persistent pleural effusion with commencement of prednisolone points to GCA as the primary cause of the pleural effusion.

**Key learning points:** This case shows that GCA can rarely present as cough and pleural effusion which may precede more typical clinical features often associated with GCA.

The importance of recognition of GCA as the cause of pulmonary manifestations in such cases is that appropriate steroid therapy can induce complete resolution and avoid the need for further, potentially harmful invasive investigations.

Our case further highlights the complexity of large vessel vasculitis such as GCA due to their varied presentation and symptoms. In patients with an unexplained weight loss, aside from malignancy the differential diagnoses should include vasculitis.

**Conflicts of interest:** The authors have declared no conflicts of interest.

## 48. DRY COUGH AND PLEURAL EFFUSION AS PRESENTING FEATURES OF GIANT CELL ARTERITIS

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**Introduction:** Giant cell arteritis is a vasculitis which predominantly affects the carotid artery and its extra-cranial branches. Its classical presentation is that of headache with scalp tenderness but should be considered in anyone over the age of fifty years with transient visual symptoms, unexplained facial pain and/or jaw claudication. As a systemic vasculitis, clinical signs may vary, however respiratory complications compared to other vasculitides are uncommon. We present a case of new onset pleural effusion as a presenting feature of giant cell arteritis.

**Case description:** A seventy-year-old man previously fit and well was referred to respiratory clinic after a CXR done for ongoing non-productive dry cough showed a small right sided pleural effusion. Accompanying symptoms included a reduced appetite with around 6kg weight loss over the preceding month.

His past medical history included a TIA, occasional palpitations and prostatectomy for a benign prostatic hypertrophy. His only regular medication was aspirin. He never smoked and was a retired communications engineer with no known history of asbestos exposure.

On examination he had no peripheral stigmata of respiratory disease and chest auscultation was clear. He had routine blood tests which revealed a normocytic anaemia (Hb 108), an elevated CRP (145.6) and elevated ESR (131). LFTs were mildly deranged and U&Es were unremarkable.

A subsequent CT scan was arranged which confirmed a small right sided pleural effusion and a small pericardial effusion. General soft tissue oedema surrounding the upper abdominal organs was also noted. There was no evidence of malignancy.

For workup of anaemia, he underwent a gastroscopy and colonoscopy. Gastroscopy was normal and colonoscopy revealed a single pedunculated sigmoid polyp which histologically was an adenoma. An echocardiogram showed no evidence of heart failure. 24 hour ECG telemetry was arranged due to palpitations but this was essentially normal.

The patient eventually developed a bilateral temporal headache and malaise. He was commenced on 40mg prednisolone. While awaiting temporal artery biopsy he subsequently developed jaw claudication and prednisolone was increased to 60mg a day. Biopsy confirmed giant cell arteritis.

All his symptoms improved with steroids including fatigue, headaches, cough and he started to regain weight. Similarly biochemically all abnormalities normalised. A subsequent CXR showed complete resolution of the pleural effusion.

**Discussion:** Giant cell arteritis (GCA), like all systemic vasculitides, has been recognised to often involve the respiratory system. This has been reported to be in as high as 31% of patients with GCA. Notably, a persistent dry cough associated with fever has been recognised to be the most common respiratory symptom and has also been found to be one of the more atypical initial manifestations of GCA. This correlates with raised inflammatory markers and both of which were seen in the subject of this report. Although presence of a cough does not appear to correlate with other clinical symptoms of this particular vasculitis, it has been shown to markedly improve in response to steroids, suggesting its aetiology is GCA driven. The presence of pleural effusion however is far less common and indeed is exceptionally rare to be part of the initial presentation of GCA. There are a handful of cases in the literature, and most often the finding of pleural effusion coexists with more typical signs of GCA. The pleural effusion in our case was relatively small and was not considered safe for diagnostic aspiration. Rapid and complete resolution of an otherwise

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