Unilateral hyperhidrosis associated with underlying intrathoracic neoplasia

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Intrathoracic neoplasia is notable for the many ways in which it may present. We would like to report two cases demonstrating a rare association between unilateral localised hyperhidrosis of the thoracic cage and underlying intrathoracic neoplasm.

Case reports

Case 1

A 67 year old retired shotblaster complained of a 3 kg weight loss, mild dyspnoea, chest pain localised to the right costal margin, and profuse sweating localised to an area below the right scapula. He smoked 15 cigarettes per day. Examination confirmed a right sided localised band of sweating at the level of T6–9 posteriorly. Apart from minimal winging of the right scapula and some wasting of the right suprascapular muscles no abnormal neurological signs were detected. A chest radiograph showed a large right pleural effusion with collapse of the underlying lung. Analysis of the pleural fluid revealed no malignant cells or acid fast bacilli, but a pleural biopsy confirmed the presence of poorly differentiated carcinoma. A technetium radioisotope bone scan showed increased uptake in the anterior ends of the right 4th and 5th ribs. The abnormal sweating continued for a further six months but the patient’s condition deteriorated with the development of intra-abdominal metastases and recurrent pleural effusions. He died at home; necropsy was not performed.

Case 2

A 58 year old engineering foreman, known to have had Crohn’s disease since 1974, complained of ache of two months’ duration in the left side of his chest, malaise, and a weight loss of 12 kg. He smoked 30 cigarettes a day and had had a brief exposure to asbestos. Examination revealed a large pleural effusion, which was confirmed on a chest radiograph. Analysis of the sputum and pleural fluid was unhelpful, as was a pleural biopsy. The appearances on a technetium radioisotope bone scan were normal. The dull ache over the left side of his chest worsened, and six months later he developed a localised band of profuse sweating at the level of T5–8 on the left side of his chest. There were no abnormal neurological signs. Repeat clinical and radiographic examination suggested a diagnosis of left pleural mesothelioma. The sweating continued until his death four months later. At necropsy the left lung was totally encased by the mesothelioma tissue, which had invaded the chest wall. There were metastatic plaques in the right hemithorax and abdominal cavity but no evidence of metastases in the brain or the spinal cord.

Discussion

The association of intrathoracic malignancy with sympathethic neurological complications, especially Horner’s syndrome, is well recognised, particularly in the case of tumours occurring at the thoracic inlet. Unilateral hyperhidrosis is an unusual phenomenon which has been reported sporadically in association with various conditions, including intracranial malignancy, encephalitis, syringomyelia, trauma, neuritis, cervical rib, osteoma of the dorsal spine, and chickenpox; in several cases no obvious underlying cause has been evident.

Unilateral hyperhidrosis associated with intrathoracic malignancy was first described in 1976 and since then nine cases associated with primary tumours, including one mesothelioma, and one with metastases have been reported. In the majority of cases the sweating has been localised to the same side as the tumour, although three cases of contralateral sweating have been reported.

Direct invasion of a nerve by a tumour may produce pain and a disturbance of motor, sensory, or autonomic function. Eccrine sweating is mediated by cholinergic sympathetic nerves arising from ganglia supplied by efferent fibres from T1–L2. Experimental studies have shown that there is cross innervation and that section of one or more anterior roots may cause no sweating loss; while electrical stimulation of a single anterior root results in widespread sympathetically mediated effects over the distribution of at least five or six sympathetic ganglia. Two main mechanisms have been postulated for the phenomenon of unilateral hyperhidrosis. Lesions of the preoptic area anterior to the hypothalamus may cause unilateral hyperhidrosis, although in our case and several others there was no neurological or postmortem evidence of cerebral metastases. Irritation of nerves, as was seen in the two cases associated with cervical ribs, may cause excitation of the autonomic efferent fibres; and in both cases excision of the cervical ribs abolished this. It has been postulated that direct infiltration of the sympathetic chain may cause unilateral hyperhidrosis, which over a period of time, has been observed to evolve into ipsilateral anhidrosis and Horner’s syndrome. None the less it is unusual that the sympathetic stimulation should persist in some cases as long as six months, when operative and postmortem findings have confirmed extensive invasion and ablation of the sympathetic chain by tumour tissue. Unilateral hyperhidrosis may remit spontaneously, as in case 1; but in several cases it has been abolished by radiotherapy or surgery. It is, however, usually a sign of advanced tumour spread and carries a poor prognosis.
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References


The sum may be divided to support two or more applicants. Applications should be sent by 31 January 1987 to Dr KM Citron, (secretary for the fellowships), Brompton Hospital, London SW3 6HP.

World Congress on Oxygen Therapy and Home Care

A world congress on oxygen therapy and home care will be held at the Marriott Hotel, City Center, Denver, on 19–21 February 1987. The congress is being held under the auspices of the Webb-Waring Lung Institute and the American Association for Respiratory Care. The date for abstracts has already passed. Those interested in attending this meeting are invited to write to the Congress Secretariat at Webb-Waring Lung Institute, 4200 E Ninth Avenue Box C-321, Denver Colorado 80206, USA.