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VIRUS IN GYNAECOLOGICAL ONCOLOGY

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Summary: We measured the antibody presence of eventual viral infections (Parotiditis, Cytomegalovirus, Herpes Simplex and Hepatitis B) in 51 patients affected by malignant gynaecological tumours.

Ninety five women composing the control group, not affected by any pathology, underwent the same tests.

Even if research has utilized various indirect methods for the antibody dosage of each viral infection, we have not confirmed other Authors' reports. It might be referable to the limited number of cases in our study up to date. In order to make a comparison among different reports we look forward to a uniformity of various laboratory methods.

Reliable results will be obtained only by increasing the number of clinical cases and by utilizing the method of determination of viral genome in tumoral cells. The technique is the only reliable among different methods for determining a previous viral infection.

INTRODUCTION

In humans, as in animals, a sort of correlation and an eventual etiopathogenetic link between virus and gynaecological tumours has been ascertained: the theory of oncogenesis, in fact, recognizes a close interaction between the genome of the guest cell and viral DNA ([1, 2, 3].

We have tried to recognize a significant antibody trace of previous viral infections in women affected by malignant gynaecological tumours, and made a comparison with a control group of healthy woment who underwent the same laboratory tests.

This preliminary report, in order to be reliable, must be correlated with other investigations on the genetic situation of these patients: genomic analysis, distribution in groups of histocompatibility, and so on, not considered in the present study.

PATIENTS AND METHODS

Fifty one women were selected for this investigation, being affected by a histologically ascertained genital tumour and in particular cervical carcinoma, endometrial carcinom and ovarian carcinoma.

Each group of oncologic patients was compared with a control group of 95 healthy women, so distributed for a reliable comparison: from 45 to 55 years old for the control of cervical carcinoma; from 55 to 65 years old for the control of endometrial carcinomas; and from 35 to 45 years old in order to compare the ovarian carcinoma.

In each woman, both patients and controls, we searched for the eventual presence of serum parotiditis virus antibodies, cytomegalovirus an-

Table 1

Oncological patients				Control groups			
			Cervix				
No.		+	−	No.		+	−
	Parotiditis	80.8%	19.2%		Parotiditis	65.4%	34.6%
	Hepatitis B	0	100 %		Hepatitis B	0	100%
18	HSV-2	73 %	27 %	34	HSV-2	38 %	62 %
	Cytomegalovirus	65.4%	34.6%		Cytomegalovirus	38 %	62 %
			Endometrium				
	Parotiditis	70%	30%		Parotiditis	40%	60%
	Hepatitis B	0	100%		Hepatitis B	0	100%
18	HSV-2	30%	60%	34	HSV-2	90%	100%
	Cytomegalovirus	40%	60%		Cytomegalovirus	60%	40%
			Ovary				
	Parotiditis	50%	50%		Parotiditis	50%	50%
	Hepatitis B	0	100%		Hepatitis B	0	50%
15	HSV-2	50%	50%	27	HSV-2	25%	75%
	Cytomegalovirus	25%	75%		Cytomegalovirus	25%	75%

tibodies, herpes simplex virus 2 (HSV-2) antibodies and hepatitis B surface antibodies.

By means of these dosages we tried to ascertain if both oncologic patients and healthy women had been infected or at least had come into contact with such viruses.

Different laboratory methods were employed to obtain the antibody dosages for each viral group and in particular:

- neutralizing test for the titer of parotiditis virus antibodies;
- fixation of complement reaction to titrate cytomegalovirus antibodies and HSV-2 antibodies;
- immunoenzymatic method for the titer of hepatitis B surface antibodies.

RESULTS

In 1985, in a period of twelve months, 51 oncologic patients were selected and so distributed:

— 18 women affected by cervical carcinoma;

— 18 women affected by endometrial carcinoma;

— 15 women affected by ovarian carcinoma.

At the same time, 95 women not affected by any tumoral pathology were so distributed:

— 34 for the comparison vs. cervical carcinoma;

— 34 for the comparison vs. endometrial carcinoma;

— 27 for the comparison vs. ovarian carcinoma.

The control women of the first group ranged from 45 to 55 ears old; the second group collected healthy women from 55 to 65 years old, whereas the third control group was composed by women from 35 to 45 years old.

This distribution was made on the basis of literature reports which recognize these age ranges as the most frequent for each oncological disease considered in this study.

If we consider for a viral contact, antibody titers from 1:4 to 1:64, we can thus resume the data of table 1.

a) Cervical carcinoma group and its correlative control group: the percentage of antibody titers are equivalent if we consider parotoditis virus, hepatitis B virus and cytomegalovirus. With regard to herpes simplex 2 virus, we observed a slight (not significant percentage) increase of an-

tibody titers in the oncological patients' compared with correlative control women.

b) Endometrial carcinoma group and its correlative control group: a similarity of antibody titers could be observed in both groups as regards parotiditis virus, hepatitis B virus and cytomegalovirus. On the other hand, if we consider HSV-2 antibody presence, an increase of positive cases (90%) coul be noted in the control group, compared with 30% in the oncologic group.

c) Ovarian carcinoma group and its correlative control group: no difference could be noticed in the percentage of antibody presence in the former group versus the latter one.

DISCUSSION

Based on the actual theory of oncogenous, our preliminary data (to this day incomplete as regard the analysis of viral genome, under study at the present time) aim at confirming eventual phenomena of mono and/or multiviral primer on a previous genomic predisposition and with the concourse of chemical and physical factors ([4, 5,6]).

Research on different virus antibodies is suggested by recent reports of virology and molecular biology considering carcinogenesis as a multiple stage process; it is certain, in fact, that a single event (mutation, genic re-arrangement) operating over an oncogene is not sufficient to promote cancer ([7, 8]).

The concurrence of at least two oncogenes is necessary to turn a normal cell into a cencerous one: an oncogene immortalizes, another one transforms the cell.

The first "tatoo" may be caused by some virus without any relation to oncogenes or by chemical carcinogens.

Our results do not allow us to confirm other Authors' reports about the correlation between virus and gynaecological tumours. We agree with the results of Vonka and coll. regarding the correlation between HSV-2 and cervical carcinoma ([9]). In fact, the percentage of antibodies is almost the same in the control group and in the oncologic one as regards cytomegalovirus, hepatitis B virus and paritiditis virus; whereas HSV-2 antibodies are lightly increased in the cervical carcinoma group.

These data do not allow us to draw infallible conclusions either for the different laboratory techniques used in titering antibodies and having a variable sensibility, or for the composition of the control group, which can strongly influence the results in relation to various parameters, and in particular sexual behaviour of women and their partners ([10, 11]).

Our control group was composed of healthy women with a normal sexual behaviour, and come to our attention for a preventive gynaecological checking, and were selected according their age for the comparison with tumoral groups.

We hold that until laboratory techniques, choice of control groups, and above all simple methods for the research of viral genomes in tumoral cells are standardized, reliable and homogeneous results will be not possible.

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