Appendix 3: differential diagnosis of OA.

In addition to WAA and IIA, there are several other conditions with similar clinical presentations to allergic OA, which may also be work-related; these need to be considered carefully during the diagnostic process, and in many cases actively excluded.[1]

- Inducible laryngeal obstruction (ILO)[2] and breathing pattern disorder are distinct and well-described clinical entities that can be caused or exacerbated by work but may also co-exist with OA. The presence of certain clinical features such as throat tightness, hoarseness, and "air hunger" are clues that may be identified in the history. Fibre-optic nasal endoscopy whilst symptomatic (in some centres following specific provocation tests), cardio-pulmonary exercise tests and specialist physiotherapy assessment may be helpful in confirming these conditions.
- Hypersensitivity pneumonitis may present with work-related symptoms of cough, wheeze and breathlessness mimicking asthma, which may, or may not, be accompanied by constitutional symptoms of weight loss, fever, and general malaise. The most frequent cause in the UK is now metal working fluid, though exposures to avian proteins, moulds (including famers' lung) and some chemical causes (eg. di-isocyanates, epoxy resins) are all reported.[3]
- Obliterative bronchiolitis is a rare condition, characterized by sub-mucosal bronchiolar inflammation and peri-bronchiolar fibrosis. Occupationally, it is recently described in popcorn workers exposed to di-acetyl in butter flavourings,[4] but cases have also occurred in coffee processors,[5] and boat builders.[6] Onset of cough and breathlessness is usually insidious. Serial spirometry often shows rapidly progressive and fixed airflow obstruction, and full lung function testing confirms air trapping. HRCT may be ~~very~~ suggestive of the diagnosis, with air trapping and oligaemia (sharply defined mosaic attenuation), but lung histology may be required for diagnosis.
- Chronic obstructive pulmonary disease (COPD); although smoking is the principal cause, occupational exposures contribute significantly to the burden of disease (population attributable fraction reported to be 14%)[7] and there is good evidence that COPD can be caused by exposures to silica, coal mine dust, agricultural dust, textile dust, welding fume and cadmium fume. In some cases, differential diagnosis may be challenging, as some cigarette smokers with OA have co-existing COPD, and in other cases, patients with chronic OA have fixed airflow obstruction.

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