Table *Laboratory infection with parvovirus B19*

| *Laboratory* | *Age* | *Sex* | *Grade* | *Seroconversion* | *IgM* | *Clinical details* |
|---|---|---|---|---|---|---|
| London, England | 30 | Female | Scientist | Yes | Yes | Malaise and myalgia for two weeks starting four weeks after first laboratory exposure, then transient rash (< 12 hours) and arthralgia. Myalgia and malaise persisted for a further four weeks |
| Paris, France[2] | 31 | Female | Technician | Yes | Yes | Vascular purpura on legs; 8 weeks pregnant—miscarriage |
| | 23 | Female | Technician | Yes | Yes | No symptoms |
| | 26 | Female | Technician | Yes | Yes | Transient rash |
| Munich, FRG[3] | 26 | Female | Technician | Yes | Yes | Malaise and myalgia for two weeks then rash and arthralgia for three weeks, and headaches for four weeks starting after the first week of the rash |
| Fukuoka, Japan | 31 | Male | Medical | Non tested | Yes | Fever (38·2°C) two weeks after first laboratory exposure, followed by rash (three weeks) and arthralgia (four weeks) |
| Washington, USA | 30 | Male | Medical | Yes | Yes | B19 DNA in serum, fever, arthralgia of knees, ankles, wrists, and lower back lasting three to four days. Neutropenia, reticulocytopenia |

infections were occupationally acquired the timing of the illnesses strongly suggests this. We propose three measures that might reduce the occupational risk from B19 virus.

Firstly, inactivate the antigen. Diagnostic tests for B19 virus infection are at present done with antigen prepared from the plasma of viraemic patients and this material has not been inactivated until now.[4] Initial studies done by one of us (BJC) indicate that doses of up to 2·4 Mrads gamma irradiation do not destroy the antigenicity of B19 virus. This treatment destroyed the infectivity of 30 viruses.[5] Inactivation of current supplies of B19 antigen by irradiation is therefore probably feasible. In the near future it is likely that a non-infectious B19 antigen will be provided by recombinant DNA technology.[6]

Secondly, keep aerosols in a safety cabinet. B19 virus has been transmitted experimentally by the respiratory route,[7] and it seems likely that this was the route of spread for the probable laboratory infections reported here. Infectious aerosols could have been generated by centrifugation, during the resuspension of virus pellets, or in washing stages of solid phase immunoassays. Where possible such procedures should be done in an exhaust protective cabinet.

Thirdly, find out if the laboratory personnel are immune. It may not be necessary to restrict work with parvovirus B19 to those known to be immune, but a strong case can be made for finding out if staff are immune, and excluding pregnant women. Exposure to B19 virus during pregnancy may carry an increased risk of abortion (Hall SM, Anderson MJ, Caul EO, *et al.* Paper presented at the joint meeting of the European Association against Virus Diseases, Switzerland, 1987).

BJ COHEN*
AM COUROUCÉ†
TF SCHWARZ‡
K OKOCHI§
GJ KURTZMAN‖
**Virus Reference Laboratory, Central Public Health Laboratory, London NW9 5HT*
*†Centre National de Transfusion Sanguine, Etablissement Cabanel, 6 rue Alexandre Cabanel 75739 Paris, Cedex 15, France*
*‡Max v Pettenkofer Institut, Pettenkoferstr 9a, 8000 Munchen 2, FRG*
*§Clinical Laboratory, Kyushu University Hospital, 3-1-1 Maidashi Higashi-ku, Fukuoka T 812, Japan*
*‖Clinical Haematology Branch, National Institutes of Health, National Heart, Lung and Blood Institute, Bethesda, Maryland 20892, USA*

**References**

1 Grist NR, Emslie J. Infections in British clinical laboratories, 1984–1985. *J Clin Pathol* 1987; **40**:826–9.
2 Lefrere J-J, Dumez Y, Courouce A-M, Deschene G. Intrauterine infection with human parvovirus. *Lancet* 1986;i:449.
3 Schwarz TF, Roggendorf M, Deinhardt F. Haufigkeit der Parvovirus-B19-Infektionen (prevalence of parvovirus B19 infections). *Dtsch Med Wochenschr* 1987;**112**:1526–31.
4 Cohen BJ, Pereira MS, Mortimer PP. Diagnostic assays with monoclonal antibodies for the human serum parvovirus-like virus (SPLV). *J Hyg (Cambridge)* 1983;**91**:113–30.
5 Sullivan R, Fossolitis AC, Larkin EP, Read RB, Peeler JT. Inactivation of thirty viruses by gamma radiation. *Applied Microbiology* 1971;**22**:61–5.
6 Sisk WP, Berman ML. Expression of human parvovirus B19 structural protein in E. coli and dectection of antiviral antibodies in human serum. *Biotechnology* 1987;**5**: 1077–80.
7 Anderson MJ, Higgins PG, Davis LR & Experimental parvoviral infection humans. *J Infect Dis* 1985;**152**:257–65.

# Other correspondence

## Sarcoma-like nodules in cystic ovarian tumours

We read with interest the recent report concerning sarcoma-like mural nodules in cystic serous ovarian tumours, and noted particularly the second case where undifferentiate sarcoma was found arising within serous cystadenocarcinoma.[1] We have recently encountered a similar tumour which occurred in a 44 year old premenopausal multiparous woman with a history of neurofibromatosis.

The patient presented with a few months' history of abdominal discomfort. At laparotomy she was found to have bilateral ovarian masses, and widespread pelvic and abdominal metastases with ascites. Hysterectomy and bilateral salpingo-oophorectomy were carried out and the patient started on chemotherapy.

Each ovary was replaced by a cyst, the larger measuring 23 cm in diameter. On histological examination these showed moderately differentiated serous cystadeno-

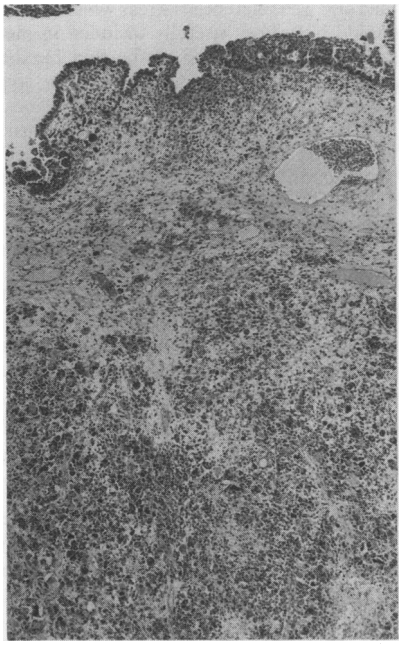

Fig 1 *Surface papillary carcinoma with underlying area showing sarcomatous differentiation (Haematoxylin and eosin).*

ocarcinoma with papillae and psammoma bodies. Examination of metastases showed adenocarcinoma. Several nodular masses lining the inner surface of the larger cyst were also adenocarcinoma. One nodule, however, showed sarcomatous differentiation (fig 1) with numerous desmin positive cells on immunohistochemical staining, both within the nodule and admixed with contiguous epithelial tumour. PTAH staining showed rhabdomyoblasts with cross striations (fig 2), which were confirmed by electron microscopy. Areas of liposarcoma were also found (fig 3). No evidence of sarcoma was found in the contralateral ovarian tumour or in the metastases. No teratomatous organoid growth pattern or neuroepithelium was found in the tumour. In addition, there was no evidence of endometriosis. Myometrial lymphatics were infiltrated with carcinoma, and the endometrium showed a normal secretory pattern. The patient was alive six months after her initial presentation.

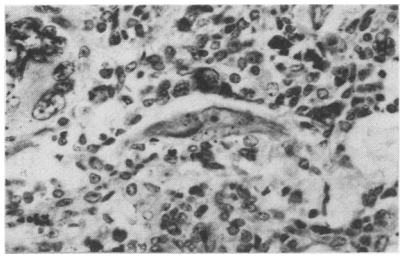

Fig 2 *Rhabdomyoblast showing cross striations (PTAH).*

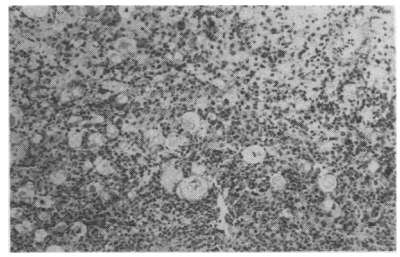

Fig 3 *Area of lipoblastic differentiation showing vacuolated cells with typical nuclear scalloping (Haematoxylin and eosin).*

We consider that this tumour is an early form of ovarian malignant mixed mesodermal tumour, and as such is unusual in that these lesions tend to present in older nulliparous patients. We also feel that sarcomatous mural nodules in cystic serous ovarian tumours may be more common than published reports would suggest, and that their true incidence may be detected by more extensive sampling of any nodular masses found in the walls of serous tumours. Furthermore, it is of interest that this tumour should arise in a patient with neurofibromatosis, a syndrome associated with an increased incidence of a variety of malignancies, including ovarian cancers.[2]

J MCCULLOUGH
P BATMAN
D PARKER
*Bradford Royal Infirmary, Bradford BD9 6RJ*

**References**

1 Clarke TJ. Sarcoma-like mural nodules in cystic serous ovarian tumours. *J Clin Pathol* 1987;**40**:1443–8.
2 Sorenser SA, Mulvihill JJ, Milsen A. Long term follow-up of von Recklinghausen neurofibromatosis. *N Engl J Med* 1986; **314**:1010–14.

## Histopathology of benign breast lesions

We read with interest the recent paper by Barnard *et al*[1] about their experience of the histopathology of benign non-palpable breast lesions identified by mammography. We were, however, concerned that no mention was made of specimen radiography. We regard this as essential to confirm that the mammographic abnormality has indeed been removed. This applies even when needle localisation is used. Specimen radiography is most helpful in confirming the presence of calcifications but may be less satisfactory in the case of a density of a distortion of travecular architecture. None the less, it is still an important step in the handling of non-palpable breast lesions identified by mammography. The specimen radiograph should also be used by the pathologist as a guide when sampling the specimen for histological examination.

With the advent of breast screening throughout the country, the number of mammographically indicated biopsy specimens taken will increase and it is important that the excised tissue should be handled in such a way as to yield maximum information and correlate mammographic and histological appearances.

RR MILLIS
AC GIRLING
IS FENTIMAN
*Imperial Cancer Research Fund Clinical Oncology Unit, Guy's Hospital, London SE1 9RT.*

**Reference**

1 Barnard NJ, George BD, Tucker AK, Gilmore OJA. Histopathology of benign non-palpable breast lesions identified by mammography. *J Clin Pathol* 1988;**41**:26–30.

Drs Barnard, George, Tucker and Gilmore reply:

We fully agree with the comments of Drs Millis, Girling and Fentiman about specimen radiography. At the beginning of our study period we did not routinely use it. We have, however, been doing specimen radiography for some time and though our previous results are comparable with our present ones, we feel much more confident that we are obtaining the maximum information from the specimens in the most efficient way.

NJ BARNARD,
BD GEORGE,
AK TUCKER,
OJA GILMORE
*St Bartholomew's Hospital, London EC1A 7BE*