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The effects of boric acid and disodium pentaborate dechydrate in metastatic prostate cancer cells

Boron and their derived molecules have prevention or treatment potential against prostate cancer. In this study, we aim to investigate the effects of Boric acid (BA) and Disodium Pentaborate Dechydrate (DPD) in metastatic prostate cancer cells such as DU-145 which is brain metastatic prostate cancer, and PC3 which is bone metastatic prostate cancer.
Metastatic human prostate cancer cell lines, PC-3 and DU-145, were used to show whether inhibition effects of BA and DPD on prostate cancer cells in this study. BA and DPD were applied for 24 hours to the cells. Cell viability determination was performed using WST-1 assay. Apoptotic cell death was evaluated with Annexin-V/PI flow cytometric analysis and caspase-3 expression immunohistochemically. A wound healing assay was also used to measure cancer cell migration after exposure to BA and DPD.
Applying BA and DPD made inhibition of cell proliferation in both BA (1 mM) and DPD (7 mM) at 24 h. The results of Annexin-V/PI showed that DPD induced higher levels of apoptosis than BA in both prostate cancer cells. Caspase-3 expressions were also higher than BA with DPD in both metastatic prostate cancer cells. We evaluated cell migration using a wound healing assay and the result showed that cell migration was inhibited with BA and DPD in both cells.
Both BA and DPD inhibited the cell viability of metastatic prostate cancer cells. Apoptotic cell death with applying DPP had a higher rate than BA treatment. Moreover, BA and DPD inhibited cell migration in both cells when we compared them with control. This study's results showed that BA and DPD of boron derivates significantly induced cells to apoptosis and the migration was inhibited by the derived form of boron in metastatic prostate cancer cells.

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The value of bone scans to predict survival time in patients with diagnosed prostate cancer: single-center retrospective study

Objective: In this study, we investigated the significance of the bone scan results as a prognostic factor to predict survival by comparing age, serum PSA level, and Gleason score.
Methods: Medical records of 313 patients were retrospectively examined. 265 patients of 313 were included in the study.
Results: 202 (76%) patients of 265 were still alive and 63 (24%) patients of 265 were dead because of prostate cancer. Patients' mean estimated survival times for those with, without, and suspected bone metastases were 47.4 ± 5.4 months, 159.1 ± 8.6 months, and 71.1 ± 14.4 months, respectively (p = 0.0001). While the mean estimated survival time of < 70 years patients old was 137.1 ± 9.4 months, the mean estimated survival time of ? 70 years old patients was 78.2 ± 5.0
(p = 0.031). 243 patients with known PSA values, of those whose PSA levels were < 10 ng/ml, between 10-20 ng/ml, between > 20-50 ng/ml, and > 50 ng/ml, the estimated mean survival time was
106.9 ± 4.2 months, 118.1 ± 14.8 months, 87.6 ± 7.4 months and 51.7 ± 6.2 month, respectively and a significant difference was determined (p = 0.0001). For patients whose Gleason scores were < 7, 7,
and >7, the mean estimated survival time was 167.5 ± 10.8 months), 86.8 ± 5.5 months, and
61.0 ± 5.4 months, respectively, and a significant difference was determined (p = 0.0001).
Conclusion: We identified that the estimated mean survival time of the patients who had bone metastases, had a high level of PSA, had a high level of Gleason score, and were older than 70 years old was shorter than other groups. We concluded the most important prognostic factor affecting survival time independently was the finding of metastasis detected in bone scintigraphy.
