During the immediate postoperative period she remained disorientated and developed left-sided deafness but to the relief and surprise of all she gradually improved and could even perform housework independently. No abnormal behavior had been observed. At the latest examination in September 1992 she was fully orientated and there were no gross neurologic deficits except for bilateral deafness. It was, however, impossible to assess her memory, intellect or olfactory functions due to communication problems.

Discussion. The classical Kluver-Bucy syndrome consists of hypersexuality, oral hyperactivity, hypermetamorphosis, placidity or aggression, memory disorder, and visual agnosia (1). To avoid this tragic outcome it is understandable that clinicians are reluctant to offer bitemporal lobectomy, even in life-saving situations with no effective alternative. The exact risk of bilateral resection in humans is poorly known as only one case has been reported in the literature (4). The incidence of Kluver-Bucy syndrome following unilaterial excision is extremely low: among the thousands of patients operated on with standard anterior temporal lobectomy for epilepsy only one case with a partial syndrome has been reported (5). Among the rare cases of this syndrome in humans the commonest etiology is bilateral temporal lobe damage due to viral encephalitis, trauma, Pick's disease, Alzheimer's disease, multiple infarction or subarachnoid cysts.

The risk of damaging the temporal lobes during radical radiation therapy for nasopharyngeal carcinoma, the peculiar clinical and CT features, and the treatment results for radionecrosis have been presented in a previous publication (6). Although the CT manifestations are often asymmetrical and non-synchronous, the basic damage is bilateral. With accumulation of long-term survivors, temporal lobe radionecrosis has been detected in 3% (n = 207) of patients with nasopharyngeal carcinoma irradiation in our institute. None has developed Kluver-Bucy syndrome. The relative scarcity of symptoms and signs in our patients is not fully understood but can possibly be attributed to the very slow rate of progression. It seems not illogical to postulate that removal of sections of the brain which have long ceased to function may not result in disastrous exacerbation of disability.

There is little doubt that surgery is the most definitive treatment for radionecrosis but the bilateral involvement poses special difficulty. Thus only ten of our patients had surgical intervention which in all cases was limited to the side with the predominant lesion. The control by unilateral surgery is, however, only temporal as relapse in the contralaterial side will inevitably occur if the patient survives long enough. Conservative treatment with corticosteroids can achieve durable objective response in about one-third of the patients detected during the initial phase of reactive edema but is ineffective once liquefactive necrosis has developed (6). Furthermore, the hazard of fatal infection as a result of severe immunodepression is alarmingly high in our crowded environment.

Although we cannot claim that bitemporal lobectomy is a safe procedure and the patient reported had not been subjected to thorough psychometric assessment, her recovery to independent normal life is gratifying. For patients severely disabled by chronic lesions not treatable by other methods the risk of surgery is well justified.

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REFERENCES

1. Kluver H, Bucy PC. Preliminary analysis of functions of the temporal lobes in monkeys. Arch Neurol Psychiat 1939; 42: 949–1000.
2. Ellis F. Dose time and fractionation. A clinical hypothesis. Clin Radiol 1969; 20: 1–7.
3. Fowler JF. The linear-quadratic formula and progress in fractionated radiotherapy. BJR 1989; 62: 679–94.
4. Fontana M, Mastrostefano R, Bernabei A, et al. Bilateral temporal lobectomy for late radionecrosis after radiotherapy for acromegaly. A case report. J Neurosurg Sci 1984; 28: 107–12.
5. Cogen PH, Antunes JL, Correll JW. Reproductive function in temporal lobe epilepsy: the effect of temporal lobectomy. Surg Neurol 1979; 12: 243–6.
6. Lee AWM, Ng SH, Ho JHC, et al. Clinical diagnosis of late temporal lobe necrosis following radiation therapy for nasopharyngeal carcinoma. Cancer 1988; 61: 1535–42.

IgD PRODUCING IMMUNOCYTOMA

A 46-year-old woman was admitted to our hospital because of fever, dyspnea, dry cough of 3-week duration and history of weight loss of 10 kg during the last year. Sixteen months before admission, she underwent a tumorectomy and axillary lymph node dissection followed by local radiation therapy for a 3 cm intraductal papillary carcinoma of the right breast. Physical exampination revealed pleural and pericardial rubs as well as massive splenomegaly. Laboratory studies revealed a hematocrit of 24%, reticulocyte count 3% and the white blood cell count was $2 \cdot 10^9$/l (56% neutrophils, 36% lymphocytes and 8% monocytes). The platelet count was $90 \cdot 10^9$/l and the erythrocyte sedimentation rate 57 mm/h (Westergren). Antinuclear, antimitochondrial and antismooth muscle antibodies were negative as well as direct and indirect coomb's tests. Serum C3 and C4 levels were normal, albumin 23 g/l, globulin 90 g/l, lactate dehydrogenase 400 IU/ml. Immunodiffusion studies of serum protein showed an IgD component at a concentration of 6 g/l and low levels of the other immunoglobulins (IgG = 2 g/l, IgA = 180 mg/l and IgM 20 mg/l); Kappa and Lambda chains were undetectable. Urine electrophoresis and immunoelectrophoresis was normal. A chest x-ray showed cardiomegaly and bilateral pleural effusions. Computed tomographic scans of the chest and abdomen showed pleuropericardial effusions and massive splenomegaly without thoracic or abdominal lymphadenopathy. Thoracentesis revealed a clear fluid with $250 \cdot 10^6$ white cells/l (91% lymphocytes, 8% neutrophils, 1% eosinophils). All cultures for bacteria, myco-bacteria, viruses and yeasts were negative as well as serological titers for mycoplasma, legionella, cytomegalovirus. A pleural biopsy showed reactive mesothelial hyperplasia with no evidence of malignancy. Biopsy of the liver was normal and bone marrow biopsy showed normal erythroid and myeloid series with 10% plasma cells. The patient underwent a laparotomy, and splenectomy was performed. The spleen weighed 1.9 kg and histological examination showed numerous non-necrotizing granulomas and diffuse infiltration by small lymphocytes and plasma cells containing 'Dutcher-bodies'. Numerous Russell bodies and rare immunoblasts were also found. The immunoperoxidase staining on paraffine-fixed tissue showed diffuse cytoplasmic positivity with anti IgD but negative with anti IgG, IgA, IgM, Kappa and Lambda chains. These findings were diagnostic of an IgD lymphoplasmacytic lymphoma (immunocytoma in Kiel classification, equivalent to the low-grade malignant lymphoma, small lymphocytic, plasmacytoid, in the International Working Formulation classification). The patient refused further investigation or therapy and was lost to follow-up.

Immunocytomas represent a monoclonal proliferation of small lymphocytes which are predominantly of the B-cell type. They are associated with lymphoplasmacytoid or typical plasma cells but immunoblasts are rarely seen. Three subtypes are prescribed: lymphoplasmacytic, lymphoplasmacytoid and polymorphous (1). A high content of epithelioid cells was described in certain immunocytomas (2) and our patient had numerous granulomas in her spleen. Russell bodies can be found in the cytoplasm of the plasma cell and Dutcher bodies are PAS positive intranuclear inculsions found in the same cells. Immunological staining for light chains demonstrates their presence in 61% of cases (3). Heavy chains presence is more easily found (89%) with a high predominance of IgM often accompanied by IgD (70%) (1, 3) and 10% express IgD and IgA. However, IgE is present in exceptional cases (4). A monoclonal gammopathy is present in about one-third of the cases and is often of the IgM type, rarely IgG or IgA. Clinically, a massive splenomegaly without peripheral lymphadenopathy can be the mode of presentation in some cases (5, 6). To our knowledge, this is the first report in the literature of an immunocytoma with the exclusive expression of heavy chains of the IgD type and no light chain expression. The monoclonal gammopathy is also of the IgD type and is associated with a depression in the secretion of the other immunoglobulins. Unfortunately, our patient was lost to follow-up and the significance of such an exceptional case is difficult to assess. Long-term follow-up of similar cases may determine whether their prognosis is different from other patients with immunocytomas.

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REFERENCES

1. Harris NL, Bhan AK. B-cell neoplasms of the lymphocytic, lymphoplasmacytoid, and plasma cell types. Immunohistologic analysis and clinical correlation. Hum Pathol 1985; 16: 829–37.
2. Patsouris E, Noël H, Lennert K. Lymphoplasmacytic/lymphoplasmacytoid immunocytoma with a high content of epithelioid cells. Histologic and immunohistochemical findings. Am Surg Pathol 1990; 14: 660–70.
3. Hall PA, D'Ardenne AJ, Richards MA, et al. Lymphoplasmacytoid lymphoma: an immunohistological study. J Pathol 1987; 153: 213–23.
4. Tian SS, Faust C. A simple and precise aberrant translocation of the rat c-myc gene into the epsilon-heavy chain switch region of the IgE producing immunocytoma, IR162. J Biol Chem 1989; 264: 1846–53.
5. Audouin J, Diebold J, Schwartz H, Letourneau A, Bernadou A, Zittoun R. Malignant lymphoplasmacytic lymphoma with prominent splenomegaly. J Pathol 1988; 155: 17–33.
6. Theml H, Burger A, Keiditsch E, et al. Klinische Beobachtungen zur Charakterisierung des splenomegalen Immunozytomas. Med Klin 1977; 23: 1019–32.

PROTEIN SYNTHESIS IN JEJUNUM OF SPRAGUE-DAWLEY RATS AFTER IRRADIATION

It is well known that irradiation causes morphological changes in tissue. However, the specific effect of radiation on protein synthesis in a tissue or organ is not completely understood. Previous studies (1–2) performed in rat skin showed an inhibition of protein synthesis with doses of 1500 R or more, while with levels near 750 R, a significant stimulatory effect was observed, leading to the conclusion that the effects of radiation on protein synthesis could be due to a mechanism which is independent of DNA damage.

The jejunum is considered particularly sensitive. Total abdominal irradiation is associated with a decline in the effective resistance of the gastrointestinal tract and a rise in the passive permeability properties of the small and large intestine (3–4). The aim of the present study was to determine protein synthesis in jejunal mucosa of rats after a single exposure to abdominal irradiation at various dose levels.

Material and methods. Sixty male Sprague-Dawley rats, (Biocentre, S.A., Barcelona, Spain), were utilized for this study. Determination of the normal range of jejunal protein synthesis was done in a control group of 15 healthy animals (mean body weight: 191.1 ± 6.2 g). The irradiation experiments were performed on 45 rats (mean body weight: 169 ± 3.1 g), housed individually in metabolic cages and fed standard chow and water ad libitum until irradiation. The animals were divided into three groups ($n = 15$) to receive 3 dose levels of radiation 4 Gy, 6 Gy and 10 Gy.

The rats were anesthetized intraperitoneally with 5% sodium pentobarbitone at a dose of 0.1 ml per 100 g of body weight. An 11×10 cm field on the abdomen from the xiphoid process to the pubis received a single x-ray exposure (Stabilipan, Siemens 250 kV, 15 mA, 1 mm Cu-filter, SSD 43 cm). The dose was calculated at midline (total thickness 3.5 cm) to deliver 1.03 Gy/min. After irradiation, the rats were kept in individual metabolic cages, fed a clinical modular diet ad libitum (Table 1) for 4 days, and killed by decapitation on the fifth.

Protein synthesis was determined in each irradiated group and in the controls with the 'flooding dose' method (5). l-(1-^{14}C) leucine was combined with unlabelled l-leucine to give c. 1 MBq and 135 μmol/mm. One ml of this solution per 100 g of animal body weight was administered through the lateral tail vein. In each irradiated group, five animals were killed 2 min after the radionuclide injection, and the remaining ten animals, 10 min after. In the control group, six animals were killed at 2 min, and nine at 10 min. After decapitation, a midline abdominal incision was made to remove the proximal small intestine, discarding the first 20 cm from the pylorus, and taking the following 20 cm after stretching with a 3.5 g weight. Jejunal tissues were obtained by slitting the intestine longitudinally and scraping the luminal side with a microscope slide. The samples were then transferred into

Table 1

Composition of modular diet used to feed rats after total abdominal irradiation

Protein (kcal)	14
Lipid (kcal)	49
Carbohydrates (kcal)	37
MCT (% kcal)	40
LCT (% kcal)	60
Total nitrogen (g)	3.2
Keltrol (g)	0.5
Total kilocalories	100

Composition for 100 ml of diet.