

PALMAR AND PLANTAR KERATODERMA WITH HYPERPIGMENTATION AND GYNECOMASTIA

Report of a Case Associated with Primary Adenocarcinoma of the Lung

Le Roy Riddick, Roger H. Brodtkin and Richard C. Gibbs

From the Department of Medicine and the Division of Dermatology, New Jersey College of Medicine and Dentistry, and from the Department of Dermatology, New York University Medical College, Skin and Cancer unit, New York, USA

Abstract. Palmar and plantar Keratoderma has been described as a cutaneous manifestation of systemic cancer. A patient is reported who developed palmar and plantar keratoderma with hyperpigmentation and gynecomastia as cutaneous manifestations of primary pulmonary adenocarcinoma. Other reported instances of the association of one or more these signs with systemic cancer are considered and their possible relationships are considered.

CASE REPORT

The patient was a 53-year-old Negro man whose illness began in May, 1969. He had been in good health until then but began to notice the gradual onset of weakness, weight loss, and thickening of the skin on his palms and soles. He also noticed that his skin was becoming generally darker, particularly over the anterior surfaces of the lower legs, and his breasts were becoming painful and swollen. About 1 month later as these complaints worsened in severity, he developed a nonproductive cough and weakness and numbness of the right arm and hand. He was admitted to the Martland Hospital Unit of the New Jersey College of Medicine in July, 1969, with pain above the right scapula, hemoptysis, and 32 lb weight loss. He had worked as a clothes presser and smoked three packs of cigarettes a day. Physical examination on admission was within normal limits, except for the following findings: His skin color was that of a dark Negro with dusky gray, macular hyperpigmentation of both shins. His breasts were markedly enlarged bilaterally and symmetrically and were diffusely firm to palpation. The palmar and plantar surfaces of the hands, fingers, toes, and feet displayed a marked thickening of the surface which was distinctly rugose, markedly accentuating the normal ridges and sulci (Figs. 1 and 2). Hyperhidrosis of these areas produced a macerated gray color. The right anterior cervical and supraclavicular lymph nodes were enlarged to palpation. The right upper hemi-

thorax exhibited percussion dullness, increased vocal fremitus, and bronchial breathing. The right upper extremity showed weakness of extension and flexion and diminished sensation. He exhibited no clubbing of the fingertips or toes. Laboratory values including CBC, eosinophiles, electrolytes, FBS, BUN, serum creatinine, total protein, and prothrombin time were within normal limits, except for the following: alpha-1-globulin 4.6% (5-7%), beta-globulin 17.9% (11-17%), gamma-globulin 28.3% (15-25%), 24-hour urine 17 hydroxy steroid 18.4 mg (5-15 mg), and normal 17 ketosteroids. Sputum for acid-fast bacilli was negative. X-rays of chest revealed consolidation of the entire upper lobe of the right lung. Barium enema, upper G.I. series, skeletal X-ray and I.V.P. and rectosigmoidoscopy were normal. Biopsies taken at bronchoscopy and from the scalene fat pad showed nonspecific inflammation. Biopsies of the palm and sole showed only hyperkeratosis and acanthosis (Fig. 3). A month after admission an open thoracotomy was performed, revealing an extensive tumor of the upper and lower lobes of the right lung which was believed to be a primary carcinoma in the lung. It was attached in several areas to the parietal pleura. Histologic examination of the tumor revealed an adenocarcinoma (Fig. 4). Postoperatively, the patient was given palliative X-ray therapy to the lung which was followed by gradual increase in strength of the right upper extremity. In September, 1969 he was discharged, but 1 month later developed a superior vena cava syndrome and was readmitted for further X-ray therapy. This was followed by another remission for 1 month. He was admitted for the last time in November, 1969. Over the 6 months since the onset of his symptoms, he had lost 60 lb and then weighed 95 lb. Dyspnea, cachexia and weakness were marked. Antibiotics and cytoxan provided no relief and his gradual deterioration continued. It was noted, however, that the palmar and plantar hyperkeratosis had diminished since his first admission. He died in January, 1970 and a post-mortem examination could not be performed.

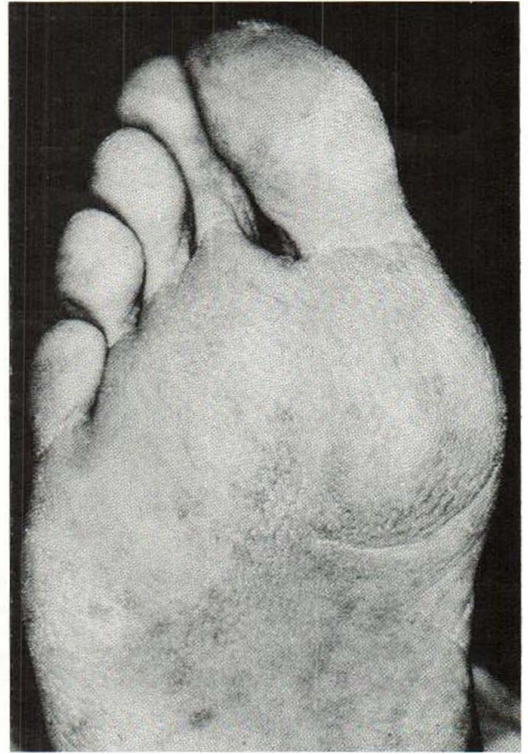


Fig. 2. Photograph of planta showing marked hyperkeratosis with accentuation of plantar ridge pattern.

Fig. 1. Photograph of palm showing marked hyperkeratosis with accentuation of palmar ridge pattern.

COMMENT

It is presumed that the palmar and plantar hyperkeratosis, gynecomastia, and generalized and localized hyperpigmentation were related to the patient's adenocarcinoma.

Palmar and plantar hyperkeratosis has been noted as a manifestation of internal malignancy in Sezary's syndrome, malignant acanthosis nigricans, ichthyosiform atrophy of Ronchese, and pachydermoperiostosis (1). Generalized and localized hyperpigmentation has also been noted in association with systemic malignancy, particularly lymphoma, and many other non-malignant diseases such as Addison's disease, porphyria cutanea tarda, polyostotic fibrous dysplasia, thyrotoxico-

sis, renal tubular acidosis, chronic malnutrition, pellagra, etc. (2). It cannot be stated with certainty on the basis of our knowledge in this case what may have caused the hyperpigmentation. This would require MSH levels and determining whether the MSH was produced by the pituitary gland or the tumor. Its appearance coincident with the other signs of neoplasm and lack of evidence of any of these other diseases suggests that the hyperpigmentation was related to the neoplasm. Gynecomastia and secondary hypertrophic osteoarthropathy associated with epithelial bronchogenic carcinoma is well known and has been reported to have regressed in the terminal stages of the malignancy as the palmar and plantar hy-



Fig. 3. Photomicrograph of palmar lesion showing marked hyperkeratosis and acanthosis. Stained with H & E, enlarged from $\times 32$.

perkeratosis was noted to regress in the present case (3). The reason for this is unknown in both cases.

Although this patient did not have typical acanthosis nigricans, his palmar and plantar hyperkeratosis seemed to present the same type of hypertrophic change. This particular change may be due to adenocarcinoma rather than epithelial carcinoma. The reason for the different localization is unknown. This case does seem to indicate, however, that our concepts of the hypertrophic

cutaneous changes (clubbing of the digits, acanthosis nigricans, gynecomastia, hyperpigmentation, and palmar and plantar hyperkeratosis) may require reevaluation and provide clinical signs of the specific type of internal malignancy. The regression observed in some of these hypertrophic changes as the patient succumbs to his neoplasm seems to indicate that they may be directly related either to host resistance to the neoplasm or inversely related to the tumor's overcoming of the host.

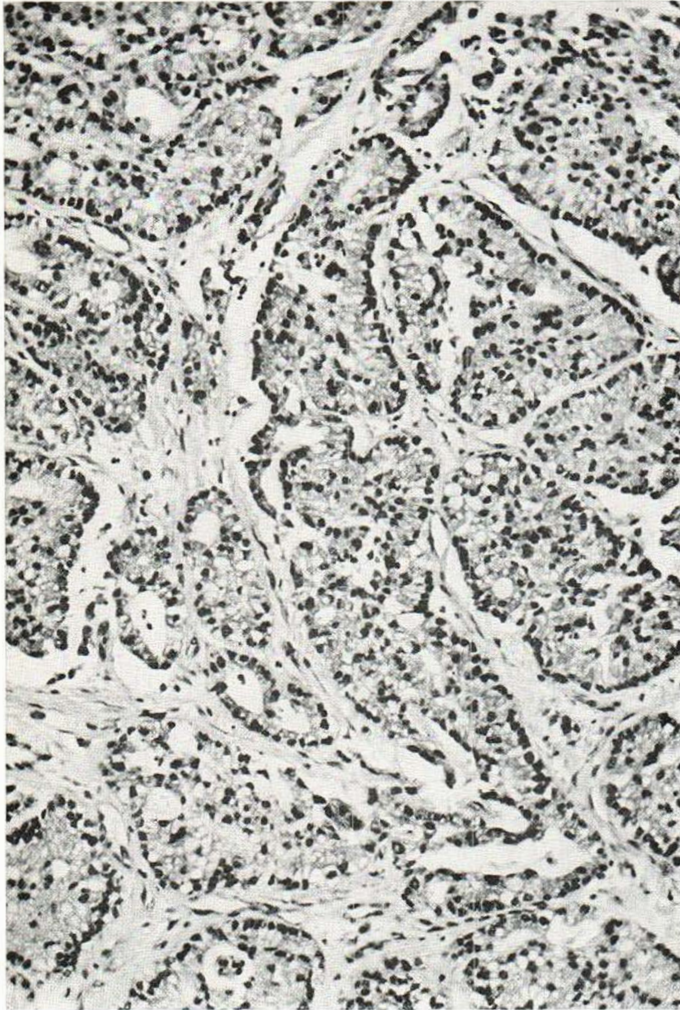


Fig. 4. Photomicrograph of primary pulmonary adenocarcinoma. Stained with H & E, enlarged from $\times 160$.

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Roger H. Brodtkin, M.D.
40 Union Avenue
Irvington, N.J. 07111
USA