

Table 1. Characteristics of the newborns

| Sex                  | Female  | Male    | Male    |
|----------------------|---------|---------|---------|
| Size (cm)            | 47      | 49      | 51      |
| Weight (g)           | 2320    | 3230    | 3300    |
| Apgar                |         |         |         |
| 1 mm                 | 8       | 10      | 9       |
| 3 mm                 | 3*      | —       | —       |
| 5 mm                 | 10      | 10      | 10      |
| Hair                 | +       | +       | +       |
| Dyspmorphic syndrome | —       | —       | —       |
| Blood count at birth |         |         |         |
| WBC                  | 13      | 8900    | 17900   |
| Hb                   | 15.4†   | 18.3    | 19.8    |
| Platelets            | 309 000 | 193 000 | 316 000 |

\*Secondary apnoea (ventilation). †Secondary anaemia at 9.5 g at day 21; spontaneous recovery.

epidoxorubicin before delivery. The children are now 35, 34 and 23 months old and growth and development remain completely normal. All 3 mothers are in complete remission. Therefore, from these limited data, it seems that vinorelbine could be safely administered during the 2nd and 3rd trimesters of pregnancy.

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## Pneumothorax Following Induction Chemotherapy for a Germ Cell Tumour

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A 26-YEAR-OLD man presented with a one-month history of progressive headache. Magnetic resonance imaging (MRI) revealed a 3.8 cm diameter round lesion in the left cerebellum. General physical examination was normal except for evident cerebellar dysfunction. Alpha-fetoprotein ( $\alpha$ FP) and lactate dehydrogenase (LDH) were within normal limits; human chorionic gonadotropin  $\beta$  ( $\beta$ HCG) was slightly elevated at 5.6 IU/l ( $N < 3$  IU/l), while carcinoembryonic antigen (CEA) was up to 457  $\mu$ g/l ( $N < 4.6$   $\mu$ g/l). Computerised tomography (CT) showed a 3  $\times$  4 cm diameter lesion of the left inferior lung. Because of a rapid worsening of cerebellar functions, cerebellar metastasis was removed, with subsequent complete recovery.

The diagnosis of malignant embryonal carcinoma (EC) was retained, although immunohistochemistry was not pathognomonic (focal staining for CEA and alpha-1-antitrypsin and isolated cells were positive for the  $\beta$ HCG). A first cycle of ifosfamide, cisplatin and etoposide (ICE) was administered. On day 16, the patient complained of a sudden cough and left pleuritic pain. A chest X-ray showed a partial collapse of the left lung (Figure 1), for which a chest tube with negative pressure was inserted and the lung fully re-expanded in 48 h. Response to chemotherapy was suggested by the decline of the CEA level from 457 to 138  $\mu$ g/l as well as by the necrosis observed on a CT scan. After the second cycle of ICE, CEA was within normal limits and the thoracic lesions were necrotic. Unfortunately, this improvement was transient, and the patient developed a fulminant carcinomatous meningitis. Second-line chemotherapy and local radiotherapy were unsuccessful, and he died within a few days.

Cancer related spontaneous pneumothorax is a rare event [1], and may be observed in three circumstances: at time of diagnosis or during progression; late after chemo- or radiotherapy; and shortly after chemotherapy [2]. The majority of cancer related spontaneous pneumothorax are present at the time of tumour diagnosis [3]. All tumours may be

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Figure 1. Chest X-ray: left-sided pneumothorax with shrinkage of the mass.

complicated by pneumothoraces, even if there is a clear predominance with metastatic osteogenic and childhood sarcomas, germ cell tumours and lung cancer [4]. The mechanisms advocated depend upon the underlying tumour. In lung cancer, spontaneous pneumothorax may be consecutive, either to various pathological alterations secondary to smoking, or to the progression of the tumour [1, 3]. In sarcomas and germ cell tumours, spontaneous pneumothoraces are more likely induced by tumour necrosis or spontaneous haemorrhages [5]. It can also occur some months or years after irradiation or chemotherapy, in association with treatment, pathological changes rendering the lung more prone to develop spontaneous pneumothorax [2, 6].

Our case fits well with a rapid chemotherapy-induced tumour shrinkage secondary to necrosis. Rosen and colleagues first noted that the risk of spontaneous pneumothorax was higher with efficient chemotherapy [7]. The average time from the initiation of the chemotherapy to pneumothorax development ranges from 1 to 32 days. Several mechanisms have been proposed to explain chemotherapy-induced spontaneous pneumothorax: an underlying co-existent emphysema [4, 9]; the rupture of a peripheral chemosensitive tumour, resulting in a leakage of air into the pleural space [6]; the enlargement of a rapidly necrotising tumour; the combination of necrosis and chemotherapy-induced impairment of repair processes (e.g. doxorubicin) [8–10]; the elevation of intrathoracic pressure due to drug-related emesis [4].

Spontaneous pneumothorax is a well known, but rare, complication occurring during chemotherapy for cancer. This complication should be kept in mind, especially in tumours known to be chemosensitive. Indeed, it may be a sign of tumour response and explain the appearance of a shortness of

breath and pulmonary pain shortly after chemotherapy without conferring a bad prognosis to the patient.

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## 2-Chlorodeoxyadenosine Inhibits Activity of Adenosine Deaminase and S-Adenosylhomocysteine Hydrolase in Patients With Chronic Lymphocytic Leukaemia

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2-CHLORODEOXYADENOSINE (2-CdA) is a new and effective drug for indolent lymphoid malignancies. However, mech-

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