Non-progressive pulmonary fibrosis as a result of aluminium phosphide poisoning

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Abstract

Aluminium phosphide is a key ingredient of pesticides commonly used by farmers.[1,2] There have been reported cases of acute lung injury and death following accidental and non-accidental poisoning but none describing the long term pulmonary effects of aluminium phosphide in survivors.[3]

Case report

A 63-year-old agriculture contractor presented at the respiratory outpatient clinic complaining of dry cough with progressive breathlessness over a 4 month period. He became breathless on walking at normal speed and unusually exhausted performing heavy physical work. He denied any recent exposure to mouldy hay or birds. He had never smoked and was not taking any medications.

Clinical examination revealed bilateral fine basal late inspiratory crackles but was otherwise unremarkable.

Pulmonary function tests showed normal lung volumes and a minimal reduction in DLCO (83% predicted) with relative preservation of KCO (93% predicted). A High resolution CT scan of the chest (figure 1) showed posterior subpleural septal thickening, parenchymal bands and minor interstitial opacification suggesting early fibrotic changes with no significant evidence of ground glass opacification.

Clinical assessment combined with serology tests including rheumatoid factor, autoimmune screen and serum ACE did not suggest a secondary cause for his lung fibrosis which was thought to be idiopathic. His symptoms of dyspnoea progressed in line with a further decline in DLCO (61%) and he was commenced on prednisolone and azathioprine after a 9 month period of observation. His clinical course was punctuated by a diagnosis of obstructive sleep apnoea requiring CPAP and a rise in alanine aminotransferase due to Azathioprine. This rise normalised when the drug was stopped.

Figure 1.

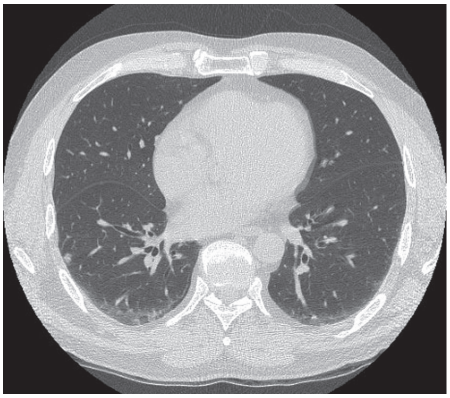
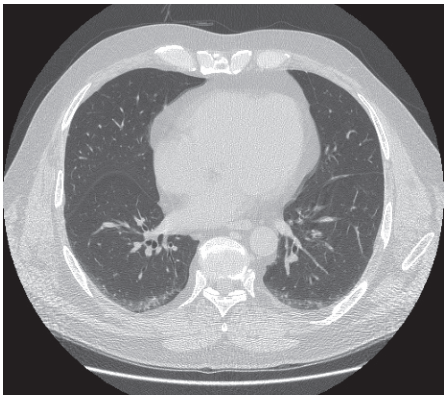


Figure 2.



Two further CT scans were performed at intervals of six and 25 months following treatment (figure 2), neither of which showed any progression in the changes of pulmonary fibrosis. Pulmonary function failed to show a clear trend of worsening restrictive defects and his breathlessness was clearly out of keeping with CT appearances. He was also shown to maintain his oxygen saturations on climbing two flights of stairs despite obvious breathlessness.

His steroid treatment was stopped and he was

referred for pulmonary rehabilitation. There was no deterioration in lung function but a persistently reduced DLCO (60% predicted).

Subsequently for the period of 3 years until his retirement 6 months following presentation, he described his work with aluminium phosphide tablets as a pest control for moles and rabbits. He regularly applied the tablets with an applicator wand into rabbit burrows and on contact with moisture phosphine gas was quickly released in the burrows appearing in other areas of the field where there were exit holes. This gas would consistently cause acute symptoms including eye irritation, dry cough, tinnitus, headache, stomach cramps and nausea.

Discussion

The manufacture and application of aluminium phosphide poses a risk of inhalation exposure to phosphine gas, which it releases upon contact with air.[4] More recent reports have identified risks of phosphine gas inhalation in association with clandestine production of methamphetamine.[5]

Phophine has been reported as a possible cause of death in individuals synthesising methamphetamine also known as "cooks". Significant exposure to phosphine may result in delayed pulmonary toxicity, although the risk of permanent injury has not been defined[6]. Due to its relatively low water solubility, phosphine has minimal warning symptoms. It has a fishy or garlic smell with an odour threshold of 1.5-5 ppm. Individuals may therefore be exposed for relatively prolonged periods without recognising the hazard.[7] Although gastrointestinal symptoms such as nausea and vomiting may occur rapidly following exposure at higher concentrations, such symptoms are not universal.

Pulmonary toxicity commonly manifests several hours following exposure, but may be delayed for 18 hours or more. Respiratory symptoms may persist for weeks, months[8], or even for years as in this case. No study has critically addressed the risk of chronic effects.

We conclude that the original diagnosis of idiopathic pulmonary fibrosis was incorrect due to lack of objective progression of his disease. It is likely that the fibrotic changes and reduced transfer factor were due to chronic low level exposure to phosphine gas causing pulmonary toxicity during the exposure period which stopped progressing once he was removed from the causative agent. His apparent progressive breathlessness on long term follow up was not explained by deterioration in objective physiological measurements but can be explained by an unresolved impairment in transfer factor (associated with pulmonary fibrosis) and patient deconditioning due to reduced activity levels following retirement from work.

References

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