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人胶质瘤细胞U251逃逸NK细胞免疫杀伤机制的初步探讨

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Aberrant Expression of NKG2D Ligand and High Expression HLA- I Molecules May Contribute to Immune Escape from Natural Killer Cell-mediated Immune Surveillance of Malignant Gliomas

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- 摘要
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全文: PDF (442 KB) HTML (0 KB) 输出: BibTeX | EndNote (RIS) 背景资料

摘要 目的 探讨人神经胶质瘤细胞U251逃逸同种异体NK细胞免疫杀伤的机制。方法以K562细胞为对照,应用LDH释放法检测不同靶比时NK细胞体外杀伤U251细胞的活性。用RT-PCR检测K562和U251细胞MHC-I类链相关分子A和B(MICA/B)、人巨细胞病毒糖蛋白UL16结合蛋白(ULBP1~3)基因,用流式细胞仪检测两细胞MICA/B、ULBP1~3和HLA-I类分子的表达情况。效靶比20:1时用单抗分别阻断K562和U251细胞表面MICA、MICB、ULBP1、ULBP2、ULBP3和HLA-I类分子,观察NK细胞对其杀伤活性的变化。结果同一靶比时NK细胞杀伤U251细胞的活性明显低于杀伤K562细胞的活性,两者之间差异有统计学意义(P<0.05);K562和U251细胞均表达基因MICA/B和ULBP1~3,但U251细胞仅低表达ULBP2分子。用单抗封闭MICA/B和ULBP1~3分子后,NK细胞对K562细胞的杀伤活性明显降低,对U251细胞的杀伤活性无明显改变。封闭HLA-I类分子后NK细胞对U251细胞的杀伤活性明显上升,对K562细胞的杀伤活性无明显改变。结论U251细胞逃逸NK细胞免疫杀伤机制可能是由于U251细胞高表达HLA-I类分子,不表达NKG2D的配体MICA/B和ULBP1~3。

关键词: 神经胶质瘤 自然杀伤细胞 自然杀伤细胞受体 细胞毒性试验 HLA-I分子

Abstract: Objective To explore the mechanism of immune escape from natural killer(NK)cell-mediated immune surveillance of malignant gliomas.Methods We took K562 cells cytotoxicity sensitively to NK cell as positive control,cytotoxicities of NK cells isolated from 5 healthy volunteers against U251 cells were analyzed by LDH releasing assay at different effector-to-target cell ratios(E:T).The genes and proteins expression of NKG2D ligands on K562 and U251 cell line were respectively measured by RT-PCR and flow cytometry.In blocking experiment s , mAbs of different N KG2D ligands and HLA2 I molecules were added to the target cells at E:T of 20:1. Results Cytotoxicity of N K cells against K562 cells was much higher than that against U251 cells at the same ET ratio. There was a significant difference between them. The genes of N KG2D ligands were positive in K562 and U251 cells , All the proteins of N KG2D ligands were expressed on K562 cell surface , but noly ULBP2 molecule was found on U251 cell surface. In blocking experiment s , the cytotoxicity of N K cells against K562 cell was partially inhibited , that against U251 cell was not influenced when mAbs of different N KG2D ligands were added ; the cytotoxicity of N K cells against U251 cell was dramatically upgraded , that against K562 cell was not influenced when mAb of HLA2 I molecules was added. Conclusion Aberrant expression of N KG2D ligand and high expression HLA2 I molecules may contribute to immune escape from N K cell-mediated immune surveillance of malignant gliomas.

Key words: Gliomas Natural killer cell NK cell receptor Cytotoxicity HLA- I molecules

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