DIAGNOSIS

The thoracic CT scan showed severe tracheal stenosis at the level of the first thoracic vertebra. It also revealed extensive supraclavicular and mediastinal lymphadenopathy, with pleural and pericardial effusions. Histological examination of the supraclavicular lymph nodes revealed non-caseating granulomatous inflammation, with sarcoidosis as the most likely diagnosis. Autopsy revealed concentric web-like tracheal stenosis extending into the right main bronchus, leaving only a pinpoint tracheal lumen.

Causes of tracheobronchial stenosis can be either intrinsic (infectious, non-inflammatory, malignant, and iatrogenic) or extrinsic (compression and infiltrating) disease. Following endotracheal intubation, both direct tissue damage and subsequent high cuff pressure can lead to ischaemia, ultimately causing tracheal necrosis and fibrosis. Therapeutic options include balloon dilations, endoscopic stenting, and laser resection. However, restenosis is common, and surgical resection can be performed when less invasive therapies fail to improve clinical outcomes.

Based on our patient’s medical history, which included recent endotracheal intubation, we speculate that tracheobronchial stenosis developed because of traumatic tracheal intubation and tracheal tube over-inflation with high cuff pressures. Additionally, external compression from enlarged lymph nodes might have contributed to progressive airway obstruction. Lymphadenopathy is common in sarcoidosis, with hilar and/or paratracheal mediastinal adenopathy occurring in up to 90% of patients.

Our case highlights that, in patients with sarcoidosis, symptoms of upper airway obstruction resulting from traumatic tracheal intubation and tracheal tube over-inflation can rapidly worsen. Early balloon dilation and corticosteroid therapy or surgery in such cases may prove beneficial.

REFERENCES