Impairment of the swallowing reflex in exacerbations of COPD

An exacerbation of chronic obstructive pulmonary disease (COPD) has a serious impact on disease progression and is associated with high medical costs, but the cause of about one-third of exacerbations cannot be identified. Adequate protective reflexes in the airways play an important role in the prevention of aspiration of bacteria-containing oropharyngeal or gastric secretions. Impairment of these reflexes, such as the swallowing reflex, therefore represents a potential risk factor for exacerbations of COPD. We have conducted a cross-sectional survey to evaluate the prevalence of impairment of the swallowing reflex in patients with COPD and to determine whether this is a risk factor for COPD exacerbations.

Fifty clinically stable patients with COPD were enrolled from the outpatient clinic of Ishinomaki Red Cross Hospital, Ishinomaki, Japan. Patients who were current smokers and those with oral corticosteroid use, oral and nasopharyngeal cancer, previous head and neck surgery, neuromuscular disease and oesophageal disease were excluded. Twenty-five patients (22 men and 3 women) had at least one exacerbation during the previous year, while the other 25 patients (21 men and 4 women) were stable. In the exacerbation group, the patients had 2.4 (range 1–10) exacerbations per year, and 20 patients (80%) required hospital admission. There was no significant difference between the stable group and the exacerbation group in age (mean (SE) 75.0 (1.3) years vs 77.2 (1.0) years), forced inspiratory volume in 1 s (FEVi) (mean (SE) 1.11 (0.11) l vs 1.07 (0.09) l), percentage predicted value of FEV1 (mean (SE) 47.1 (3.7%) vs 50.1 (3.7%)) and the rate of home oxygen therapy (4% vs 20%). All patients were eating an entirely oral diet without complaining of dysphagia prior to enrolment.

We evaluated the swallowing reflex on the basis of the latency of response to the onset of the swallowing action timed from the injection of 1 ml distilled water into the pharynx through a nasal catheter. The mean (SE) latency of the swallowing reflex was significantly longer in the exacerbation group than in the stable group (8.6 (1.3) s vs 2.6 (0.3) s, p<0.001; fig 1). We classified a response as normal or impaired according to whether the swallowing reflex was induced within 3 s of the injection. In the exacerbation group 22 of 25 patients (88%) exhibited an abnormal response as normal or impaired according to whether the swallowing reflex was induced within 3 s of the injection. In the exacerbation group 22 of 25 patients (88%) exhibited an impaired response compared with 8 of 25 patients (32%) in the stable group (p<0.001).

Impairment of the swallowing reflex was significantly associated with an exacerbation of COPD (relative risk 2.8, 95% confidence interval 1.5 to 5.0).

These results indicate that there is a high incidence of impairment of the swallowing reflex in patients with COPD and this is a risk factor for an exacerbation of COPD. To date, there are few published reports available on swallowing dysfunction in patients with COPD.3 The impact of impairment of the swallowing reflex in COPD exacerbations has not been clarified. The findings of our study highlight a novel risk factor for exacerbations of COPD and raise the possibility that precautions against aspiration could be useful to prevent these exacerbations.

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References


Intravascular air and CT

Dr Ku and co-workers wrote an interesting article describing a patient with air embolism in the superior vena cava surrounding a central venous catheter (CVC) and bilateral pulmonary opacities recognised on contrast enhanced chest CT. The round pulmonary opacity was noted on chest radiograph soon after insertion of the CVC. From these findings, they suggest that this is a rare case of venous air pulmonary infarction mimicking round pneumonia. However, further evidence should be obtained to support this assumption.

CT is highly sensitive for the detection of small amounts of intravascular air, which can be found in the central veins in up to 23% of patients on contrast-enhanced CT and it rarely results in symptoms unless there is a right to left shunt.4 5

It is introduced during insertion of the venous catheter or more frequently accidental injection of air during intravenous injections (fig 1).

Normal lung tissue receives dual blood supply from pulmonary and bronchial arteries. Pulmonary infarct is infrequent after acute obstruction of the pulmonary artery because the bronchial circulation plays an important role.
GM-CSF may cause neutrophil and alveolar macrophage blood and tissues. Neutralisation of the biological activity of cytokine-macrophage colony stimulating factor (GM-CSF) in the associated with high levels of autoantibodies against granulocyte-macrophage colony stimulating factor (GM-CSF) is insufficient. Pulmonary infarcts may be observed several hours later. The small air bubbles in the superior vena cava may originate from intravenous contrast media injection during CT scan and round pneumonia is a reasonable diagnosis of pulmonary opacities in this case.

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Reference

PULMONARY PUZZLE

Answer
The patient was submitted to lung and prostate biopsies which showed pulmonary alveolar proteinosis (PAP) and embryonal rhabdomyosarcoma (fig 1A and B). A radical cystoprostactectomy with an ileal conduit was performed without curative intention due to lymph node metastasis and 1 month later the patient underwent pelvic radiotherapy and doxorubicin-based chemotherapy. The patient refused treatment of the pulmonary disease. There was progression of the lymph node metastasis and no improvement in the pulmonary symptoms or radiological findings after treatment. The patient died 1 year after surgery as a result of intra-abdominal spread of the neoplasm.

PAP is a rare cause of respiratory failure which results from accumulation of lipoproteinaceous material in the alveolar space. It is believed to be caused by dysfunction of the clearance of surfactant from the alveoli by macrophages. PAP is associated with high levels of autoantibodies against granulocyte-macrophage colony stimulating factor (GM-CSF) in the blood and tissues. Neutralisation of the biological activity of GM-CSF may cause neutrophil and alveolar macrophage dysfunction, which would explain the pathogenesis of PAP. It may be secondary to many conditions such as acute silicosis and other inhalation syndromes, immunodeficiency disorders, infections, haematological malignancies (predominantly myelogenous leukaemias), metastatic melanoma to lung and breast cancer, but no association with prostate cancer is known.

These associated diseases and GM-CSF neutralisation suggest that PAP is characterised by defective immune function. Embryonal rhabdomyosarcoma of the prostate occurs predominantly in male infants and children and is a rare and highly malignant tumour. Fewer than 20 cases of prostate rhabdomyosarcoma have been reported in adults. In the present case there was a temporal association between the urinary and pulmonary symptoms. The PAP may have been a secondary manifestation of the prostate tumour, but its treatment did not improve the pulmonary symptoms or the radiological findings.

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References

Figure 1 (A) Alveolar proteinosis (H&E stain, magnification 100×). (B) Embryonal rhabdomyosarcoma of prostate with myogenin positivity (immunohistochemical stain, magnification 400×).