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A Phase II Study of Epirubicin in Patients with Advanced Adenocarcinoma of the Pancreas

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THE PROGNOSIS for patients with pancreatic carcinoma remains dismal [1] and effective treatments are required. Wils *et al.* reported 8 responses in 34 evaluable patients who had received epirubicin [2]. This response rate of 24% encouraged us to study epirubicin in inoperable pancreatic carcinomas.

Patients were eligible if they fulfilled the following criteria: histologically and/or clinically confirmed adenocarcinoma of the pancreas, aged below 75, performance status (ECOG) 0–3, no previous treatment with irradiation or chemotherapy, measurable disease, adequate bone marrow, cardiac, hepatic and renal

functions, no severe complications, no active second cancer, estimated life expectancy of 4 weeks or more and informed consent given. Computed tomography and sonography were used to measure lesions. Epirubicin was given (90 mg/m²) intravenously on day 1 and repeated every 4 weeks. Response was evaluated every 4 weeks. Definition of response and toxicity was according to WHO criteria. When the disease progressed, the treatment was changed to another chemotherapy.

14 patients entered the study and all were evaluable (Table 1). There were neither complete responses nor partial responses (3 no change and 11 disease progression). 8 patients (57%) had severe leukopenia (more than grade 3). Moderate to severe nausea and vomiting was also frequently seen, and 10 patients (71%) had alopecia. There were no toxic deaths.

Thus, we saw no response with epirubicin as single agent in the treatment of advanced pancreatic carcinoma. Further trials of combination with epirubicin are not recommended.

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Table 1. Patients, responses and toxicity

	No. of patients
Entered/evaluable	14/14
Median age (range)	56 (45–69)
M/F	6/8
Performance status	
0–1	5
2–3	9
Location of pancreatic cancer	
Head	7
Body and tail	7
Site of measurable disease	
Pancreas	13
Liver	7
Lung	1
Lymph node	1
Median no. of courses	1.7 (1–3)
Response	
No change	3
Disease progression	11
Toxicity (≥ grade 3)	
Leukopenia	8 (57%)
Anaemia	3 (21%)
Nausea/vomiting	4 (29%)

Acute Febrile Neutrophilic Dermatitis (Sweet's Syndrome) in Metastatic Breast Cancer

Henrik Nielsen

ACUTE FEBRILE neutrophilic dermatitis (Sweet's syndrome) was initially described as a benign disease of unknown aetiology [1]. The disorder is characterised by high temperature, neutrophilic leukocytosis and multiple, raised, erythematous, painful cutaneous plaques. The skin lesions show a dense dermal infiltrate of mature neutrophils on histological examination. The disease responds promptly to steroids.

Subsequently, it became clear that Sweet's syndrome may be related to malignancy as a paraneoplastic phenomenon. The neoplastic condition most frequently associated with Sweet's syndrome is acute myelogenous leukaemia, but in addition several other haematological malignancies are reported [2, 3]. In contrast, few patients with solid cancers have been described [2–5], curiously most with pelvic origin of the primary tumour. Moreover, despite the high prevalence of breast cancer, only 2 cases have been associated with Sweet's syndrome [4, 5].

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A female patient was first seen at age 47 in 1977, when a malignant tumour of the left breast was removed. No sign of disseminated disease was found, and the patient received no additional therapy. In 1988, a pathological fracture of the left femoral neck revealed an osteolytic metastasis of an adenocarcinoma. Following osteosynthesis the patient was given cyclophosphamide, methotrexate, and 5-fluorouracil for 6 months. A stable, partial remission was obtained and the patient remained in good performance status (WHO stage 0–1). In May 1991, progressive bone pain was shown to be caused by multiple osteolytic lesions in the pelvic and columnar bones. Prophylactic irradiation towards large lesions in the lumbar column was given but without success, as multiple vertebral fractures emerged during the following months. At the same time as the diagnosis of disseminated bone involvement, the patient complained of multiple, painful, erythematous, raised lesions of the fingers and dorsal surfaces of the hands, the largest measuring 5 cm. The distribution of the lesions was confined to the hands, and the other parts of the body were without cutaneous symptoms. A short treatment with penicillin was without effect. After 14 days, a skin biopsy was performed, and findings characteristic for Sweet's syndrome were demonstrated. Therapy with prednisolone led to disappearance of skin lesions within a few days, but an attempt after 3 weeks to discontinue steroid therapy was followed by recurrence over the next month. The patient was therefore given prednisolone 15–25 mg daily continuously during the following months with complete remission of skin lesions. The patient presented no symptoms or haematological evidence of a myelodysplastic or leukaemic disorder. The malignant disease, however, showed progression

and the patient died from disseminated breast cancer 5 months after the diagnosis of Sweet's syndrome.

The patient showed several clinical characteristics of Sweet's syndrome: female, age 60, lesions on the upper extremities only and prompt response to steroid therapy. Symptoms recur in 30% of cases [2]. Sweet's syndrome is an established paraneoplastic condition, but is hitherto rarely described in association with solid tumours. Our case suggests that the syndrome may reflect progression of an underlying cancer.

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Correction

Nutrients and cancer prevention.—This book (Vol. 28, 236) was published in 1991 and the correct ISBN number is 0896031713.