tumours in this environment. In contrast, the clinicopathological spectrum of the more common B-cell neoplasms has been well-documented. In an immunophenotyping study of 100 consecutive cases of NHLs seen in the Pathology Department of the University College Hospital, Ibadan within a 3 year period, 12 T-cell neoplasms stained positively with the T-cell markers, anti-CD3 and UCHLI, but were negative with the B-cell markers (L26, 4KB5). Table 1 shows the characteristics of the cases. The patients' ages ranged from 10 to 68 years with a mean age of 42 years, and a male to female ratio of 5:1. Of the 12 cases, 10 were post-thymic T-cell neoplasms, and 9 of these were node-based. Only one case each of T-lymphoblastic lymphoma with mediastinal tumour and *Mycosis fungoides* were identified. The two cutaneous T-cell tumours included the cases of *Mycosis fungoides* with multiple skin nodules, and subcutaneous breast nodule with angio-invasive T-cell proliferation. In Western countries, approximately 2% of chronic lymphocytic leukaemias are of T-cell origin [7] and T-cell small lymphocytic lymphoma is considered to be rarer still. However, 3 cases of T-cell lymphocytic lymphomas were identified in this series. The other morphological types of T-cell malignancies seen are listed in Table 1.

Although the number of cases seen are few, some conclusions can still be inferred. T-cell malignancies occur in Ibadan, Nigeria and post-thymic neoplasms appear to be more common than thymic neoplasms, the majority of which are node-based. It would seem that cutaneous T-cell lymphomas are not as prevalent as reported in the Western literature, nor are the post-thymic T-cell malignancies in Ibadan, Nigeria as common as in the HTLV-1 endemic areas of South East Asia.

1. Kadin ME, Berard CW, Nanba K, Wakasa H. Lymphoproliferative diseases in Japan and Western countries. *Hum Path* 1983, **14**, 745–772.
2. Su IJ, Wang C, Cheng A, *et al.* Characterization of the spectrum of postthymic T cell malignancies in Taiwan. A clinicopathologic study of HTLV-1-positive and HTLV-1-negative cases. *Cancer* 1988, **61**, 2060–2070.
3. Kikuchi M, Mitsui T, Matsui N, *et al.* T cell malignancies in adults. Histopathological studies in lymph nodes in 110 patients. *Jpn J Clin Oncol* 1985, **15**, 645–651.
4. Lutzer M, Edelson R, Schein P, Green I, Kirkpatrick C, Ahmed A. Cutaneous T cell lymphomas: The Sezary syndrome, *Mycosis fungoides* and related disorders. *A Intern Med* 1975, **83**, 534–552.
5. Jaffe ES. Pathologic and clinical spectrum of postthymic T cell malignancies. *Cancer Invest* 1984, **2**, 413–426.
6. Weiss LM, Crabtree GS, Rouse RV, Warnke RA. Morphologic and immunologic characterization of 50 peripheral T cell lymphomas. *Am J Path* 1985, **118**, 316–324.
7. Collins RD, Waldron JA, Glick AD. Results of multiparameter studies of T cell lymphoid neoplasms. *Am J Clin Path* 1979, **72**, 699–707.

European Journal of Cancer Vol. 31A, No. 11, pp. 1885–1886, 1995.

0959–8049/95 $9.50 + 0.00

0959-8049(95)00372-X

Splenic Metastases in Patients with Portal Hypertension

P. Pedrazzoli, A. Catona, L. Pavesi, M. Gossemberg and G. Robustelli della Cuna

Involvement of the spleen in the course of solid tumours usually occurs in patients with widespread disease and multiple organ involvement [1]. We describe 2 cases in which splenic metastases were the first evidence of distant disease. Both patients had liver cirrhosis, and a possible haemodynamic mechanism favouring spleen colonisation is discussed.

In a 64-year-old woman (patient 1) with a 7 year history of chronic active hepatitis progressing to cirrhosis, a large irregular pelvic mass (10 cm) and a focal lesion of the spleen were diagnosed by computed tomography (CT) scan. Serum CA125 antigen was greatly increased, and fine needle biopsies under ultrasound guidance disclosed the diagnosis of ovarian adenocarcinoma with splenic metastasis. Six cycles of chemotherapy (PEC (cisplatin, epirubicin and cyclophosphamide) regimen) were administered. Due to a very good partial response to chemotherapy, the patient underwent cytoreductive surgery (hysterectomy plus bilateral salpingo-oophorectomy, omentectomy, splenectomy, sigmoid colon resection) which confirmed the presence of left ovarian carcinoma infiltrating the sigmoid colon and a solitary splenic metastasis. No further chemotherapy was given, and the patient was well until 7 months later when pelvic wall recurrence was diagnosed.

A 65-year-old man (patient 2) underwent surgical resection for adenocarcinoma of the descending colon. The patient was HCV (hepatitis C virus) positive and a liver biopsy performed during intervention disclosed cirrhosis. Adjuvant chemotherapy was given (5-fluorouracil plus leucovorin for eight cycles). The patient was well until 18 months after surgery when a large spleen lesion (7 cm) was documented by ultrasonography. The patient was referred for surgery. Splenectomy was performed confirming the presence of a secondary lesion from adenocarcinoma of the gastrointestinal (GI) tract with no other abdominal organ involvement. No further treatment was given and the patient is alive and well with no signs of recurrence at 20 months from surgery.

Metastasis to the spleen from various neoplasms is very rare. In autopsy studies [2], splenic involvement is found in approximately 7% of patients, breast cancer, lung cancer and melanoma being the most common sources. Splenic metastases are usually part of widespread abdominal disease with lymph node involvement, and it has been suggested that they arise from

Correspondence to P. Pedrazzoli.
A. Catona and M. Gossemberg are at the Divisione di Chururgia, Ospedale C. Mira—Casorate Primo (MI); all other authors are at the Divisione Oncologia Medica, Fondazione Clinica del Lavoro, Via S Boezio 26, 27100 Pavia, Italy.
Received 5 Apr. 1995; accepted 12 Apr. 1995.

cells that have been conveyed to that organ by the arteries [3]. Several theories have been postulated to account for the low incidence of metastases to the spleen compared to other parenchymatous organs [4], including a possible role of spleen contractions in forcing the blood from the sinusoids into the splenic vein which keeps tumour cells in constant motion.

The 2 patients we describe here had spleen metastases from ovarian and colonic cancer in the absence of other distant organ involvement. These unusual presentations of advanced disease raise the question of whether an underlying condition may represent a risk factor for spleen colonisation in subjects with solid tumours. Both patients had liver cirrhosis causing portal hypertension. When the portal venous pressure rises, blood stasis, retrograde blood flow and diversion of portal blood into systemic veins, in an attempt to decompress the portal system, can occur. We suggest, that in such conditions, a neoplastic embolus may reach the spleen via the mesenteric veins and by retrograde blood flow in the splenic vein. Moreover, implantation of neoplastic cells may be facilitated by blood stasis which increases the time of contact with the splenic tissue. The 2 cases described here seem to support this hypothesis. Patient 1 had primary ovarian cancer involving the sigmoid colon and patient 2 had a primary tumour of the descending colon; the venous system of the left region of the colon drains into the portal circulation via the inferior mesenteric vein which enters the splenic vein.

Whether neoplastic cells from tumours draining into the portal system can more easily seed the spleen in patients with portal hypertension needs to be confirmed. However, based on our experience, we suggest a more careful evaluation of the spleen at intervention and during follow-up in these subjects.

1. Berge T. Splenic metastases: frequencies and patterns. *Acta Pathol Microbiol Scand* 1974, **82**, 499–506.
2. Morgenstern L, Rosenberg J, Geller SA. Tumors of the spleen. *World J Surg* 1985, **9**, 468–476.
3. Marymount JH Jr, Gross S. Patterns of metastatic cancer in the spleen. *Am J Clin Pathol* 1963, **40**, 58–60.
4. Klein B, Stein M, Kuten A, *et al.* Splenomegaly and solitary spleen metastasis in solid tumors. *Cancer* 1987, **60**, 100–102.

European Journal of Cancer Vol. 31A, No. 11, pp. 1886–1887, 1995.

0959–8049/95 $9.50 + 0.00

0959-8049(95)00378-9

Problems with the Evaluation of Response After Induction Chemotherapy in Breast Cancer

C.K. Mannesse, R. van Pel, J. van Spengler, C. H. van Eijck and T.A.W. Splinter

INDUCTION CHEMOTHERAPY has become part of the standard treatment of locally advanced breast cancer. Recently, it has also been used to induce "lumpectability" in tumours larger than 3 cm [1–3] or as part of induction chemoradiotherapy [4]. Moreover, the response to induction chemo(radio)therapy may be used as a prognostic factor [5, 6].

Since 1990, we have performed a prospective study of preoperative induction chemotherapy in patients with different stages of breast cancer. After clinical examination, mammography, ultrasound and cytological proof of the primary tumour, the patients were treated with cyclophosphamide 100 mg/m^2 orally days 1–14, doxorubicin 30 mg/m^2 and 5-Fluorouracil 600 mg/m^2 intravenously (i.v.) on days 1 and 8. Courses were repeated every 4 weeks. After two and four courses, the response of the primary tumour was assessed by palpation, mammography and ultrasound. For stable disease or progression after the first two courses, the patient underwent surgery. Responders received another two courses of chemotherapy and then surgery. Standard criteria of response according to the WHO [7] were used. The study is ongoing. However, in a recent analysis, three problems with the evaluation of response were observed in 7 of the 22 patients (Table 1). In patients 1–3, who had major clinical responses, a very small lesion of viable invasive ductal carcinoma (IDC) was seen adjacent to extensive ductal carcinoma *in situ* (DCIS) in the resection material. These findings indicate a discrepancy between clinical and pathological complete remissions, but more importantly a lack of chemosensitivity of DCIS. Since the extent of DCIS is not predicted by pre-chemotherapy cytology nor expected from the distribution of mammographic microcalcifications [8, 9], extensive DCIS may still be found in resection material after a major response to induction chemotherapy of the invasive tumour.

Table 1. Patients' characteristics

Patient	Clinical stage	Response	Surgery	Histopathology	
1	T2N2 (2.5 cm)	CR	L*	DCIS	IDC
2	T1N2 (2 cm)	CR	Q*	DCIS	IDC
3	T4bN2 (6 cm)	PR (1.8 cm)	M	DCIS	IDC
4	T2N1 (4 cm)	CR	L*	—	IDC
5	T1N2 (1.5 cm)	MR (1.1 cm)	L*	—	ILC
6	T2N0 (4 cm)	MR (2.1 cm)	L*	LCIS	ILC
7	T4cN1 (5 cm)	PR (1.4 cm)	M	—	ILC

CR, complete response; PR, partial response; MR, minor response; L, lumpectomy; Q, quadrantectomy; M, radical mastectomy; DCIS, ductal carcinoma *in situ*; LCIS, lobular carcinoma *in situ*; IDC, invasive ductal carcinoma; ILC, invasive lobular carcinoma. *After ultrasound localisation.

Correspondence to C.K. Mannesse.
C.K. Mannesse and T.A.W. Splinter are at the Department of Medical Oncology; R. van Pel is at the Department of Pathology; J. van Spengler is at the Department of Radiology; C.H. van Eijck is at the Department of Surgery; and C.K. Mannesse is also at the Department of Internal Medicine, University Hospital Rotterdam Dijkzigt, Erasmus University Rotterdam, The Netherlands.
Received 13 Mar. 1995; accepted 16 May 1995.