CELL-MEDIATED IMMUNITY AND CONDYLOMATOSIS

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Summary: Cell-mediated response in 60 patients affected by acuminated condylomas has been studied; this was investigated with aspecific in vivo tests (intradermal-reaction with tuberculin, trichophytin, candidine) and by in vitro tests (blood test with cell count, lymphocyte typing, serum proteins, serum immunoglobulin) and was compared with a control group.

The results obtained confirm the hypothesis that condylomas acuminata usually occurs in patients with well-preserved cell-mediated immunity.

Key words: condylomas acuminata; sexually transmitted diseases; cell-mediated immunity; lymphocyte typing.

It has been known for some time that in Human Papillomavirus (HPV) infection, the B-cell immunity does not seem to intervene in defence mechanisms; however, according to recent studies, cell-mediated immunity always plays an important role in preventing viral infections ([1, 2, 3, 4, 5, 6]).

In fact, in spite of the numerous works which implicated an increase in the frequency of HPV diseases during immunodeficiency, it was nevertheless demonstrated that only in patients with depression of T-lymphocytes, has it been possible to document this increase. The frequency of these patients is, on the other hand, comparable to that of control groups with humoral immunodeficiency ([7, 8, 9, 10, 11, 12]).

Recently, in subjects affected by multiple verrucae, a statistically significant reduction in T-lymphocytes both in percentage and numerical count, and a significant increase in the non T non B cells was shown ([13]).

In view of the above, this work aims to evaluate the cell-mediated immunity in patients affected with condylomatosis.

MATERIAL AND METHODS

We studied 60 patients affected by condylomatosis, subdivided into two group of 30 patients: *Group A*, with 18 females (average age 30 years, range 18-37) and 12 males (average age 31, range 17-45) affected by recurrent codylomas acuminata for at least 6 months, resistant to all conventional local treatments; *Group B*, consisted of 16 females (average age 28 years, range 16-35) and 14 males (average age 33, range 18-47) also affected by condylomas acuminata, but in whom the infective episode represented the first infective manifestation which, after local therapy and a 6 months follow-up, did not present relapses.

In the two groups studied, we did not take into consideration those patients who had previously been submitted to immunomodulators or had presented other pathologies referable to a deficient of cell-mediated immunity. The localizations of the condylomatous manifestation in the two groups are reported in Table 1.

In conclusion, we also evaluated a third control group, *Group C*, composed of 30 healthy subjects, 15 females (average age 29 years, range 16-36) and 15 males (average age 31 years, range 18-41).

In order to study cell-mediated immunity, in vivo and in vitro tests were carried out on all subjects in the 3 groups:

1) *In vivo tests:*

Intradermal reactions:

- Tuberculin: purified protein derivative (PPD) 1:1000 (Sclavo, Siena; Italy).
- Candidine: Dermatophitin "0" diluted 1:1000 (Hollister Stier, Berkeley; USA).
- Trichophytin: Dermatophitin diluted 1:30 (Hollister Stier, Berkeley; USA).

Table 1. – *Localisation of the condylomatous lesions.*

	Group A	Group B
In Female		
Cervix	3	4
Vagina	–	1
Vulva	4	4
Perineum	2	3
Cervix + Vagina	2	1
Cervix + Vulva	4	2
Vulva + Perineum	3	1
In Male		
Glands	1	2
Prepuce	2	3
Shaft	–	1
Perineum	3	2
Glands + Prepuce	3	5
Glands + Prepuce + Shaft	1	–
Gland + Prepuce + Perineum	2	1
Total	30	30

2) *In vitro tests:*
- Blood with leucocytes count
- Protein profile
- Serum immunoglobulins
- Total T-lymphocytes
- T4 lymphocytes
- T8 lymphocytes.

Lymphocyte typing was carried out by direct immunofluorescence. The monoclonal antibodies (T3 - T4 - T8) used as detectors, were marked with fluorescein isothiocyanate (FITC) or tetramethylrhodamine isothiocyanate.

RESULTS

Aspecific in vivo tests: the number of negative responses for tubercolin were sligthly higher compared with the controls in the subjects affected by condylomatoses.

For the other allergens (candidine and trichophytin) the controls gave negative responses higher than or equal to those

Table 2. – *Response to aspecific in vivo tests (intradermal-reaction in group A.*

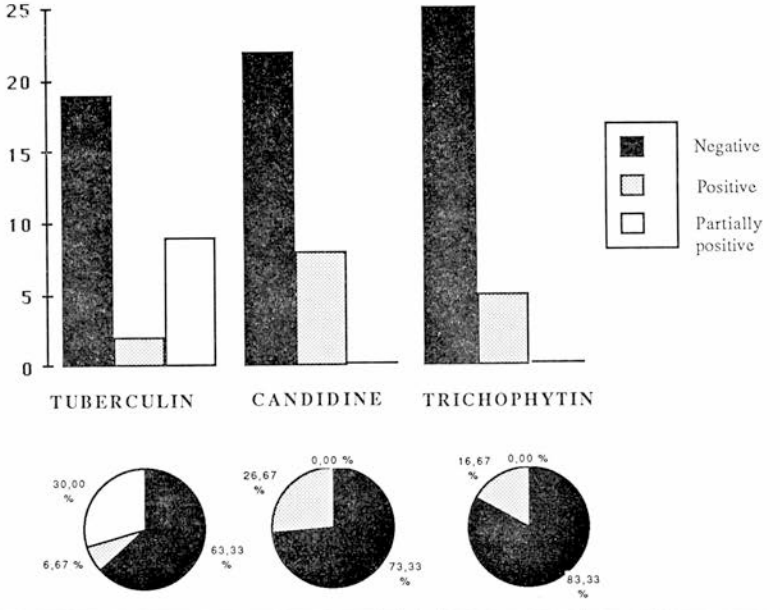


Table 3. – *Response to aspecific in vivo tests (intradermal-reaction) in group B.*

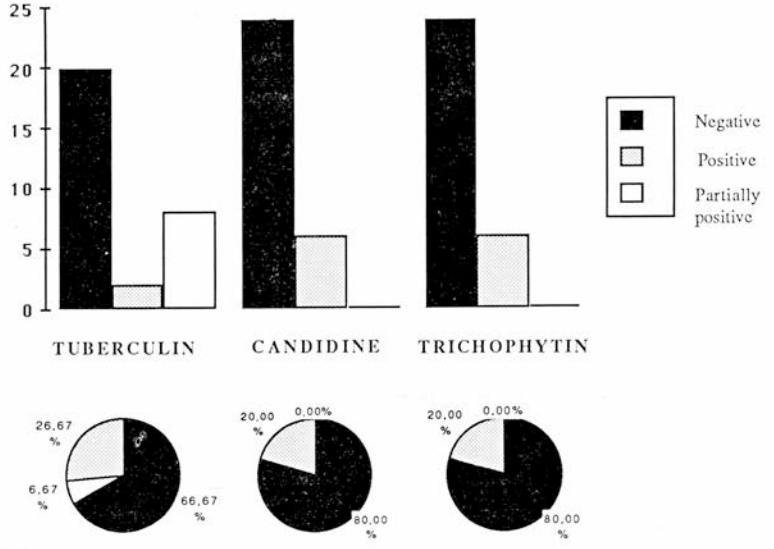


Table 4. – *Response to aspecific in vivo tests (intradermal-reactions) in group C.*

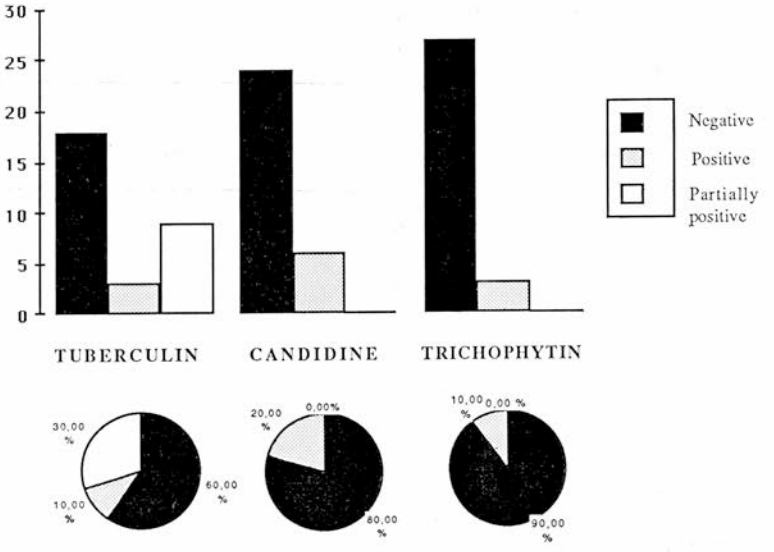
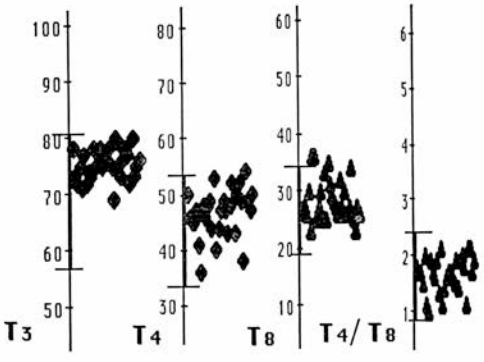


Table 5. – *Distribution of T lymphocytes in group A.*



of the patients affected with HPV infection (Table 2, 3, 4).

In vitro tests, on the other hand, gave more uniform responses, since no substantial statistically significant differences were shown in the levels of T-lymphocytes (Table 5, 6, 7).

Blood tests with relative leucocyte count, serum proteins and immunoglobulins never showed any major pathological variations in any of the three groups studied.

In particular, no significant differences were shown in the values considered in the two groups affected by condylomatoses (Table 8, 9, 10).

DISCUSSION AND CONCLUSIONS

Condylomas represent a frequent dermato-gynecologic lesion. They are often difficult to treat on account of the high incidence of relapses. In recent years, both the infection and the relapses have been considered in relation to a deficit of the immune system and in particular of the cell-mediated response ([14, 15, 16]).

Thus a numerical and/or functional deficit of these cells involved in these responses could explain or at least favor the occurrence of this lesion ([2, 17, 18]).

With regard to the results we obtained, it seems to us that because of their homogeneity, they generally exclude a T dependent immunodeficiency in patients with condylomas acuminata.

However, it should be pointed out that in this study the immune response was only partially evaluated, excluding both those aspects that might play an important pathogenetic role (study of macrophage activity, production and activity of the lymphokines, macrophage chemotaxis and killer cells), and those that by limiting the in vitro tests to blood analysis, the study

Table 6. – *Distribution of T lymphocytes in group B.*

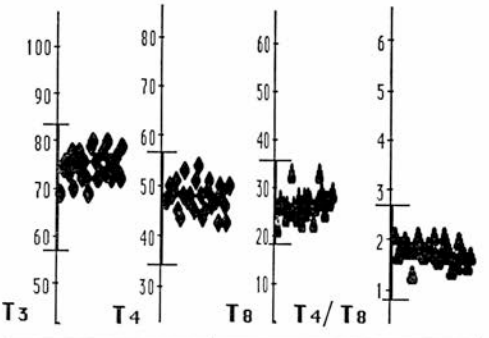


Table 7. – *Distribution of T lymphocytes in group C.*

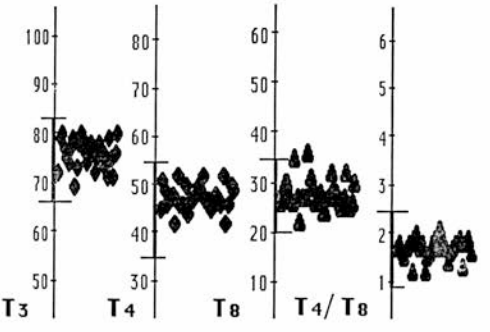


Table 8. – *Distribution of the concentrations of immunoglobulins (IgG, IgA, IgM) in group A.*

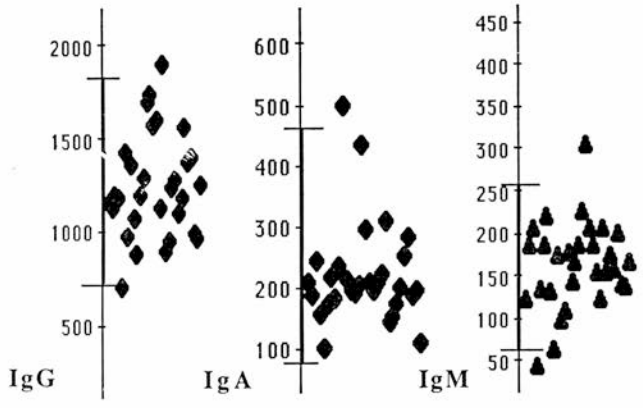


Table 9. – *Distribution of the concentrations of immunoglobulins (IgG, IgA, IgM) in group B.*

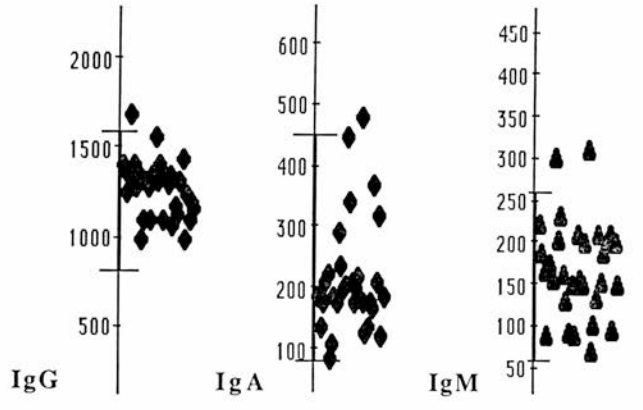
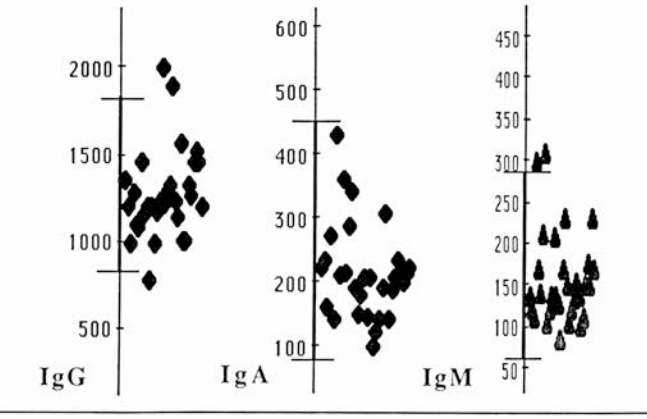


Table 10. – *Distribuithion of the concentrations of immunoglobulins (IgG, IgA, IgM) in group C.*



of the immunitary phenomena in the tissutal site was neglected on account of objective difficulty and lack of knowledge about procedures.

Our data, even with the limitations referred to above, seem to convalidate the hypothesis that factors of local reactivity and sexual habits greatly influence the onset of papovavirus infection.

Immunodeficiency would, therefore, represent only one of the possible secondary co-factors. In fact, in particularly evident cases with abundant growth of condilomas they are accompanied by a normal immunitary picture. It is also a fact that, while in immunodepression a complication of viral character is pathogenetically predictable, viruses do not generally need an immunitary deficit in order to manifest themselves. Since they depend on many endogenous and exogenous factors which intervene to modify the host/virus equilibrium in favor of the latter.

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