Evaluasi ekspresi VDAC1 dan hubungannya dengan ekspresi gen protein keluarga Bcl-2 pada galur sel kanker prostat PC-3 yang diinduksi apoptosis oleh Zinc = Evaluation of Expression VDAC1 and Its Relationship with Expression Bcl-2 family protein gene in strain Prostate Cancer Cells PC-3 Apoptosis Induced by Zinc

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Abstrak

[ABSTRAK
Latar Belakang: Kanker prostat adalah kanker yang paling umum pada pria. Kanker terjadi karena hilangnya kontrol atas proliferasi sel dan apoptosis sehingga sel berproliferasi terus menerus tanpa ada kematian sel. Apoptosis diregulasi oleh beberapa protein tertentu diantaranya protein keluarga Bcl-2 dan protein kanal. Perkembangan kanker prostat memerlukan transformasi dari sel epitel yang normal menjadi sel ganas yang kehilangan kemampuan untuk mengakumulasi zinc. Salah satu efek utama zinc adalah mencegah pertumbuhan sel kanker prostat dengan menginduksi apoptosis dengan memfasilitasi proses pembentukan pori Bax yang memulai apoptogenesis mitokondria. Selain keluarga Bcl-2, VDAC1 juga berperan penting dalam proses apoptosis. Beberapa penelitian menyatakan Bcl-2 mempunyai kaitan erat dengan VDAC1 terkait proses apoptosis dan protein pro-apoptotik Bax juga secara langsung berinteraksi dengan VDAC yang kemudian menginduksi keluarnya sitokrom c dari membran mitokondria.
Tujuan: Mengevaluasi ekspresi mRNA dari gen mengkode keluarga protein Bcl-2 (Bax dan Bcl-2) dalam proses apoptogenesis pada galur sel kanker prostat yg diinduksi oleh zinc; Mengevaluasi ekspresi mRNA dari gen VDAC1 dalam proses apoptogenesis pada galur sel kanker prostat yang diinduksi oleh zinc; Menganalisis hubungan antara ekspresi VDAC1 dengan protein keluarga Bcl-2 pada apoptogenesis galur sel kanker prostat.
Desain: Penelitian ini menggunakan eksperimental in vitro dan analisis statistik
Metode: Untuk memperbanyak galur sel kanker prostat (PC3) dilakukan kultur sel, kemudian diberi perlakuan dengan tiga kelompok (kontrol, zinc 20 M dan staurosporin 0,16 M). Selanjutnya dilakukan isolasi RNA dan elektroforesis RNA untuk mengetahui keutuhan RNA. Terakhir dilakukan qRT PCR yang kemudian datanya dianalisis secara statistika.
Hasil: Ekspresi Bax, Bcl-2 dan VDAC1 pada galur sel kanker prostat (PC-3) yang diberi perlakuan zinc mengalami penurunan dibandingkan dengan kontrol (tidak diberi perlakuan). Akan tetapi penurunan ekspresi tersebut tidak bernilai signifikan karena nilai p > 0,05 (nilai signifikansi Bax = 0,309; nilai signifikansi Bcl-2 = 0,236; nilai signifikansi VDAC1 = 0,437). VDAC1 mempunyai korelasi yang signifikan (p < 0,05) dengan Bax (p = 0,01) dibandingkan dengan Bcl-2 (p = 0,118).
Kesimpulan: Terjadi perubahan ekspresi pada setiap gen (Bax, Bcl-2 dan VDAC1) pada galur sel kanker prostat yang diberi perlakuan zinc dengan yang tidak diberi perlakuan, akan tetapi tidak bernilai signifikan. VDAC1 mempunyai korelasi yang bermakna dengan Bax dan mempunyai korelasi yang tidak bermakna dengan Bcl-2.
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ABSTRACT

Background: Prostate cancer is the most common cancer in men. Cancer occurs due to loss control of cell proliferation and apoptosis thus continuously proliferating cells without cell death. Apoptosis is regulated by specific proteins including Bcl-2 family proteins and channel proteins. The development of prostate cancer requires the transformation of normal epithelial cells into malignant cells that lose the ability to accumulate zinc. One of the main effects of zinc is to prevent the growth of prostate cancer cells by inducing apoptosis by facilitating the process of pore formation Bax that started apoptogenesis mitochondrial. In addition to Bcl-2 family, VDAC1 also plays an important role in the process of apoptosis. Some studies suggest Bcl-2 has close links with related VDAC1 apoptosis and pro-apoptotic protein Bax also directly interact with VDAC which then induces the release of cytochrome c from the mitochondrial membrane.
Objective: To evaluate the expression of mRNA of the gene encoding the Bcl-2 family proteins (Bax and Bcl-2) in the process apoptogenesis on prostate cancer cell line that is induced by zinc; Evaluate the mRNA expression of genes in the process VDAC1 apoptogenesis on prostate cancer cell line induced by zinc; Analyzing the relationship between the expression of VDAC1 with Bcl-2 family proteins in prostate cancer cell lines apoptogenesis.
Design: This study used an experimental in vitro and statistical analysis
Methods: To reproduce the prostate cancer cell lines (PC3) performed cell culture, then treated with three groups (control, zinc 20 M and staurosporin 0,16 M). Furthermore, the isolation of RNA and RNA electrophoresis to determine the integrity of the RNA. Recently performed qRT PCR and the data were analyzed statistically.
Results: The expression of Bax, Bcl-2 and VDAC1 on prostate cancer cell line (PC-3) were treated with zinc decreased than the control (untreated). However, a decrease in the expression of no significant value because the value of $p > 0.05$ (Bax significant value = 0.309; the value of the significance of Bcl-2 = 0.236; VDAC1 significant value = 0.437). VDAC1 has a significant correlation ($p < 0.05$) with Bax ($p = 0.01$) than Bcl-2 ($p = 0.118$).
Conclusion: There is a change in the expression of each gene (Bax, Bcl-2 and VDAC1) in prostate cancer cell lines that treated with zinc than untreated, but no significant value. VDAC1 has a significant correlation with Bax and had no significant correlation with Bcl-2.;Background: Prostate cancer is the most common cancer in men. Cancer occurs due to loss control of cell proliferation and apoptosis thus continuously proliferating cells without cell death. Apoptosis is regulated by specific proteins including Bcl-2 family proteins and channel proteins. The development of prostate cancer requires the transformation of normal epithelial cells into malignant cells that lose the ability to accumulate zinc. One of the main effects of zinc is to prevent the growth of prostate cancer cells by inducing apoptosis by facilitating the process of pore formation Bax that started apoptogenesis mitochondrial. In addition to Bcl-2 family, VDAC1 also plays an important role in the process of apoptosis. Some studies suggest Bcl-2 has close links with related VDAC1 apoptosis and pro-apoptotic protein Bax also directly interact with VDAC which then induces the release of cytochrome c from the mitochondrial membrane.
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