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Desmoplastic small round cell tumour of unknown primary origin with lymph node and lung metastases: histological, cytological, ultrastructural, cytogenetic and molecular findings

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Abstract Desmoplastic small round cell tumour (DSRCT) is an extremely aggressive neoplasm belonging to the family of "small round blue cell tumours", which includes primitive neuroectodermal tumour (PNET), Wilms' tumour and Ewing's sarcoma. DSRCT is considered to be a neoplasm derived from a primitive cell. It is immunohistochemically reactive with epithelial, neuronal and mesenchymal cell markers, demonstrating divergent differentiation along three cell lines. Originally thought to arise from serosal surfaces, the tumour has recently been reported in the central nervous system and ovary. The tumour in this case is a neoplasm that meets the histological, immunohistochemical, cytological and cytogenetic criteria of DSRCT; it is not associated with serosal membranes, and it has supraclavicular and axillary lymph node deposits and multiple pulmonary and brain metastases. Tumour cells from our case show cytogenetic similarities with Ewing's sarcoma and PNET. Electron microscopic findings suggest similarities between DSRCT and Wilms' tumour. Cloning and sequencing of PCR products showed in-frame fusion of EWS exon 7 to WT1 exon 8.

Key words Desmoplastic round cell tumour · DSRCT · EWS · WT1 · Cili

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Introduction

Originally described by Gerald and Rosai in 1989 [9], desmoplastic small cell tumour with divergent differentiation is an extremely aggressive neoplasm belonging to the family of "small round blue cell tumours" [10, 18, 22, 23]. The tumour affects primarily children and young adults, presents with widespread intra-abdominal and pelvic involvement, and has a male-to-female ratio of 4:1. Although most patients are found to have relatively large tumour masses and extensive serosal seeding on laparotomy, no consistent association with a particular intra-abdominal organ has been documented.

Desmoplastic small round cell tumour (DSRCT) has been described in the pleural cavity [2, 24], the tunica vaginalis testis [25] and, most recently, in the central nervous system [34]. This case report describes a metastatic neoplasm of unknown origin that fulfils the morphological, molecular and immunohistochemical criteria for DSRCT.

Case report

In a 34-year old man, left supraclavicular lymphadenopathy was found during a routine physical examination. Further questioning revealed that the adenopathy had been present and enlarging slowly for approximately 1 year and that it was occasionally painful. A nonproductive cough and occasional night sweats had also been present for approximately 1 year. The patient denied weight loss, fever, nausea, vomiting or bone pain. Physical examination revealed a 4×5 cm matted, left supraclavicular mass and a tender 5×5 cm mass in the left axilla. The remainder of the physical examination, including testicular examination, was within normal limits. Chest X-ray demonstrated multiple, bilateral intrapulmonary nodules. Computed tomographic (CT) scan of chest, abdomen and pelvis revealed multiple bilateral intrapulmonary nodules, the largest of which measured 2×4 cm, and carinal and mediastinal lymphadenopathy (Fig. 1). No pleural involvement was identified. The abdomen, retroperitoneum and pelvis showed no evidence of disease. Tissue was obtained by fine-needle aspiration of the supraclavicular lymph node and a subsequent biopsy of the axillary mass. Tissue from the biopsy specimen was submitted for light microscopic, ultrastructural, and cytogenetic examination and also for molecular analysis. After the diagnosis of DSRCT was es-

tablished, two cycles of chemotherapy (cyclophosphamide, doxorubicin and vincristine) were administered. Repeat CT scan of the chest, abdomen and pelvis demonstrated a partial response, with decreased tumour nodule size and no evidence of further metastases. A third cycle of chemotherapy was administered with clinical improvement, but the patient complained of intermittent frontal headaches. A head CT scan revealed three hypodense lesions in the brain, which were consistent with metastasis, and radiation to the brain was administered. A restaging CT of the brain revealed a partial response, and the patient subsequently received four more cycles of alternating CAV and ifosfamide/etoposide. Restaging CT of chest, abdomen and pelvis revealed and almost complete resolution of axillary and mediastinal lymphadenopathy and regression of the pulmonary nodules to linear opacities consistent with scarring. The patient did well until 6 months later, when he became symptomatic with coughing. Repeat CT scan of the chest and abdomen revealed recurrent pulmonary nodules consistent with recurrent disease.

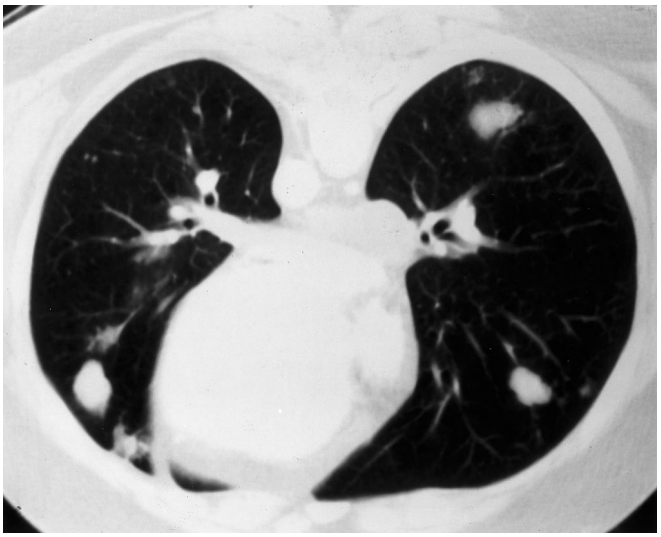

Fig. 1 CT scan showing bilateral intraparenchymal pulmonary tumour nodules

Materials and methods

Aspirate smears were ethanol fixed (95%) or air dried and stained by the Papanicolaou or May-Grunwald-Giemsa techniques, respectively. A portion of the biopsy tissue was preserved in 10% neutral buffered formalin (room temperature for approximately 6 h, embedded in paraffin, sectioned at 6 µm and stained with haematoxylin and eosin.

Immunohistochemical reactions were performed on an automated analyser (Techmate 1000, Ventana, Tuscon, Ariz.) according to the manufacturer's specified procedure. Heat-activated antigen retrieval was employed. The primary antibodies, their sources and dilutions are presented in Table 1. An additional portion of the biopsy was fixed in half-strength Karnovsky's fixative (2.5% glutaraldehyde, 2.0% paraformaldehyde), postfixed in osmium tetroxide and embedded in Spurr's resin according to conventional protocols.

An approximately cm^3 portion of tissue was procured for cytogenetic analysis. This was minced and treated with collagenase for 1 h. After disaggregation, a portion of the sample was washed, re-

Table 1 Antibodies used for immunohistochemistry

Antibody	Type	Source[a]	Dilution	Reaction
Cytokeratin (AE1/AE3)	M	BM	1:150	+
Cytokeratin (CAM 5.2)	M	BD	neat	–
Chromogranin	M	H	1:5000	–
Vimentin	M	Dako	1:200	+++
Synaptophysin	M	S	neat	+++
Desmin	M	B	1:40	+++
HMB45	M	E	1:100	–
HBA-71 (Ewing's sarcoma)	M	S	1:100	–

[a] *BM* Boehringer Mannheim, Indianapolis, Ind., *BD* Becton Dickenson, San Jose, Calif., *H* Hybritech, Westbrook, Me., *Dako* Dako, Santa Barbara, Calif., *S* Signet, Malvern, Pa., *B* Biogenex, San Ramon, Calif., *E* Enzo, New York, NY

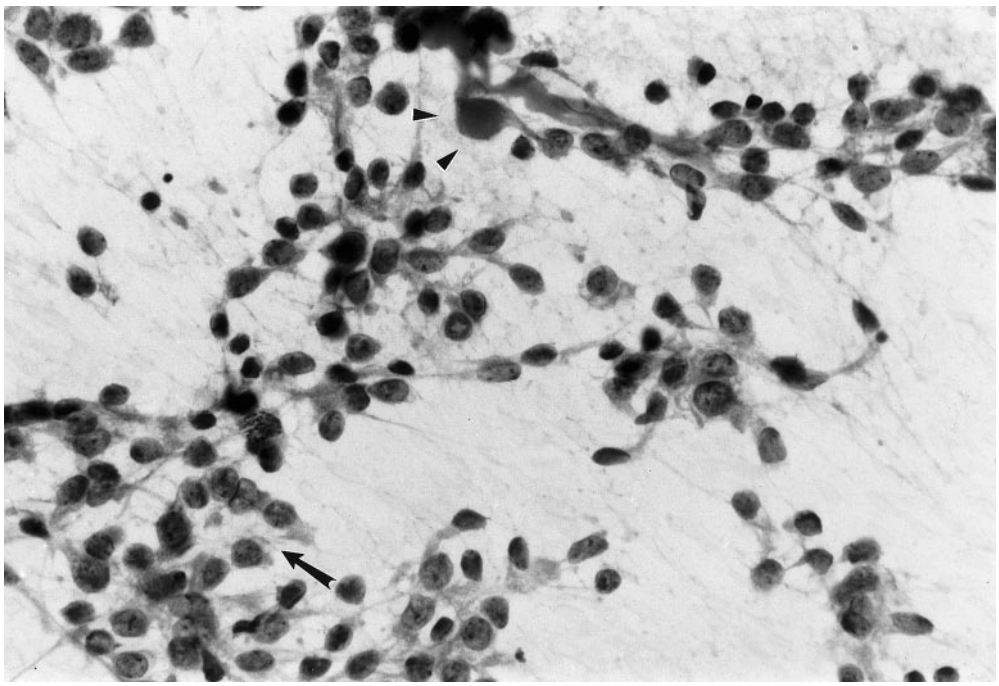

Fig. 2 Papanicolaou-stained smear from FNA of lesion. Note small stromal fragment (*arrowheads*) and rosette formation (*arrow*)

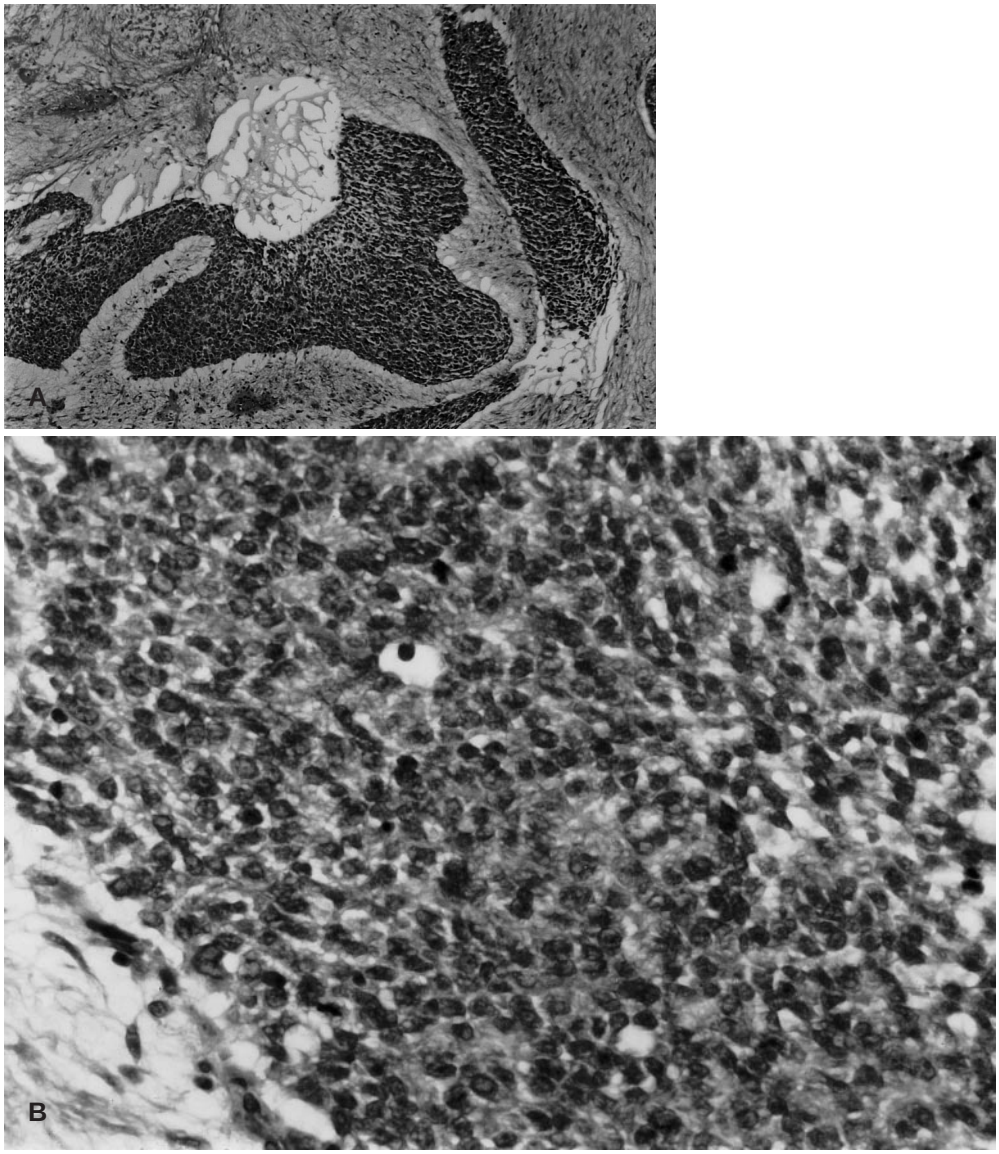

Fig. 3 **A** Sharply outlined nests/trabeculae of small round undifferentiated cells separated by myxomatous "desmoplastic" stroma. **B** Higher magnification (×400) of tumour, demonstrating "small round blue cells" with frequent mitotic figures

suspended in culture medium and harvested. The remainder of the sample was plated onto cover slips and allowed to grow for 1 week. Karyotypes for the direct harvest and 1-week cultures were obtained.

RNA extraction from frozen tissue, reverse-transcriptase PCR (RT-PCR), isolation, cloning and sequencing of amplified products were performed as described elsewhere [4, 11].

Results

Smears from the fine-needle aspiration showed a dispersed, loosely cohesive, population of epithelioid cells with round, oval nuclei with diameters approximately 1.5–2.0 times the diameter of a red blood cells, surrounded by a scant to moderate amount of delicate, wispy cytoplasm. Many of the cells had a plasmacytoid appearance. Intact single tumour cells and bare, naked nuclei were present. The tumour cells often formed sheets, clusters, loosely cohesive rosettes, and arborescent structures with small vessels (Fig. 2). They had delicate, powder-like, evenly distributed chromatin with occasional tiny, inconspicuous nucleoli, better appreciated on the Papanicolaou stain slides. Nuclear moulding and mitotic figures were present, but were not prominent features. The cytoplasm of the cells stained light green with Papanicolaou and light grey-blue with microvacuoles with May-Grunwald-Giemsa. In addition, many of the aggregates of tumour cells had a delicate, magenta coloured, reticulated and cord-like, extracellular matrix surrounding individual tumour cells and clusters of tumour cells on the MGG-stained material.

The excisional biopsy specimen taken from the left axilla was composed of an ovoid lymph node measuring 4.2 cm in its greatest dimension. The cut surface was tan to white, lobulated and mucoid; no areas of necrosis or calcification were grossly apparent.

The tumour was composed of sharply outlined nests, clusters and trabeculae of small round to oval cells separated by a myxomatous, desmoplastic stroma (Fig. 3A, B). The cells were relatively uniform, with scant cytoplasm and small hyperchromatic nuclei with inconspicu-

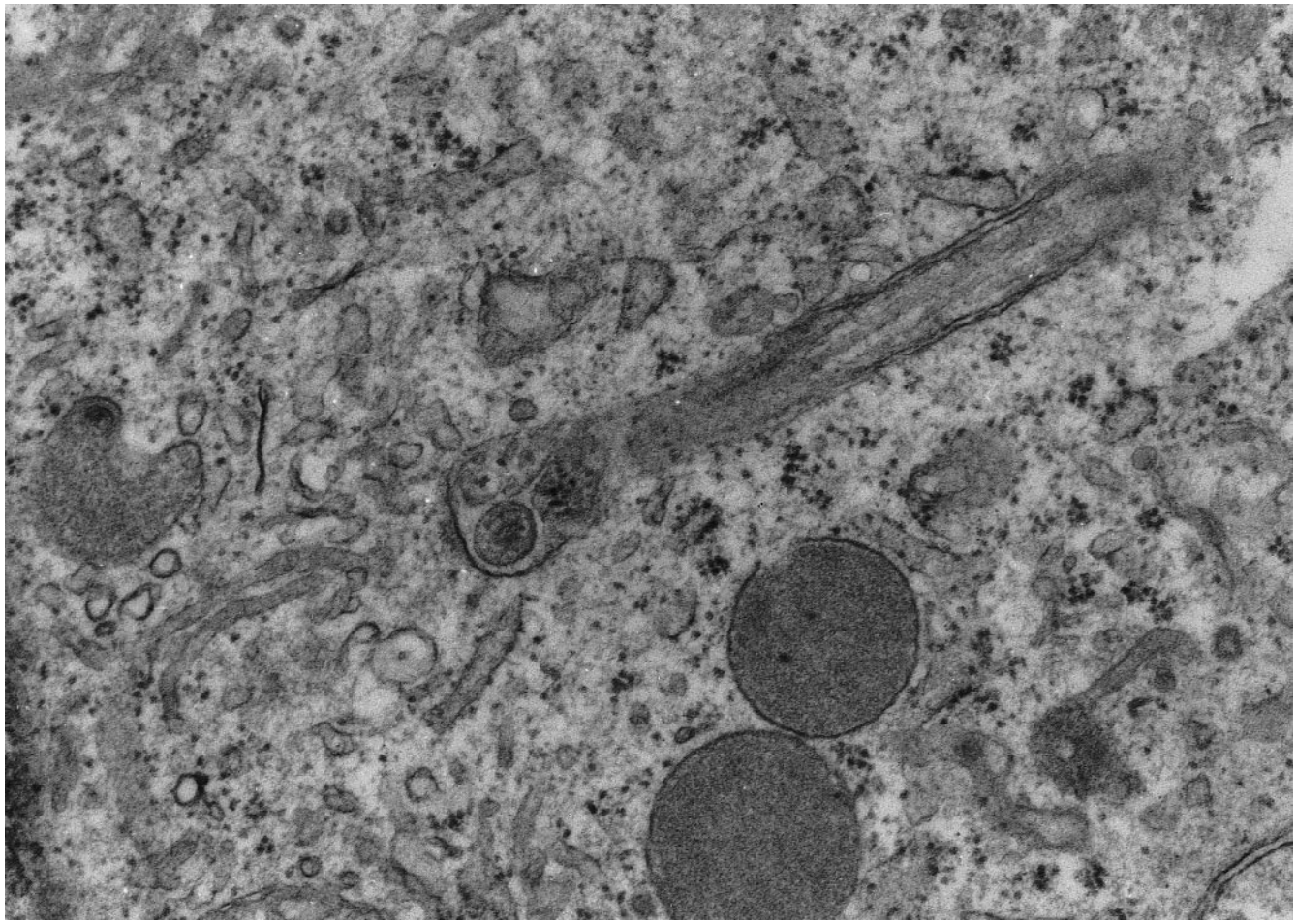

Fig. 4 Electron photomicrograph of tumour cell, showing a solitary intracytoplasmic cilium. ×50000

ous nucleoli. Mitoses were frequent, with nuclear apoptosis and microscopic foci of necrosis. Occasional rosettes were identified. The desmoplastic stroma was composed of elongated spindled cells with fibroblastic features. Lymph node architecture was entirely effaced. Table 1 shows the immunohistochemical reaction profile. Cytokeratin reactivity was present as a focal, perinuclear cytoplasmic dot.

Ultrastructurally, the tumour was composed of cells with smooth nuclear contours and high nuclear-to-cytoplasmic ratios. The chromatin was evenly dispersed, with rare, inconspicuous nucleoli. The cytoplasm contained mitochondria of normal morphology, well-developed Golgi apparatus and numerous ribosomes. Desmosomes and desmosome-like junctions were present. Several cells contained neurosecretory-like granules. Intermediate filaments were present randomly distributed in the cytoplasm. An intracytoplasmic cilium was identified in one of the tumour cells (Fig. 4).

The direct harvest yielded cells with the karyotype 47,XY,+8,–10, t(11;14)(q24;q21),t(17;22)(p12;q12),+20 (Fig. 5). Cells cultured for 1 week yielded cells with a normal male karyotype, 46,XY.

RNA isolated from frozen tumour tissue was analysed by RT-PCR for the presence of chimaeric transcripts corresponding to either EWS-FLI1 or EWS-WT1 gene fusions. The EWS-WT1 chimaeric transcript was detected by RT-PCR using primers for EWS exon 7 and WT1 exon 10 (Fig. 6). Cloning and sequencing of PCR products in this case showed in-frame fusion of EWS exon 7 to WT1 exon 8. No chimaeric transcripts corresponding to the EWS-FLI1 gene fusion were detected.

Discussion

Desmoplastic small round cell tumor (DSRCT) is now a well-characterized neoplasm with distinctive clinical, histological and immunohistochemical findings [9, 18, 22, 23, 28]. The vast majority of cases have involved the intra-abdominal peritoneum, although some arising from the tunica vaginalis [25], the pleura [2, 24], the ovary [36] and the central nervous system have been reported [34]. This last case is the first reported that does not arise from a serosal surface. In our case, although multiple intraparenchymal pulmonary and brain lesions were documented by CT scan, there was no evidence of pleural involvement; nor was there evident disease in the peritoneum of the pelvis. The axillary tissue excision showed tumour replacing lymph node, with a rim of residual lymph node

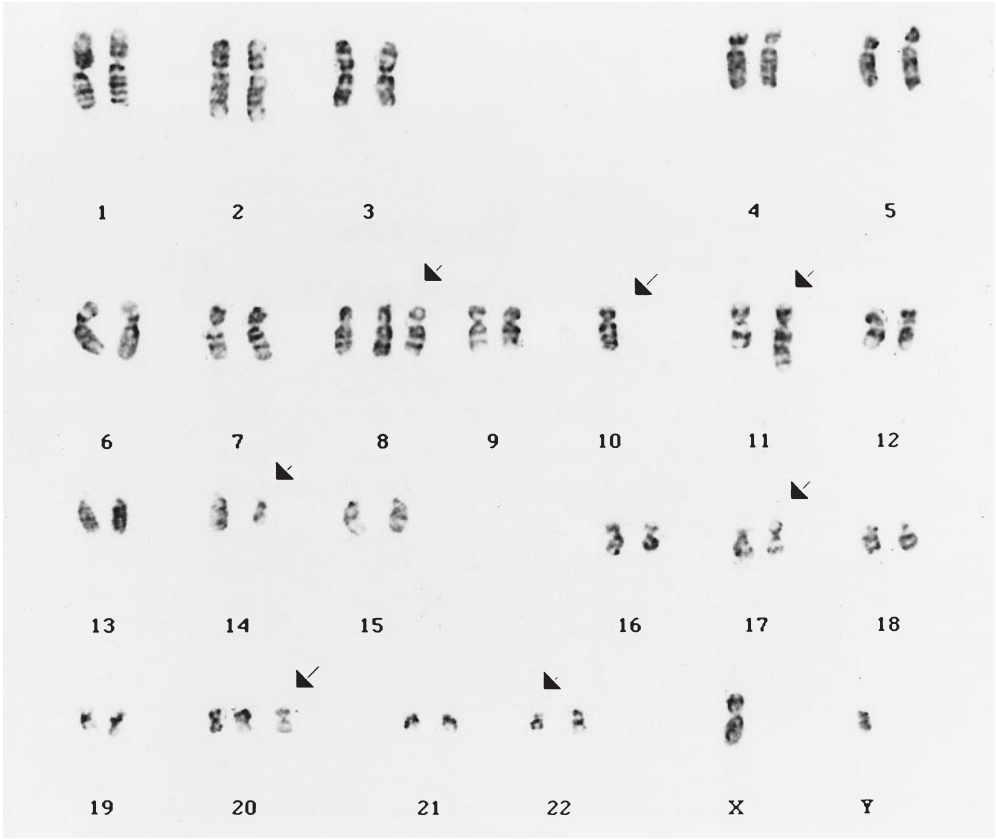

Fig. 5 G-banded karyotype from a direct harvest, 47,XY,+8,–10, t(11;14)(q24;q21), t(17,22)(p12;q12),+20

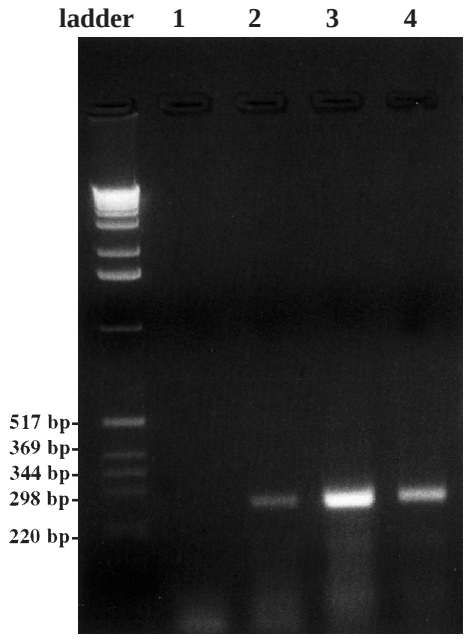

Fig. 6 RT-PCR analysis of tumour RNA for EWS-WT1 chimeric transcripts. The *panel* is a photograph of the ethidium bromide-stained agarose gel on which products of the PCR were electrophoresed. The size of DNA fragments in the marker ladder are shown to the *left* of the *panel*. *Lane 1* negative control (no added RNA), *lane 2* RNA from the present case as template; *lanes 3, 4* RNA from two different cases of desmoplastic small round cell tumor as template. The 270-bp fragment is the expected size of the RT-PCR product corresponding to the most commonly detected EWS-WT1 chimeric RNA transcript

tissue. Although a multicentric origin of the tumour is possible, because of multiple lymph node involvement and lung and brain lesions in a distribution suggestive of metastatic spread, we felt that the neoplasm would be best classified as of unknown primary origin.

Cytologically, like other small round blue cell tumours, the present case revealed small tumour cells with scant to moderate eccentric cytoplasm and nuclei with delicate chromatin patterns and occasional inconspicuous nuclei. Other case reports have described cytological features such as round to oval hyperchromatic nuclei [2], occasional prominent nucleoli and irregular and variable nuclear contours [2], features not present in this case. Loose rosette-like and arborescent vascular structures had not hitherto been described cytologically in a tumour of this kind. In addition, although “intercellular amorphous material” has been described in DSRCTs [29], the present case exchibited a prominent reticulated thread and cord-like extracellular matrix in the May-Grunwald-Giemsa-stained material. Cytological descriptions of this and previous cases [2, 29] suggest that variable nuclear and extracellular patterns may be expressed.

Immunohistochemically, Ewings sarcoma, PNET, Wilms’ tumour and DSRCT overlap, with all expressing vimentin. Cytokeratin, although uncommon, has been reported in a subpopulation of cells in both Ewing’s sarcoma [20] and PNET [13, 30]. Co-expression of vimentin and cytokeratin in the same tumour cell has been demonstrated most recently by immunoelectron microscopy in a case of extrarenal (Wilms’ tumor [21]).

The ultrastructural features of desmoplastic round cell tumour, as described by Gerald et al. [10], are not specific. Tumours with and without electron microscopic evidence of neuroendocrine differentiation (neurosecretory granules) have been described. The finding of cilia in the current case is of interest; although well described in Wilms' tumour, this ultrastructural feature has not previously been reported in desmoplastic round cell tumours. The significance of solitary cilia in tumour cells is unclear. Early investigators proposed that the existence of cilia may indicate a reversion to a more primitive condition, since cilia are found more commonly in the excretory ducts of lower animals and in the biliary epithelial cells of several nonmammalian vertebrates [12, 14, 17]. Other authors have proposed that the stimulation of centriolar reproduction without subsequent mitosis may lead to production of a cilium [19]. Although solitary cilia have been found in neoplasms of diverse origin [7], it is intriguing that cilia have now been described in two variants of small round blue cell tumour.

The 22q12 breakpoint seen in the karyotype of this case is a consistent finding in several small round blue cell tumours. Studies of Ewing' sarcoma have shown a characteristic translocation t(11;22)(q24;q12) [1, 32, 35]. This same translocation is seen in PNET [31]. Molecular studies have shown this translocation results in the fusion of the EWS gene on chromosome 22 with the FLI-1 gene from 11q24 [5]. This same EWS gene is part of a fusion gene in the other translocations that have been seen in cases of Ewing's sarcoma, such as t(21;22)(q22;q12), which produces an EWS-ERG gene fusion [3, 32]. Another, alternate, Ewing's sarcoma translocation is t(7;22)(p22;q12) [32], which fuses EWS to ETV1. Other tumours showing involvement of the 22q12 breakpoint include extra skeletal myxoid chondrosarcoma, t(9;22) [33], malignant melanoma of soft parts or soft tissue clear cell sarcoma, t(12;22) [8], and undifferentiated sarcoma of infancy, t(17;22) [15].

In other cases of DSRCT the chromosome 11 breakpoint has been seen at 11p13, the site of the Wilms' tumour suppressor gene [6, 26]. The karyotype of this tumour did not show the same derived 11 or derived 22 as seen in previous cases of DSRCT. Both the 11 chromosomes appear to have normal short arms and the derived 22 is smaller than it would be if the 11p13 to 11p terminus was translocated to the 22q12 band. However, a cryptic translocation in a case of Ewing's sarcoma, with molecular evidence of the EWS-ERG fusion, has been reported [16]. It is likely that several additional rearrangements have taken place in our case, as the loss of a 10 and the trisomies for chromosomes 8 and 20 indicate changes attributable to tumour progression. These rearrangements have masked the expected t(11;22)(p13;q12) translocation previously associated with DSRCT, an event that may have occurred because the chimaeric product of this fusion gene in DSRCT has been identified by reverse transcriptase PCR. The presence of the EWS-WT1 chimeric transcript is indicative of a functional EWS-WT1 gene fusion. In this case, it has resulted from a complex chromosomal translocation which has not been identified by karyotype analysis [27].

Our report describes a DSRCT of unknown origin with metastases, and further supports the findings of Tison et al. [34] showing that DSRCT can arise in anatomical locations not associated with serosal surfaces. The features of this neoplasm demonstrate cytological, immunohistological, ultrastructural, cytogenetic and molecular characteristics similar to those of DSRCTs associated with serosal surfaces.

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