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Beneficial Effects of Octreotide in a Patient With a Metastatic Paraganglioma

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UPTAKE OF a radiolabelled somatostatin analogue has already been reported in malignant paraganglioma [1, 2], as well as *in vitro* inhibition of catecholamine secretion by octreotide [3].

A 44-year-old man had been treated for a metastatic paraganglioma with several modalities: surgery for adrenal paraganglioma and bone metastases, metaiodobenzylguanidine (MIBG) therapy, chemotherapy and external radiotherapy for bone metastases.

In February 1993, he presented with a low performance status, paraplegia, pseudo-obstruction of the large bowel not responsive to classical treatments, left parotid metastasis measured by ultrasound (45 × 30 × 40 mm), multiple bone metastases and bone marrow involvement with thrombocytopenia (platelets: 16 × 10^9/l) and anaemia (Hb: 8.9 g/100 ml). MIBG uptake was seen in known metastases on scintigraphy, but no hormonal overproduction was found in repeated urinary measurements.

Uptake of a radiolabelled somatostatin analogue (OctreoScan®, Mallinckrodt Medical) was seen in distant metastases on whole body scintigraphy and led us to initiate a treatment with octreotide (500 μg subcutaneously per day). An improvement of the performance status, the normalisation of intestinal function, a decrease of 50% in the size of the parotid metastasis, the normalisation of bone marrow production with an improvement in haematological parameters (platelets: 166 × 10^9/l and Hb: 12.2 g/100 ml) and a decrease of more than 50% in the intensity of metastatic MIBG uptake were noted. No unexpected side-effect was noted. These positive effects lasted for 6 months, after which the disease progressed and the patient died 10 months after initiation of octreotide therapy.

In conclusion, the beneficial effects of octreotide treatment could be quantified by clinical, tumour and scintigraphic criteria, which is rarely the case [4]. These data suggest that octreotide can be useful in the treatment of malignant paraganglioma at a certain stage of the disease.

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Rapid Tumour Lysis Syndrome in a Metastatic Colorectal Cancer Increased by Treatment with Irinotecan (CPT-11)

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TUMOUR LYSIS syndrome (TLS) is usually manifested by hyperuricaemia, hyperkalaemia, hyperphosphataemia and hypocalcaemia and can lead rapidly to an acute nephropathy with renal failure. It is due to massive necrosis of neoplastic cells, sometimes occurring spontaneously but more often after effective cytotoxic therapy. Commonly described in haematopoietic malignancies, it occurs rarely in solid tumours. We report a case of TLS in a patient with metastatic colon carcinoma.

A 42-year-old female underwent a left hemicolectomy in March 1994 for an adenocarcinoma of Dukes' C stage. She subsequently received six courses of adjuvant chemotherapy with 5-fluorouracil (5-FU) and folinic acid. Six months after the end of the treatment, she relapsed with a perirectal disease and multiple liver metastases. She then received pelvic irradiation (60 Gy) and chemotherapy with continuous infusion of 5-FU over 15 days. At the beginning of the treatment, laboratory values revealed elevated uric acid (570 μmol/l), kalaemia (5.4 mmol/l) and phosphataemia (1.43 mmol/l). Lactic dehydrogenase (LDH) was 700 UI/l. Other values including renal function and calcaemia were within the

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