Androgen receptor levels during progression of prostate cancer in the transgenic adenocarcinoma of mouse prostate model

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Abstrak

Tujuan Untuk menyusun konstruksi tissue microarrays (TMAs) dan mengevaluasi level reseptor androgen pada perkembangan kanker prostat dengan pemeriksaan imunohistokimia pada jaringan prostat tikus model the transgenic adenocarcinoma of mouse prostate (TRAMP) dan tikus non transgenik. Metode Konstruksi tissue microarrays (TMAs) dilakukan terhadap sampel yang berasal dari lobus dorso-lateral tikus kelompok kontrol (non-transgenic), kelompok tanpa kastrasi (intact TRAMP) dan kelompok kastrasi (castrated TRAMP) yang di pulas dengan haematoxylin eosin (H&E). Ekspresi reseptor androgen dievaluasi pada sampel TMAs dengan video image anlysis (VIA). Hasil Ekspresi reseptor androgen dijumpai pada jaringan prostat normal maupun patologis baik pada lesi non-neoplastik maupun neoplastik sampai lesi ganas, sedangkan pada kanker prostat stadium lanjut ekspresi menurun atau menghilang. Ekspresi reseptor androgen meningkat pada kelompok kastrasi (kondisi pelucutan androgen) dibanding pada kelompok tanpa kastrasi. Kesimpulan Sama seperti pada manusia, pada tikus TRAMP kanker prostat menunjukkan variasi ekspresi AR sampai kondisi castrate resistant, yang menunjukkan bahwa AR turut memfasilitasi pertumbuhan tumor lebih lanjut.
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Abstract
Aim To construct tissue microarrays (TMAs) that consisted of prostate tumours from the transgenic adenocarcinoma of mouse prostate (TRAMP) mice and non-transgenic murine prostates and to assess androgen receptor (AR) levels during progression of prostate cancer in TRAMP mice by immunohistochemistry. Methods Haematoxylin and eosin (H&E) sections from the ventral and dorso-lateral prostate lobes of non-transgenic, intact TRAMP and castrated TRAMP were used to demarcate regions of interest for TMAs construction. The samples on TMAs were used to evaluate AR expression using video image analysis (VIA). Results AR was expressed during cancer progression, but AR levels were reduced or absent in late stage disease. Furthermore, when AR levels were compared in tumours from intact and castrate animals, a significant increase in AR levels was observed following androgen ablation. Conclusion Similar to clinical prostate cancer, in the TRAMP model, prostate tumours evolve mechanisms to maintain AR expression and AR responsive gene pathways following castration to facilitate continued tumour growth.