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Mediastinal germ cell tumour and myelodysplastic syndrome

Sir,
Nichols *et al.*¹ have recently described 16 cases of haematological neoplasia associated with non-seminomatous mediastinal germ cell tumours and suggest that these neoplasms may 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 17.² 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,³ was found.

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

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Doxycycline-induced parotitis

Sir,
Sialadenitis is an uncommon side effect of drugs and chemicals such as nifedipine,^{1,2} alpha-methyldopa,³ phenylbutazone,⁴ interferon alpha,⁵ H2 receptor antagonists,⁶ oxyphenbutazone,⁷ iodine compounds⁸ and nitrofurantoin.⁹ 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/mm), 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.^{1,3,5–7,9} In the case reported here, the association between serum sickness and parotitis during doxycycline therapy points to an immunological pathogenesis.

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