

Becker's Naevus Associated with Connective Tissue Naevus

Sir,

Becker's naevus or Becker's melanosis is a relatively common disorder, which appears as a hyperpigmented, hypertrichotic patch located mainly on the trunk (1). Becker's naevus has been associated with various soft tissue and bony structural anomalies, such as hypoplasia of the ipsilateral breast, pectoralis major muscle, leg, arm and contralateral breast, spina bifida, and pectus carinatum (1–3). Furthermore, variable smooth muscle hamartomatous changes may be associated with Becker's naevus, thus it is classified as a cutaneous organoid hamartoma (4). We report here a case of Becker's naevus associated with connective tissue naevus.

CASE REPORT

A 27-year-old Korean man presented with an asymptomatic, 8 × 10 cm brownish patch with multiple, soft, movable nodules on the back, which was found incidentally 1 year ago (Fig. 1). His medical and family history was non-contributory and no other abnormality was found on physical examination. Histopathological examination showed slight acanthosis, regular elongation and clubbing of the rete ridges in the epidermis, which were consistent with the findings of Becker's naevus. In the dermis, coarse and thickened fibres were found, which were disclosed as collagen on Masson's trichrome stain (Fig. 2). Verhoeff-van Gieson staining revealed no change in elastic fibres.



Fig. 1. Asymptomatic 8 × 10 cm brownish patch with multiple, soft and movable subcutaneous nodules on the patient's back.

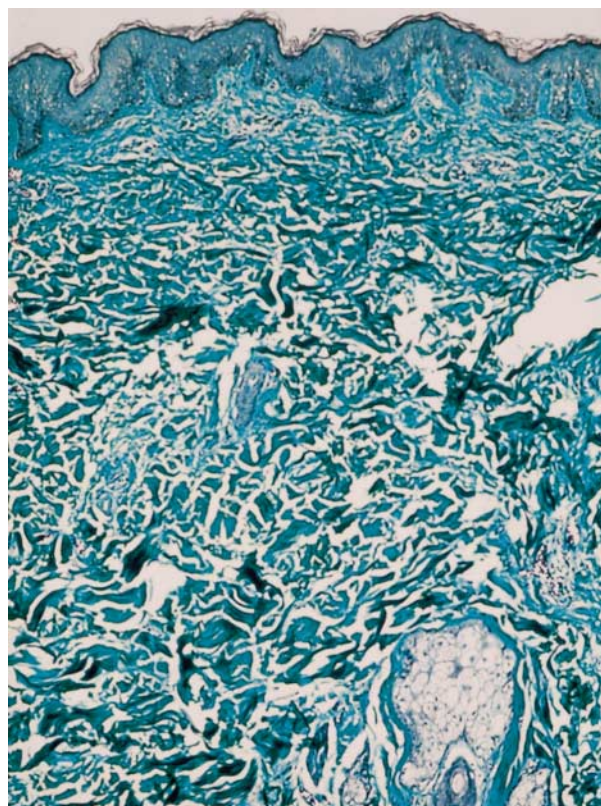


Fig. 2. Increased amount of collagen fibres in the lesion (Masson's trichrome stain; original magnification × 40).

DISCUSSION

Connective tissue naevi represent various hamartomas characterized by abnormalities in quality and structure of collagen, elastin or proteoglycans. In the collagen type of connective tissue naevus, collagen fibres are increased in amount and often normal in appearance, although they show thickened and homogeneous appearances in some cases. Elastic tissue in the collagen type connective tissue naevi may be normal, increased, or decreased in amount. According to the classification of Uitto et al. (5), our case pertained to the acquired, eruptive type.

Fenske & Donelan (6) described the first case of Becker's naevus coexistent with connective tissue naevus, but it was not determined whether the case represented previously unrecognized association or coincidental occurrence of 2 unrelated diseases. To our best knowledge, this is the second case of Becker's naevus coexistent with connective tissue naevus. Our case may suggest that concurrence of Becker's naevus and connective tissue naevus is the possible association, not the coincidental finding.

REFERENCES

1. Glinick SE, Alper JC, Bogaars H, Brown JA. Becker's melanosis: associated abnormalities. *J Am Acad Dermatol* 1983; 9: 509–514.
2. Formigon M, Alsina MM, Mascaro JM, Rivera F. Becker's nevus and ipsilateral breast hypoplasia-androgen receptor study in two patients. *Arch Dermatol* 1992; 128: 992–993.

3. Moore JA, Schosser RH. Becker's melanosis and hypoplasia of the breast and pectoralis major muscle. *Pediatr Dermatol* 1995; 3: 34–37.
4. Chapel TA, Tavafoghi V, Mehregan AH, Gagliardi C. Becker's melanosis: an organoid hamartoma. *Cutis* 1981; 27: 405–406.
5. Uitto J, Santa Cruz DJ, Eisen AZ. Connective tissue nevi of the skin. *J Am Acad Dermatol* 1980; 3: 441.
6. Fenske NA, Donelan PA. Becker's nevus coexistent with connective-tissue nevus. *Arch Dermatol* 1984; 120: 1347–1350.

Accepted January 9, 1999.

Dai-Ho Kim, Chung-Won Kim and Tae-Yoon Kim
Department of Dermatology, Kang-Nam St. Mary's Hospital,
College of Medicine, The Catholic University of Korea, 505,
Banpo-dong, Seocho-gu, Seoul, 137-040, Korea.

Erythromelalgia as a Paraneoplastic Syndrome in a Patient with Abdominal Cancer

Sir,

Erythromelalgia (EM) is a symptom-diagnosis characterized by red, warm and painful extremities. Symptomatic relief is achieved by cooling and sometimes by elevation of the affected limbs. Attacks are initiated or aggravated by exposure to heat and often by exercise and standing. Microvascular arterio-venous shunting combined with tissue hypoxia is thought to cause the symptoms (1). EM can be a primary condition, or secondary to a primary disease. We report here on a patient with EM of the lower extremities associated with abdominal cancer. EM symptoms preceded the cancer diagnosis by several years. EM has been reported secondary to myeloproliferative disorders, but to our knowledge EM associated with an internal malignant disease has not previously been described.

CASE REPORT

A 37-year-old woman with EM presented at our clinic in December 1989. The patient had had symptoms consistent with irritable colon from the age of 16 years. Constipation had been the predominant symptom and she used laxatives regularly. During pregnancies in 1973 and 1978 she easily developed lower limb oedema with extreme swelling. She had had moderate to severe anaemia during the years 1981–89, which was thought to be caused by iron deficiency, and was treated with iron supplements. From 1986 she suffered from intermittent burning pain, warmth, redness and swelling of the feet, ankles and legs, which was worsened by standing, walking, running, exposure to the sun, and warm shoes. The symptoms and signs occurred more often during warm weather and in the evening and were relieved by elevation of the feet. The symptoms gradually worsened from 1988 to 1990. She started active cooling by walking barefoot on cold floors and in 1989 the symptoms exacerbated markedly and she cooled the feet in ice-cold water for up to 1 h/day. Symptoms often got worse during her work, where she was standing by an assembly line all day. She was on sick leave for long periods and lived an inactive life at home in order to prevent EM attacks.

On clinical examination in November 1989, she had no symptoms and signs of EM. A complete blood count was normal. Inspection of the peripheral blood smear and bone marrow revealed no pathology. Electrolytes, creatinine and urea, albumin and s-protein, glucose, IgE, lactate dehydrogenase, urine examination and liver function tests were all normal. Antinuclear antibodies and rheumatoid factor were negative. Histology from affected areas was normal and evaluation of the same biopsy with immunofluorescence showed non-specific

changes with fluorescence against IgM, complement C3, terminal complement complex and fibrinogen in some dermal vessels.

Treatment with aspirin, metoprolol and propranolol during 1989 had no effect. Prostaglandin E1 infusions were tried in March 1990 with excellent relief of the symptoms for 4 months. At this time the patient experienced periods of nausea, reduced appetite, weakness, fatigue and poorly localized abdominal pain. She had noticed increasing constipation with mucous and fresh blood in the stools. Two weeks following prostaglandin E1 treatment she was hospitalized with vomiting and increased abdominal size. During laparotomy 12 l ascites fluid and large tumour masses of the left ovary, sigmoid colon, peritoneum and liver was found. Adnexa, omentum majus, parts of sigmoid colon and metastases of fossa Douglasi were extirpated. Histological examination showed middle-differentiated adenocarcinoma compatible with a primary colonic cancer. No irradiation or further surgery was indicated. Intermittent treatment with 5-fluorouracil intravenously was tried for 3 months without effect. She then received palliative treatment until she died in November 1990.

This patient is one of 124 cases of EM collected over a period of 15 years (2, 3). In this patient colonic cancer was diagnosed after 4 years of suffering from EM, but the adenocarcinoma was probably undiagnosed for several years. The symptoms of EM gradually worsened when the cancer progressed, consistent with a parallel course of the conditions. It has been reported that myeloproliferative disorders may be preceded by EM (4, 5). In this study 1 patient developed chronic myeloid leukaemia and 4 polycythaemia vera.

REFERENCES

1. Kvernebo K. Erythromelalgia—a condition caused by microvascular shunting. *VASA* 1998; Suppl. 51: 1–39.
2. Mørk C, Kalgaard O, Kvernebo K. Erythromelalgia: a clinical study of 102 cases. *Australas J Derm* 1997; 38 Suppl 2: 50.
3. Kalgaard OM, Seem E, Kvernebo K. Erythromelalgia. A clinical study of 87 cases. *J Intern Med* 1997; 242: 191–197.
4. Kurzrock R, Cohen PR. Erythromelalgia and myeloproliferative disorders. *Arch Intern Med* 1989; 149: 105–109.
5. Redding KG. Thrombocythaemia as a cause of erythromelalgia. *Arch Dermatol* 1977; 113: 468–471.

Accepted April 19, 1999.

Cato Mørk¹, Ole M. Kalgaard¹ and Knut Kvernebo²

¹Department of Dermatology, The National Hospital and ²Department of Surgery, Ullevaal Hospital, University of Oslo, Norway.
E-mail cato.mork@rh.nio.no