

CAP ID # 7186701
CLIA ID # 99D1030993
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SAMPLE REPORT

Clinical:

45-year-old male with a history of the prostate carcinoma, now presenting with metastatic disease.

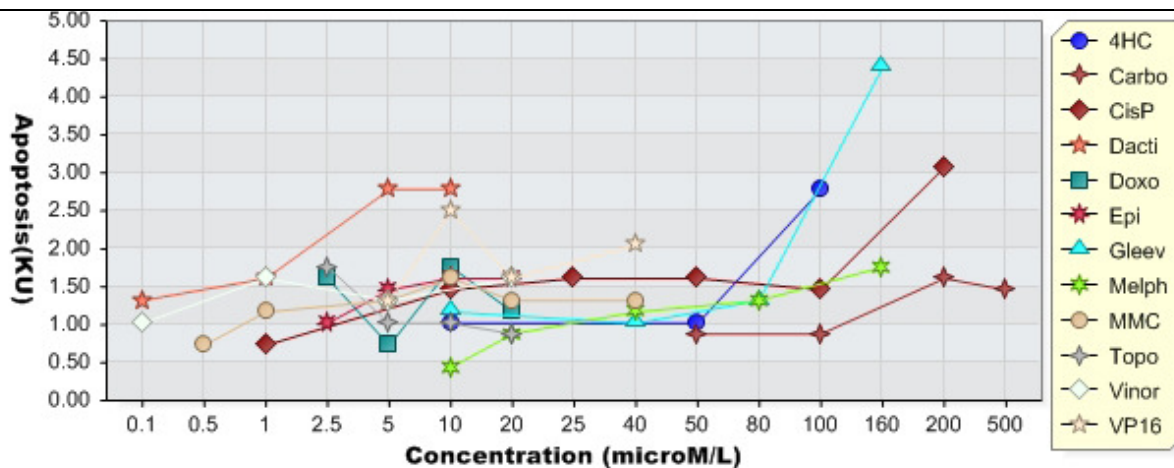
INTERPRETATION:

Liver, exisional biopsy:

1. Population of cells with morphologic and immunocytochemical features consistent with an epithelial neoplasm is identified (see comment).
2. In the MiCK assay, the patient's tumor cells were most sensitive to Gleevec (see comment).
3. Responses to other tested agents were consistent with lower sensitivity of the patient's cells to these compounds (see comment).

Maximum Apoptotic Response (Kinetic Units):

Gleevec	CisP	Dacti	4HC	VP16	Topo	Melph	Doxo	Epi	MMC	Carbo	Vinor
4.39	3.07	2.78	2.78	2.49	1.76	1.76	1.76	1.61	1.61	1.61	1.61



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COMMENT:

Purified viable neoplastic cells were tested for their sensitivity to multiple doses of Cytoxan (4HC), Ifosfamide (4HI), Taxol (Txl), Taxotere (Tax), Doxorubicin (Doxo), Epirubicin (Epi), Mitoxantrone (Mitox), Cisplatin (CisP), Carboplatin (Carbo), Gemzar (Gmz), Topotecan (Topo), Irinotecan (CPT11), Gleevec (Gleev), Dactinomycin (Dacti), Etoposide (VP16), Melphalan (Melf), Mitomycin (MMC), Vinorelbine (Vinor), Oxaliplatin (Oxali), and Fluorouracil (5FU), as single agents. Of note, alkylating agents Cytoxan and Ifosfamide require metabolic transformation by hepatocytes and, thus, cannot be tested in vitro. In this study, active metabolites of the two drugs (4HC and 4HI) were used.

The MiCK assay identifies drugs most effective in killing patient's tumor cells by apoptosis. Extent of drug-induced apoptosis is measured in Kinetic Units (KU). In this study, Gleevec was the most effective inducer of apoptotic death in the patient's cells causing 4.39 KU maximal apoptotic response. In a limited flow cytometry study, the patient's tumor cells showed positivity for c-kit (CD117). This finding justified including Gleevec in the testing panel. Of note, responses from 3 to 5 KU are consistent with a moderate drug sensitivity of the tumor cells and have been previously seen in patients with partial clinical response to chemotherapy. Cisplatin was the second best inducer of apoptosis causing 3.07 KU maximal response. Responses to Dactinomycin, Cytoxan, and Etoposide were consistent with a moderately low sensitivity of the tumor cells to these agents. Responses to Topotecan, Melphalan, Doxorubicin, Epirubicin, Mitomycin C, Carboplatin, and Vinorelbine were consistent with low sensitivity of the tumor cells to these agents. Other tested compounds, including Taxotere, Taxol, Oxaliplatin, Mitoxantrone, Ifosfamide, Gemzar, CPT11, and 5FU induced less than 1.5 KU maximal response and are not shown in the table in the "Interpretation" section.

In conclusion, results of this study would support including Gleevec in the treatment protocol if clinically indicated. Based on the pattern of the dose response curve of Gleevec, use of the drug in its maximal tolerable dose should be considered. Based on the results of this study, combination of Gleevec with Cisplatin would also be reasonable to consider if clinically indicated.

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

MICROSCOPIC/IMMUNOPHENOTYPIC STUDIES:

Wright stained cytospin preparations of the disaggregated tissue showed predominantly small to medium sized atypical epithelioid cells with nuclear irregularities, nucleoli, and high N:C ratio, singly and in small aggregates. ICC studies showed these atypical cells were positive for cytokeratin and PSMA. Approximately 60-65% atypical cells expressed nuclear Ki-67. In appropriate clinical settings, these findings are consistent with involvement by a malignant neoplasm of epithelial origin. A limited flow cytometry study showed that neoplastic cells (CD45-negative) were positive for CD117 (c-kit), a finding seen in some of the neoplasms of mesenchymal and epithelial origin.

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

Attending Pathologist
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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.