


**Mediastinal germ cell tumour and myelodysplastic syndrome**

Sir,

Nichols et al. have recently described 16 cases of haematological neoplasia associated with non-seminomatous mediastinal germ cell tumours and suggest that these neoplasms arise from a common progenitor cell.

We wish to report a 20 year old man with an enormous primary mediastinal germ cell tumour with histological evidence of yolk-sac elements in combination with teratoma, and high alpha-fetoprotein (AFP) serum levels (2130 ng/ml). He was treated with six courses sequentially of combinations of cisplatin, etoposide, ifosfamide and bleomycin with a partial response as measured by thoracic scan and the reduction of AFP levels to 190 ng/ml.

During the chemotherapy treatment, the patient developed persistent pancytopenia and a severe myelodysplastic syndrome with abnormal megakaryocytes in two bone marrow biopsies. The cytogenetic analysis demonstrated three abnormal clones: trisomy 1 with an extra marker chromosome, a second clone with, added to the first, a trisomy 18 and a third clone with an extra 8 chromosome. The karyotype yielded 48, XY, +1, + mar/49, XY, +1, +18, + mar/50, XY, +1, +8, +18, + mar.

In the patients who develop a chemotherapy or radiotherapy related leukaemia or myelodysplastic syndrome, the karyotype usually shows a hypodiploid modal number and abnormalities in chromosomes 3, 5, 7 and 17. However, in this patient’s karyotype the deletions and monosomies often found in people with therapy related haematological neoplasms were not present. On the other hand, trisomy 8, frequently observed in myelodysplastic syndromes, was found.

We feel that this young man with non-seminomatous primary mediastinal germ cell tumour and severe myelodysplastic syndrome with cytogeneric abnormalities beginning after chemotherapy treatment, is similar to those patients reported by Nichols et al.1


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**References**


**Doxycycline-induced parotitis**

Sir,

Sialadenitis is an uncommon side effect of drugs and chemicals such as nifedipine, alpha-methyl dopa, phenylbutazone, interferon alpha, H2 receptor antagonists, oxypenbutazone, iodine compounds and nitrofurantoin. To our knowledge, no cases of doxycycline-induced sialadenitis have been described until now.

An 18 year old female developed serum sickness while receiving doxycycline for inflammatory acne. She had been taking doxycycline 200 mg/day orally for 15 days when she voluntarily discontinued the treatment for 1 week. Forty eight hours after reintroducing this drug, she developed fever (38.2°C), chills, generalized urticaria, hand and foot oedema, and arthralgia in both knees, ankles and wrists. She had not received any other medication during the previous 3 months. Physical examination also revealed bilateral cervical lymphadenopathy up to 2 cm in diameter. Seventy-two hours later, she developed bilateral painful parotid swelling. There was tenderness, reddening and an increased temperature of the skin over the glands. Simultaneously, a recrudescence of urticarial lesions was observed. Other studies showed mild leukocytosis (12,700/mm³), elevated ESR (52 mm in the first hour), hypocomplementaemia (C4, 10 mg/dl) and proteinuria (1.6 g/l) and haematuria (33 red cells per high power field). Serum protein electrophoresis and serum immunoglobulins were within normal limits. Antinuclear antibodies and hepatitis B surface antigen were not detected in serum. She was treated with methylprednisolone 80 mg daily intravenously and hydroxyzine 100 mg/day orally. The parotitis resolved in 24 hours and the rest of the clinical manifestations in 5 days.

The mechanism of drug-induced sialadenitis remains unclear in most cases. Either oedema and spasm of smooth muscle in the salivary gland or a hypersensitivity reaction could be responsible. In the case reported here, the association between serum sickness and parotitis during doxycycline therapy points to an immunological pathogenesis.


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