

CAP ID # 7186701
CLIA ID # 99D1030993
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SAMPLE REPORT

Clinical:

77 y.o. female with a history of breast CA diagnosed in 1994, status post several rounds of chemotherapy with gemzar,xeluda,taxotere,navelbine.

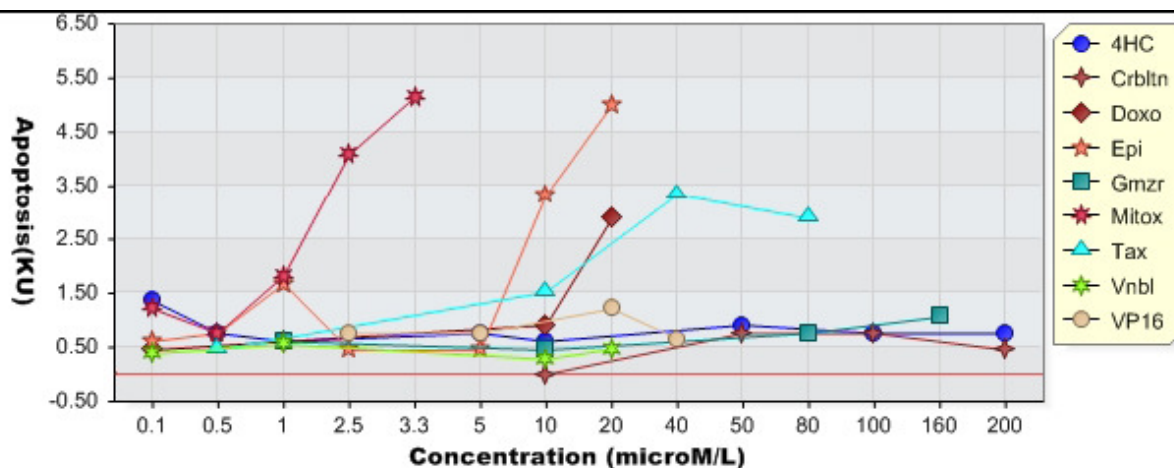
INTERPRETATION:

Pleural fluid, drainage:

1. A population of cells with morphologic and immunocytochemical features consistent with an epithelial neoplasm is identified (see comment).
2. In the MiCK assay, Mitoxantrone was both the most effective and most potent inducer of apoptotic death in the patient's cells (see comment).
3. Responses to other tested agents were consistent with variable levels of sensitivity of the patient's cells to these compounds (see comment).

Maximum Apoptotic Response (Kinetic Units):

Mitox	Epi	Tax	Doxo	4HC	VP16	Gmzr	Crbltn	Vnbl
5.15	5.00	3.33	2.88	1.36	1.21	1.06	0.91	0.56



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COMMENT:

Approximately 650 mL pleural fluid specimen received in the lab. The specimen contained viable cells and it was sufficient for the study. Wright stained cytopsin preparations of the pleural fluid showed medium sized cells with nuclear irregularities. Immunocytochemical studies demonstrated these cells were positive for estrogen and/or progesterone receptors. Intermixed with the infiltrate were lymphoid cells, rare mesothelial cells, neutrophils, and eosinophils. In the appropriate clinical settings, these findings could be consistent with a malignant pleural effusion secondary to a metastatic breast carcinoma.

Neoplastic cells were purified and tested for their sensitivity to multiple doses of VP16, Doxorubicin (Doxo), Gemzar (Gmzr), Carboplatin (Crbpltn), Vinblastine (Vnbl), Taxotere (Tax), Cytoxan (4HC), Epirubicin (Epi), and Mitoxantrone (Mitox) as single agents (see chart above). In the MiCK assay, Mitoxantrone was both the most effective and most potent inducer of apoptosis in patient's cells. Extent of apoptosis induced by Epirubicin was similar to that of Mitoxantrone, however it was achieved at higher doses of the drug (see chart above). Apoptosis induced by Taxotere was at the level that was previously seen in patients with partial clinical response to this agent. Other tested agents were less effective in inducing apoptosis in the patient's cells. A table in the "Interpretation" section shows maximal apoptotic responses achieved with each agent.

In conclusion, if single agent chemotherapy of this patient is clinically indicated, the results of this study would justify including Mitoxantrone in the treatment protocol as the most effective and potent inducer of apoptosis in the patient's cells.

All tested chemotherapeutic agents induced apoptosis in a control cell line.

Doctor was contacted regarding the results of the study on 00/00/0000.

MICROSCOPIC/IMMUNOPHENOTYPIC STUDIES:

The report was faxed to Doctor on 00/00/0000.

Attending Pathologist
Phone: 123-456-7890

Electronically signed on 00/00/0000

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The pathologist's signature on this report indicates that the case was personally reviewed and the findings confirmed by the attending pathologist. This test was performed at DiaTech Clinical Pathology Laboratory. This laboratory is certified under CAP and CLIA-88 and is qualified to perform high complexity clinical testings. The MiCK assay measures drug induced apoptosis and its performance characteristics were determined at Vanderbilt University and at DiaTech Oncology. Clinical use of the MiCK assay is based on a statistically significant increase in CR rate and overall survival of AML patients whose treatment protocol included a drug to which the patient's tumor cells were sensitive in the assay. When used with solid tumors, the MiCK assay is expected to identify drugs most effective in killing patient's tumor cells by apoptosis. This test has not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that such approval was not required.