

hospitals, distance and laboratory works or diagnostics for the Initial Physician Interval; waiting for laboratory work-ups for the First System Interval; and waiting for other departments' clearance for the Second System Interval. Length of time intervals was not found to be significantly associated with extent of surgery and final stage.

IGCS20_1080

109 CORRELATION OF HPV GENOTYPING, P16 AND HPV IMMUNOHISTOCHEMISTRY WITH CLINICOPATHOLOGICAL FEATURES IN VULVAL SQUAMOUS CELL CARCINOMAS

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Introduction Human papilloma virus (HPV) associated vulval squamous cell carcinoma (VSCC) has better prognosis than HPV-independent VSCC. Ancillary tests are necessary as morphology alone is unreliable in differentiating these.

Methods We analysed 18 VSCC cases by comparing their morphology with three ancillary tests performed to identify the HPV status.

Results Basaloid/warty type VSCC considered to be HPV-associated; accounted for 6/18 cases (33.3%). Of these, 5 cases (83.3%) showed block p16 positivity and 3 cases (50%) had a positive HPV genotyping result.

Keratinising VSCC considered to be HPV-independent; accounted for 11/18 cases (61.1%). Amongst these, 3 cases (27.3%) showed block p16 positivity and 2 cases (18.2%) had positive HPV genotyping results. In one case, the VSCC was too small to assess the morphology.

Usual type VIN considered to be HPV-associated, was seen in 11/18 cases (61.1%). Of these, 8 cases (72.7%) showed block p16 positivity and 6 cases (54.5%) had a positive HPV genotyping result.

Differentiated VIN considered to be HPV-independent, was seen in 4/18 cases (22.2%). Amongst these, 0 cases (0%) showed block p16 positivity and 0 cases (0%) had a positive HPV genotyping result.

HPV genotyping detected high risk HPV in 6/18 cases (33.3%) and all these cases showed block p16 positivity.

In 3/9 cases (33.3%), with block p16 positivity, genotyping did not reveal any HPV.

HPV immunohistochemistry was negative in all cases.

Conclusion Our study illustrates that in the absence of a gold standard test for HPV, p16 immunohistochemistry and HPV genotyping are complimentary ancillary tests in VSCC.

IGCS20_1081

110 A NEW INSIGHT INTO RESOLVIN E3 AS THE ITRACONAZOLE INDUCED ANTICANCER EFFECT ON CERVICAL CANCER

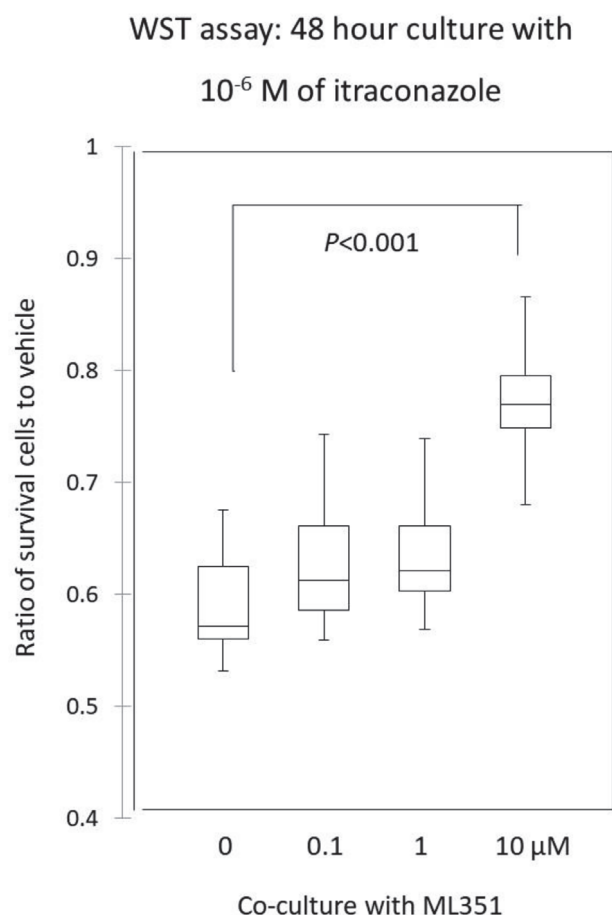
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We have been studying drug repositioning of itraconazole for an anticancer drug. Previously, we reported clinical trials of patients with various types of cancer, and a window opportunity trial is ongoing (jRCTs051190006). Using uterine cancer cells, itraconazole inhibited cancer growth by downregulation of signal transduction (Akt/mTOR, hedgehog, Wnt/ β -catenin). In this report, we investigated the bioactive lipid mediators (LMs) associated with itraconazole induced anti-cancer effect.

Methods CaSki cervical cancer cells before and after exposure to itraconazole were scraped and stored at -30 °C. LC-MS/MS-based metabololipidomics were performed. Deuterated internal standards representing each chromatographic region of identified lipid mediators were added to samples to facilitate quantification. The samples were extracted by automated SPE system on C18 columns and were then subjected to LC-MS/MS analysis with a Qtrap 6500 (Sciex) connected with a Shimadzu LC-30AD HPLC system. Biochemical pathway for LMs which increased more than 2 fold or decreased less than a half either at 30 min and 60 min after incubation with 10^{-6} M itraconazole were subjected to the cell growth inhibitory experiments using WST assay.

Results Among downstream metabolites of eicosapentaenoic acid, resolvin E3 and resolvin E2 increased over 2 fold at 30 min and at 60 min, respectively. Coculture with 10M ML351, 12/15-LOX inhibitor responsible for the metabolism from 18-HEPE to resolvin E3, did not effect on the growth of CaSki cells. Coculture with 10M ML351 and 10^{-6} M itraconazole attenuated the growth inhibitory effect of itraconazole (figure 1).



Abstract 110 Figure 1