Gynecological screening for HPV infection

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Summary

HPV infection is the most common sexually transmitted disease today and is strongly related to cervical cancer. We studied 1,500 women in a limited area of the Calabria region to determine the best method of screening for cevical cancer.

Introduction

Cervical cancer is the second gynecological neoplasia in women. The worldwide incidence is nearly 500,000 women/year and mortality is estimated to be about 170,000 women per year [1, 2]. Distribution of the disease is related to economic and social status [1, 3] and about 80% of cervical carcinoma cases are diagnosed in developing countries [4]. In the last 50 years the incidence of this neoplasia has declined in developed countries due to an increase in screening programs [4].

Human papillomavirus (HPV) infection, particularly HPV 16/18, is strongly related to the development of cervical cancer [5, 6]. In 99% of the cases HPV DNA is present [5, 7, 8].

HPV infection, whether clinical or latent, is the most common sexually transmitted disease today [9] and the number of asymptomatic women affected is reported to be about 5-20% [10].

The risk of developing cervical cancer is higher in women with persistent, long-term, high-risk HPV infection [6].

Materials and Methods

In April 2002, a cervical cancer screening program was initiated in a limited area of the Calabria region of Italy which included 1,500 women. Of these, 1,185 women (group 1) underwent only Pap smears at public clinics and the other 315 women (group 2) were submitted to complete screening which included gynecological examination, vulvoscopy, colposcopy, cytology, vaginal flora, and vaginal pH in the presence of suspected areas by HPV investigation.

HPV typization was performed with standardized methods with samples from the labia minor and exocervix. In case of both low- and high-risk results in the same sample, only high risk was considered.

All HPV-positive patients (group 2) were treated by topical antiviral therapy and diathermal coagulation of the lesion. Only in one case was conization performed.

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Results

HPV positivity was 30.7% in the first group and 24.1% in the second with overall HPV positivity reaching 29.5% (Table 1) confirming the well-known relation between HPV and cervical cancer. The higher positivity of the first group may be attributed to the fact that a higher percentage of immigrants with a lower social and economic status undergo public screening. In contrast, the second group of women underwent testing in private clinics, always with the same medical team. Risk distribution for the two groups is shown in Table 2.

The outcome after treatment of the second group is reported in Table 3. Since the first group of patients were screened in public clinics, follow-up data was not available.

In conclusion, no statistically significant differences were found between the two groups even though the best results overall were in the women who underwent complete screening. This suggests that from a cost/benefit point of view regular Pap screening in either a private or public facility is a sufficient and beneficial screening method for cervical cancer.

Table 1.

	Only Pap smear	Complete screening	Total
Number of patients	1,185	315	1,500
HPV-positive patients	364	76	440
	(30.7%)	(24.1%)	(29.3%)

Table 2. — *Risk distribution.*

High-risk genotype	Group 1	Group 2	Low-risk genotype	Group 1	Group 2
66	24	8	6	40	16
16	76	12	44	8	1
18	33	9	53	19	1
31	33	7	74	8	3
45	8	1	70	7	2
52	57	10	42	7	1
56	11	1	54	8	2
58	16	1	11	9	1
	258	49		106	27
	(70.8%)	(64.4%)		(29.1%)	(35.5%)

Table 3. — *Outcome after treatment (6 months).*

	No.	%
Disease free	47	61.8%
Persistence	25	32.9%
Lost at follow-up	3	3.9%
	76	98.6%

References

[1] Ferlay J., Bray F., Pisani P., Parkin D.M., Globocan 2002: "Cancer incidence, mortality and prevalence worldwide". IARC CancerBase No. 5, Version 2.0. Lyon, IARC Press, 2004.

[2] Koutsky L.: "Epidemiology of genital human papillomavirus infection". *Am. J. Med.,* 1997, *102*, 3.

[3] Parkin M., Bray F., Ferlay J., Pisani P.: "Global Cancer Statistics, 2000". *CA Cancer J. Clin.,* 2005, *55,* 74.

[4] Koushik A., Franco L.F.: "Epidemiology and the role of human papillomavirus". The Cervix, 2nd edition, 2006, 259.

[5] Walboomers J.M., Jacobs M.V., Vianos M.M., Bosch F.X., Kummer J.A., Shah K.V. *et al.*: "Human papillomavirus is a necessary cause of invasive cervical cancer worldwide". *J. Pathol.,* 1999, *189*, 12.

[6] Muñoz N., Bosch F.X., Castellsangue X., Herrero R., Castellsangue X., Shah K.V. *et al.*: "Epidemiologic classification of human papillomavirus types associated with cervical cancer". *N. Engl. J. Med.,* 2003, *384*, 518.

[7] Collins Y., Einstein M.H., Gostout B., Herzog T.J., Massad L.S., Rader J.S. *et al.*: "Cervical cancer prevention in the era of prophylactic vaccines: A preview for gynecologic oncologists". *Gynecol. Oncol.,* 2006, *102*, 552.

[8] Bosch F.X., Lorincz A., Munoz N., Meijer C.J.L.M., Shah K.V. *et al.*: "The causal relation between human papillomavirus and cervical cancer". *J. Clin. Path.,* 2002, *55*, 885.

[9] Cox J.T.: "Epidemiology of cervical intraepithelial neoplasia: the role of human papillomavirus". *Baillieres Clin. Obstet. Gynaecol.,* 1995, *9,* 1.

[10] Bosch F.X., Lorincz A., Muñoz N., Meijer C.J.L.M., Shah K.V.: "The causal relation between human papillomavirus and cervical cancer". *J. Clin. Pathol.,* 2002, *55,* 244.

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