. This case highlights an important benign cause for mediastinal widening and emphasizes the role of comprehensive history taking and imaging in averting unnecessary clinical measures.

Case presentation

A 54-year-old, previously unevaluated male presented with shortness of breath of one month's duration. He complained of increased weight gain over the last 3 – 4 months and non-specific joint pain for over one year for which he was taking dexamethasone on and off for symptom relief without proper medical supervision. He was a loud snorer at night

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and has been found dozing off during the daytime, according to the information provided by his wife. On examination, he showed centripetal obesity class 3 with a BMI of 40.4 kg/m^2 with features of hypercortisolism, such as purple striae distributed in the abdomen and axillae with redistribution of fat in the face, neck and upper back and the abdomen, thin extremities with grade 4 proximal muscle weakness on the MRC (Medical Research Council) scale and a diffuse itchy annular rash with an active edge with central clearing resembling a tinea corporis infection.

His haemogram did not reveal any acute infection. His anthropometry and lipid profile were in favour of metabolic syndrome. Despite his features of hypercortisolism, his 9 am cortisol level was very low (0.8 µg/dL), with evidence of a suppressed hypothalamic–pituitary–adrenal axis, supported by a low serum ACTH (adrenocorticotropic hormone) level of <5 pg/mL. His chest X-ray showed mediastinal widening without lung parenchymal changes (Figure 1).

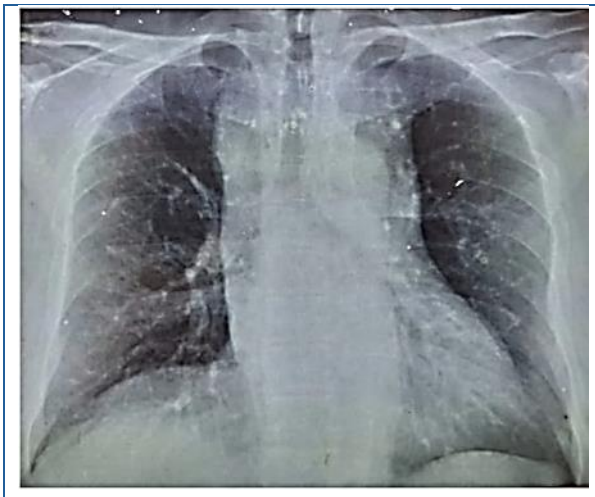
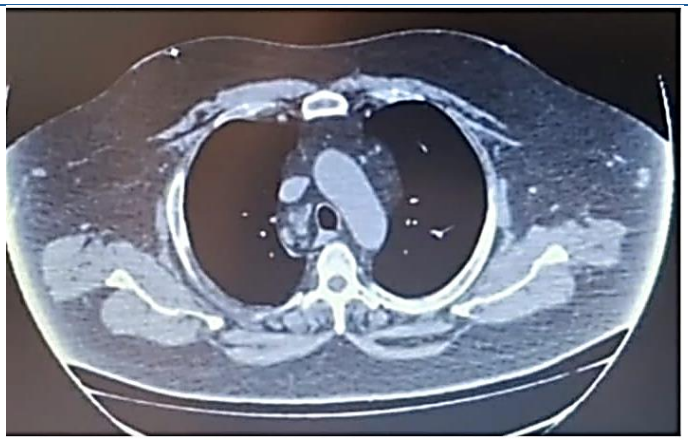


Figure 1: Chest X ray showing mediastinal widening



CECT Chest evidence of mediastinal lipomatosis

His ultrasound abdomen was reflective of fatty liver grade 1 and no adrenal masses were noted. Contrast enhanced computed tomography (CECT) of the chest showed evidence of mediastinal lipomatosis. His non-contrast computed tomography of the brain was unremarkable. Since he showed features of obstructive sleep apnoea on screening, with a score of 7 in the STOP BANG tool and 16 in the Epworth Sleepiness Scale, a sleep study was arranged which showed severe obstructive sleep apnea with nocturnal desaturation qualifying him for continuous positive airway pressure (CPAP). He was managed with a multidisciplinary approach, liaising with the endocrine, nutrition, rheumatology and dermatology team.

Discussion

Mediastinal widening in our patient raised concerns of more sinister causes like malignant space occupying lesions such as lymphoma and thymoma. However, CECT chest, the imaging modality of choice, revealed regions of homogenous unencapsulated fat attenuation involving the mediastinum, sharply demarcating vessels and lymph nodes [3].

Obesity and prolonged glucocorticoid exposure are the primary causes of excessive central fat accumulation. They increase lipoprotein lipase activity, promote adipocyte differentiation, and shift the metabolic balance toward lipogenesis, ultimately leading to mediastinal lipomatosis [1]. Fat redistribution is further facilitated by enzymatic factors such as 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), which converts inactive cortisone into active cortisol, thereby amplifying local glucocorticoid effects [4].

Unexplained shortness of breath in this patient is likely multifactorial. The mass effect from mediastinal lipomatosis may compress the airways and lung parenchyma, leading to a restrictive ventilatory pattern [5]. Long term steroid use can result in proximal myopathy, which may also involve diaphragmatic and intercostal muscles, thereby reducing respiratory effort [6]. Lung expansion and chest wall compliance can be further hindered by centripetal obesity and visceral fat redistribution [7]. Chronic steroid therapy is a major contributor to various cardiopulmonary comorbidities [8]. Moreover, exogenous Cushing syndrome can lead to volume overload, exacerbating dyspnea through pulmonary oedema and pleural effusions in susceptible individuals [9]. Additionally, features suggestive of obesity hypoventilation syndrome may have further implications on the patient's unexplained dyspnoea.

Management of mediastinal lipomatosis warrants a multidisciplinary approach. Gradual tapering of steroids to avoid adrenal insufficiency while allowing regression of lipomatosis should be carefully implemented. Weight reduction and dietary modifications should be adhered to, with close monitoring of cardiovascular health.

Conclusion

Mediastinal lipomatosis is a rare diagnostic possibility to be considered in a case of unexplained mediastinal widening, specifically in patients with Cushing syndrome. This case highlights the need for thorough clinical and radiological evaluation to avoid unnecessary interventions and to ensure optimal management through a multidisciplinary approach.

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