Case 9—In 1968 this 44-year-old man complained of pain in both thighs; this was relieved by phenylbutazone. During the next few years he developed recurrent low back pain with morning stiffness that was relieved by indomethacin. In 1972 peripheral polyarthritis began in the index metacarpophalangeal joints and became widespread, affecting hands, feet, elbows, knees, and shoulders. In 1975 he suffered increasing backache. Examination showed limited lumbar and cervical spinal movement. He had active symmetrical peripheral polyarthritis with synovitis, and he developed two rheumatoid nodules, one on the extensor aspect of the forearm and another on a finger. Radiographs showed considerable bilateral sacroiliitis (fig 7) and mild erosive changes in hands and feet. ESR was 62 mm in one hour, the latex test gave a positive result (differential agglutination titre was 1/2048), he was negative for antinuclear factor, and HLA-27 antigen was present.

Discussion

The classification of these nine patients is difficult. They do not fall into the group of "seronegative spondarthritides" described by Moll *et al*,⁸ for they all had seropositive disease. None had a history of urethritis, psoriasis, or colitis. Eight of the nine patients had the histocompatibility antigen HLA-27 on their lymphocytes, the expected proportion in patients with ankylosing spondylitis.⁹ About 9% of a rheumatoid population would be expected to exhibit HLA-27 histocompatibility antigen,⁷ but this does not mean that these patients will all have sacroiliitis any more than such a finding would in the ordinary population.

The picture presented by the appearance of spinal ankylosis (and widespread peripheral joint ankylosis) in elderly rheumatoid patients leading a sheltered life in long-stay hospitals is also different. Only one of 11 such patients reported by Calin *et al*¹⁰ and Grahame *et al*¹¹ had the histocompatibility antigen HLA-27. Conlon *et al*¹² have shown that it is rare for ankylosis to occur in rheumatoid arthritides.

Our nine cases all fulfilled the diagnostic criteria of the American Rheumatism Association for both rheumatoid arthritis¹³ and ankylosing spondylitis.² One explanation of this phenomenon is that rheumatoid arthritis had occurred by chance in patients already suffering from ankylosing spondylitis—for example, cases 1, 2, 6, 7, and 9—or, conversely, that ankylosing

spondylitis had developed by chance in patients already suffering from rheumatoid arthritis—for example, case 5. In cases 3 and 4 the spondylitis was asymptomatic and the time of onset could not be identified. Severe erosive rheumatoid arthritis occurs in about 1 in 100 of the general population,¹⁴⁻¹⁶ and ankylosing spondylitis occurs in 1 in 500 to 1 in 2000,^{17,18} so, assuming the two diseases are independent of each other, the chance of this association in the same person is about 1 in 50000 to 1 in 200 000 and, by the same token, in nine cases, about 1 in 450 000 to 1 in 1 800 000. Hence if our cases were not the result of a simple coincidence between rheumatoid arthritis and ankylosing spondylitis one would be forced to reconsider the basic concepts that, at present, define these two diseases.

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False-positive lung scans and radiotherapy

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Summary

It has been suggested that obliterative vasculitis in the lung might mimic pulmonary embolism on a ventilation-perfusion scan. The combined scans of six patients with breast cancer who had undergone radiotherapy to the chest wall, which can induce pulmonary vasculitis, were therefore examined. Eleven of the 12 scans showed

perfusion defects with ventilation-perfusion mismatch on the irradiated side. Special care is needed in interpreting the lung scans of patients who have received an appreciable tissue dose of radiation to the lungs; mismatch need not indicate pulmonary embolism.

Introduction

The combined ventilation-perfusion lung scan is more accurate than the perfusion scan alone in diagnosing pulmonary embolism.¹⁻³ It has been suggested⁴ that diseases producing an obliterative vasculitis in the lung might show ventilation-perfusion mismatch on the combined lung scan and might be confused with pulmonary embolism. We therefore studied the combined lung scans of patients who had undergone radiotherapy that included a significant tissue dose to the lung, which is known to induce pulmonary vasculitis.⁴

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Patients and methods

The six patients studied had all had carcinoma of the breast that had been treated by simple mastectomy followed by a full course of radiotherapy to the chest wall and cervicoaxillary lymph nodes six months to 12 years earlier. The maximum calculated tissue dose was 3000-3400 rads at the lung apex and base. They were aged from 44 to 71 years, and none gave a history of tuberculosis or chest disease. At the time of scanning all had a chest radiograph on which the lung fields were reported normal. All patients gave informed consent.

An Ohio Nuclear Series 100 gamma-camera equipped with a diverging collimator was used to record the scans on polaroid film. The ventilation scan was performed with ^{133}Xe gas and the perfusion scan with $^{99\text{m}}\text{Tc}$ -macroaggregated albumin.² Each scan was mixed with six other normal or abnormal scans before being reported on by two experienced observers who knew none of the patients.

Results

Eleven of the 12 reports on the scans of the patients who had undergone radiotherapy noted abnormalities. Perfusion defects with ventilation-perfusion mismatch were noted at apex and base on six scans (three patients) and at the apex alone in the other five. One scan was reported normal. All the defects reported were on the irradiated side and corresponded to the areas of maximum calculated tissue dose. The figure shows mismatch (normal ventilation, impaired perfusion) in the scan of one of the patients.

Discussion

The diagnosis of pulmonary embolism depends on a suggestive clinical history and clinical signs, a diagnostic or normal chest radiograph, and ventilation-perfusion mismatch on the combined lung scan.¹⁻³ False-positive scans are caused by the destruction of the pulmonary arterial bed while ventilation to the area is maintained. This occurs after radiotherapy. Radiotherapy may also cause breathlessness and non-specific chest pains in the presence of a normal chest radiograph,⁵ thus clinically mimicking pulmonary embolism. In these circumstances the diagnosis will depend heavily on the lung scan and we have shown that radiotherapy will produce ventilation-perfusion mismatch on the combined lung scan. We therefore recommend that special care should be taken when interpreting the lung scans of patients who have received a significant tissue

Mismatch on ventilation-perfusion lung scan of one patient. (a) ^{133}Xe ventilation, posterior view, first breath held. (b) ^{133}Xe ventilation, posterior view, one-minute washout. (c) $^{99\text{m}}\text{Tc}$ perfusion, anterior view. (d) $^{99\text{m}}\text{Tc}$ perfusion, posterior view.

dose of radiation to the lung, as mismatch in affected areas may follow the radiotherapy and need not indicate pulmonary embolism.

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SHORT REPORTS

Traumatic division of pharyngeal branch of vagus causing dysphagia

Trauma to the neck produced dysphagia thought to be due to division of the pharyngeal branch of the vagus nerve and consequent failure of relaxation of one side of the cricopharyngeus.

Case report

A 55-year-old foreman supervising the pouring of concrete was knocked off a platform 6 m high by a crane. His fall was partly broken by a ledge 2 m above the ground and when he landed he impaled his neck on one of several upright iron rods forming the reinforcing structure. The rod was 3 cm in diameter and 76 cm high. He did not lose consciousness and received no other injuries in the fall. When seen he had a jagged triangular wound below the left mandible measuring $6 \times 10 \times 12$ cm. There was little bleeding but the submandibular gland was dislocated and lay almost free on the skin of the neck. Examination revealed a left hypoglossal nerve palsy. The soft palate was also partially paralysed on the left so some degree of vagal damage was likely. The wound extended behind the pharynx to beyond the midline but the large vessels of the neck were intact. The submandibular gland

together with some devitalised skin and muscle was removed and healing was obtained by first intention.

The only subsequent problem was dysphagia. Oesophagoscopy established that there was no stricture or extrinsic pressure but the cricopharyngeus failed to relax. Barium swallows showed pooling of barium on the left above the cricopharyngeus—the appearance of cricopharyngeal achalasia.¹ Interestingly, the patient was able to describe food “sticking” on the left and taught himself a curious trick of rocking his head over to the right to empty this pocket (see fig). Further neurological examinations during his recovery confirmed persistence of paralysis of the left side of the tongue and soft palate. There was full movement of both vocal cords. Sensibility was lost from the left side of the soft palate and the left tonsillar fossa. It was preserved over the posterior third of the tongue and the oropharynx. The gag reflex was not obtained by stimulation to the left of the midline.

Discussion

The neurological deficit is interesting and its production by this type of trauma would appear to be unique. The sensory changes imply damage to the left glossopharyngeal nerve. The palatal palsy implies an injury to some part of the left vagus although as the left cord moves the recurrent laryngeal nerve and therefore the left vagal trunk must be intact. It therefore appears that the pharyngeal branch of the vagus