SHORT REPORTS

The normal chest radiograph in bronchial carcinoma

The "early" diagnosis of bronchial carcinoma is often too late to save the patient, and improved clinical practice and better diagnostic methods are needed. The best single diagnostic tool remains the chest radiograph and our purpose here is to examine some of its limitations. These are determined by (a) the size of the lesion, (b) its location, and (c) the experience and number of readers assessing the radiograph. Tumours as small as 0.3 cm across may be detected in rib-free lung fields, but often tumours under 0.6 cm are not seen. In lungs obscured by bony or mediastinal shadows tumours as large as 2 cm may remain undetected.¹ In patients of 20 years and over the mean percentage of unobscured lung is only 24.5%.² Clearly additional views—for example, lateral projections, penetrated postero-anterior films, and films taken in expiration—will yield further information, but such pictures are not normally undertaken on initial assessment. Routine lateral films are wasteful and are only indicated when chest disease is suspected or in screening patients aged 40 years or more.³

The incidence of normal chest radiographs in bronchial carcinoma has received little attention. This has been reported as "very occasional"⁴ and as 0.1%.⁵ Over the past six years we have seen 15 patients with histologically proved bronchial carcinoma presenting with what were originally regarded as normal chest radiographs. These patients represent about 1.7% of the 900 patients with bronchial carcinoma seen during this period. We examined this problem further by reviewing these 15 radiographs.

Methods and results

The radiographs were mixed with another 15 from patients of about the same age who had undergone bronchoscopy because of suspected carcinoma and had been followed up for two years. Since no abnormality materialised in this time, we have good evidence that the second group was free of tumour. These 30 radiographs were viewed in random order and independently by a senior chest physician from another area, a senior thoracic surgeon from another area, and a senior radiologist with a special interest in chest radiology. These readers were told that some of the 30 films they were viewing were from patients with proved bronchial carcinoma, but that the remainder were known to be normal. They were asked to indicate whether there was a certain abnormality or a suspicious abnormality and to indicate its location.

Carcinoma was correctly diagnosed in six of the possible 15 cases by readers 1 and 2 and in three cases by reader 3. False-positive assessments were also made—in one case each by readers 2 and 3 and twice by reader 1. The readers usually identified different patients and in only one case did all three correctly diagnose the same carcinoma. In another two patients two readers agreed on an abnormality in the right hilar area, but only one of these patients had bronchial carcinoma. A similar analysis for suspicious abnormalities on the radiographs showed broadly similar results. The effects of dual independent reading were tested by variously combining the correct assessments. Readers 1 and 2 together diagnosed 11 of the 15 carcinomas. Readers 1 and 3 diagnosed 7 of the 15 cases, as did readers 2 and 3.

Comment

We suspect that many clinicians who do not personally interpret chest radiographs or perform bronchoscopies do not fully appreciate the limitations of these procedures. Our purpose here is to draw attention to the limitations of the standard chest radiograph in detecting bronchial carcinoma. Chest radiographs should, whenever possible, be read by two people, and when there are suspicious symptoms or signs additional investigations must be undertaken despite an apparently normal chest radiograph.

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³ Sagel, S S, et al, *New England Journal of Medicine*, 1974, **291**, 1001.

⁴ Crofton, J, and Douglas, A, *Respiratory Diseases*, p 527. Oxford and Edinburgh, Blackwell Scientific Publications, 1969.

⁵ Le Roux, B T, *Bronchial Carcinoma*, p 23. Edinburgh and London, Livingstone, 1968.

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Leptospirosis as a cause of erythema nodosum

Manifestations of leptospirosis are protean, but it has never been described in association with erythema nodosum. We report such a case.

Case report

An 8-year-old girl was admitted to hospital with a 24-hour history of pain in the right knee that precluded weight-bearing. Ten days previously she had been bitten on the right buttock by a dog. She was febrile (39.5°C), with a swollen, painful right knee and a small scar on the right buttock. Next day tender, raised erythematous areas developed on both shins. She remained feverish, and her sleeping pulse rate was 132/min. The knee was no longer painful. During the next week she improved; the temperature settled, sleeping pulse returned to normal, and the lesions on the shins slowly disappeared.

Investigations on admission showed: haemoglobin 13 g/dl; white cell count $10 \times 10^9/l$ ($10\,000/mm^3$), normal differential; erythrocyte sedimentation rate (ESR) 13 mm in the first hour (Westergren); throat swab, negative on culture; antistreptolysin O (ASO) titre 170 Todd units; tine test result repeatedly negative; radiographs of chest and right knee normal; urine, 20-25 leucocytes/high-power field, protein++, sterile on culture.

Four days after admission the acute-phase leptospiral antibody complement fixation titre was 1/40, the ESR was 68 mm in the first hour, and urine showed scanty leucocytes and protein++. Liver function studies showed nothing abnormal. The ASO titre two weeks after admission was 250 Todd units, and the titre of the convalescent-phase complement-fixing antibodies to leptospiral infections was 1/320. Agglutination of *Leptospira canicola* was positive in dilution of 1/1000.

Discussion

Initially the diagnosis was uncertain. Acute rheumatism was excluded in the early stages because of a normal ESR. Since leptospirosis is rife among dogs in Liverpool this condition was suspected. Leptospiral infections may present as feverish illnesses, arthralgias, various rashes, lymphadenopathy, aseptic meningitis, and jaundice.¹⁻³ There is a world-wide distribution of leptospiral serotypes, and infections may follow contact with an environment that has been infected by animal tissues and urine. Entry to the body is usually through mucous membranes and damaged skin. Leptospirae are not found in saliva, and animal bites are rarely causative.¹

In the summers of 1942-4 in the USA an outbreak of Fort Bragg (pretibial) fever occurred among troops, about 40 cases a year being diagnosed. The patients presented with fever, raised erythematous areas over the pretibial regions, and splenomegaly. The infecting agent was shown to be a *Leptospira* of the autumnalis group.⁴

Although there was a small rise in our patient's ASO titre, the illness was probably due to *L. canicola* with associated erythema nodosum. Classically erythema nodosum is described as raised erythematous areas over the pretibial regions, which are painful to

touch. Was the original Fort Bragg fever, therefore, erythema nodosum caused by *L. autumnalis*?

Although *L. canicola* is believed to infect a high proportion of unimmunised dogs, few infections have been described.³ In 1973 only 10 such cases were proved in the British Isles.⁵ Possibly only a minority of leptospiral infections are being detected, since feverish illnesses occur at home, where they are treated by family doctors, often with penicillin. Indeed, our patient was treated with oral penicillin 250 mg four times daily for 10 days.

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² Turner, L H, *British Medical Journal*, 1973, 1, 537.

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Vitrectomy in subarachnoid haemorrhage

Until recently, persistent vitreous haemorrhage was a blinding condition. We present a case of bilateral vitreous haemorrhage resulting from subarachnoid haemorrhage that developed suddenly but persisted without improvement of vision. Sight was successfully restored by vitrectomy.

Case report

A previously healthy 34-year-old teacher presented in 1974 with sudden severe headache followed by unconsciousness for 48 hours. He also had bilateral vitreous haemorrhage, which, when consciousness returned, had reduced his vision to perception of hand movements only. His blood pressure was 150/90 mm Hg. Blood values—including full cell count, erythrocyte sedimentation rate, urea, electrolytes, and sugar—were normal, as were x-ray appearances of skull and chest, and the results of electroencephalography, electrocardiography, and bilateral carotid angiography. The cerebrospinal fluid was blood-stained. Vertebral angiography was not performed.

The patient made an excellent general recovery, and his wife said that his rather obsessional personality was more relaxed. The vitreous haemorrhages did not resolve spontaneously, however, and B-scan ultrasonography showed bilateral dense posterior vitreous membranes (see fig). Just over a year after the subarachnoid haemorrhage he underwent staged bilateral vitrectomies via the pars plana approach, the vitreous infusion-suction cutter being used.⁵ Subsequently the visual acuity in his right eye was 6/60 due to a juxta-papillary retinal fold and premacular fibrosis, but the rest of the retina was normal. This may have been due to a vitreoretinal adhesion resulting from a choroidal haemorrhage at the time of the subarachnoid episode, although this eye had always been the worse eye. He made a spectacular visual recovery in his left eye, however, with a visual acuity of 6/9, and is leading a normal life.

Discussion

Bilateral vitreous haemorrhage resulting from subarachnoid haemorrhage was first described by Terson.¹ Most cases are associated with rupture of congenital internal carotid or anterior circle of Willis aneurysms in young people with otherwise normal blood vessels.² A clot may have obscured the causative aneurysm in our patient. Other, much rarer causes are intracerebral and intraventricular haemorrhages.² A postulated mechanism for subarachnoid, intracerebral, and intraventricular bleeding causing intraocular haemorrhage is an acute massive increase of intracranial pressure. Sudden raised intracranial pressure embarrasses the cerebral circulation, and high-pressure carotid blood is shunted, without time for vascular compensation, through its first intracranial branch (the ophthalmic) into both

B-scan ultrasonograms of left eye showing (top) vitreous opacities and posterior vitreous membrane (arrowed) preoperatively, and (bottom) disappearance of vitreous opacities postoperatively

orbits.² This causes leakage and rupture of venous capillaries, associated with the relative compression of venous exit channels from the globe. The resulting intraocular haemorrhages either are confined to the nerve-fibre layer of the retina (retinal haemorrhages) or lift the internal limiting membrane (so-called subhyaloid haemorrhages), such as may occur after the Valsalva manoeuvre.³ Rupture through the internal limiting membrane causes vitreous haemorrhage.

Spontaneous return to normal vision may occur in these patients, but once a posterior vitreous membrane forms it cannot be absorbed and vision does not return.⁴ Vitrectomy, by means of an instrument inserted into the eye via the pars plana that will cut and aspirate abnormal tissue while maintaining the intraocular pressure by infusion of physiological fluid, offers a fairly simple sight-saving procedure and replaces previous operations, which have had a poor rate of success.⁵

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³ Valsalva, A M, *De Aure Humana tractatus*, ch 5, sect 8, p 84. Utrecht, vande Water, 1707.

⁴ Blach, R K, *Transactions of the Ophthalmological Society of the United Kingdom*, 1975, 95, 407.

⁵ Machemer, R, Panel, J M, and Buettner, H, *American Journal of Ophthalmology*, 1972, 73, 1.

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