Supposition about Cancer's Cellular Therapy

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Short Communication

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ABSTRACT

Probably no other field suggests such a vast number of hypotheses and theories as the aspect of etiology and pathogenesis of cancer. Scientists have firmly established experimental and epidemiological data on malignant tumors causal genesis, but pathogenesis of this fatal disease remains absolutely vague. Numerous hypotheses and theories of carcinogenesis, even most popular ones, are insufficient to explain various aspects of this fatal disease. Numerous fundamental and very important questions of cancer's pathology so far remain without any clear answer. The essence of the tumor process consists of unlimited proliferation of hybrid somatic cells. At the same time tumorous synkaryon probably have the ability for hereditary transmitting properties of intensified and uncontrolled proliferation to the following generation of cells.

INTRODUCTION

In the light of biological achievements, in particular, the discovery of the somatic cells hybridization process, the so-called karyogamic theory of carcinogenesis acquires more real countours [1-3]. According of this theory, cancer cell represent hybrid cell, formed after fusion and karyogamy of 2 normal somatic cells. In some cases, spontaneously or after influence of different carcinogenic agents, two normal cells of the same or different types and degree of maturity can be fused with each other (such a cell is refferred as dikaryon). After the fusion of their nuclei (karyogamy), they create hybrid (precancerous) cell with a tetraploid set of chromosomes. Infrequently, as a result of circumstances at subcellular and molecular levels (reciprocal and non-balanced translocations, duplications, deletions of chromosomes, genes amplification) can trans-formed into true cancer cell.

Influence of physical, chemical and biological carcinogenic agents and factors on cells are adequate [4]. In all probability, during the perforation of the cellular membranes, i.e., after the formation of pores, induced by different carcinogenic (and noncarcinogenic) agents and factors, the total negative charge of plasma membranes decreases and the cells develop the ability to come closer to each other, which frequently, especially upon coincidence of the perforated parts of this organoid, will probably be the prerequisite to a fusion process. After karyogamy is possible structural and quantitative unstability of chromosomes KA [5].

After fusogeny, together with dikaryons arise giant polynuclear cells so called polykaryocytes or symplasts. Polykaryocytes formed after endomitosis are functionally active. Unlike from these cells, polykaryocytes formed after fusogeny in most cases, are nonviable cellular formations, i.e., in the genetic respect, they probably are defective peculiar forms, with the lost abilities to enter in S-period of cellular cycle and mitosis and die quickly.

In the case of mitosis without cytotomy (endomitosis), solely viable homokaryons are formed, but in the second variant, i.e., in fusion process of diploid cells homo, as heterokaryons are formed. Exactly these cells (dikaryons), may be potentially tumorigenic, i.e., they may represent oncological danger.

Giant polykaryocytes, must be interpreted solely as a reactive process, carried out in all tissues and organs of macro organism of pathologic states. Thus, it is necessary to take into consideration that the appearance in certain tissues and organs, of the cells of such morphology can signify presence of conditions for cells' hybridization and consequently, potential possibility of appearance of cancer cells.

We have proposed a parallelism between the carcinogenic and fusogenic properties of different substances and influences [6]. An interesting relationship can be observed: the higher the carcinogenic qualities of agents or factors, the lower their fusogenic activity; and the stronger the fusogenic properties, the lesser the carcinogenic properties. The possession of strong carcinogenic qualities by weak fusogens can be explained by the fact that they are able to generate a large quantity of the dikarions with high oncological potency. As for the weakly expressed carcinogenicity of PEG, it may be explained by the ability of this substrate to create in great quantity giant unviable polykaryocytes.

HYPOTHESIS DEVELOPMENT

The non-viability of polykaryocytes created through fusion process (but not through endomitosis) is of special interest. They represent some kind of genetic deadlock and die quickly. As a consequence of nonviability of polykaryocytes, formed by mean of fusion process, our supposition is that it would be necessary to produce simultaneous transformation of tumor cellular substrate (tumor hybrid synkaryons) to the stage of nonviable polykaryocytes. If this could be achieved, there might be a dissociation of tumor substrate and considerable reduction of the mass of the tumor or even its full resolution. Moreover, as already stated, tumorous cells are rather sensitive to the influence of fusogenic agents and factors, in comparison to the normal ones, i.e., they are more easily transformed into the stage of giant polykaryocytes [7]. Some tumorous cell lines are so fusogenic, that they fused spontaneously more efficiently than in the presence of fusogenic agents, for example, PEG A.

How might the transformation of tumorous cells into the stage of polykaryocytes be achieved? It would be ideal to have such chemical substance or biological agent (in example, virus) that would be capable of tropism towards a particular tissue ot organs together with strong fusogenic properties. Carcinogenic substances, tropism of what towards either tissue or organ has been in some cases established, usually express the fusogenic properties rather weakly. For this aim it is possible to use PEG, lysolecithin, polyarginin, glycerol monooleate, electric fields, virus Sendai or other viruses with fusogenic abilities and so on.

For demonstration of above-mentioned, we adduce example of using of PEG in lysis of tumor substrate. One possibility is to use PEG 40-50% solution together with 15% of dimethylsulfoxide (DMSO) approximately 0.01 mg on 1 gram of tumor mass. This substance (DMSO) induces considerable increase of permeability of plasma membranes. This mixture must be introduced into the organ, affected by the tumor (preferable into the tumor focus), by means of radiographic (echoscopy) control. Creation of massive destruction should begin, i.e., dissociation and resolution. If possible chemical substances and biological agents, that have improved organotropic and fusogenic properties simultaneously, should be identified. This would avoid the need for radiographic control and make the whole process much more simple.

We have to admit, that our idea of cancer's treatment, is much more effective on compact (nodular) cancer, than on disseminate (diffuse) cases of this fatal disease. Thus, a possible way to treat cancer is to convert cancer cells to the stage of unviable polykaryocytes, resulting in dissociation of tumor substrate and diminution of the whole mass of tumor and even leading to its resolution.

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