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Research Article

Use of Pidotimod as an adjuvant treatment in women with human papillomavirus infection: a retrospective, observational, multicenter study in a Mexican popu lation

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Abstract

Background: Genital warts (condylomata acuminata) associated with Human Papillomavirus (HPV) exhibit a high recurrence rate after conventional ablative treatments due to viral persistence in perilesional epithelium. Systemic immunomodulation represents a complementary approach of great clinical.

Objective: To describe the clinical evolution of Mexican women with HPV-associated genital warts treated with ADIMOD® (Pidotimod) as monotherapy or combination therapy in private practice.

Materials and Methods: A retrospective, observational, longitudinal, descriptive, and multicenter study was conducted (period 2007-2025). We included 149 immunocompetent women (18-60 years old) with a confirmed HPV diagnosis via visible genital warts, cytology showing LSIL/CIN I, and/or biopsy/PCR, treated with Pidotimod (800 mg/day for 60 or 90 days) and a minimum 12-month follow-up. Statistical analysis was performed using SPSS 26.0 with frequencies, percentages, non-parametric tests, and Kaplan-Meier remission.

Results: The predominant age group was 25-34 years old (35%). Pidotimod was administered as monotherapy in 69% of patients and as combination therapy in 31.5%. The short-term milestone demonstrated that 79.2% (n=118) achieved complete clinical remission before 6 months, increasing to 86.5% (n=129) by the seventh month. Kaplan-Meier curves revealed a significantly faster remission kinetics in patients aged 18-34 years (>90% at 6 months) compared to the 45-54 age group (40%). During the 12-month follow-up, 96% (n=143) of the patients remained recurrence-free.

Conclusion: Pidotimod proved to be an effective and safe therapy, achieving a high rate of short-term clinical remission and a drastic reduction in long-term recurrence rates among immunocompetent patients with genital warts.

Keywords: Human Papillomavirus; Condylomata Acuminata; Pidotimod; Immunomodulation; Clinical Remission; Recurrence

Background

Human papillomavirus (HPV) infection is the most common sexually transmitted infection worldwide and represents a significant public health issue due to its high prevalence and associated complications. More than 200 HPV genotypes have been identified, classified into low- and high-oncogenic-risk types. Low-risk genotypes—primarily HPV 6 and 11—are responsible for approximately 90% of cases of genital warts (condylomata acuminata), whereas high-risk genotypes—especially HPV 16 and 18—are closely linked to the development of intraepithelial neoplasia and cervical cancer. Persistent HPV infection remains a challenge for healthcare systems due to its widespread distribution and the clinical consequences resulting from viral persistence.

Genital warts represent one of the most common clinical manifestations of HPV infection in women. These lesions can occur on the vulva, vagina, cervix, perineum, and perianal region, causing significant physical, sexual, and psychological consequences. Although conventional therapeutic modalities—such as cryotherapy, electrocoagulation, laser vaporization, and topical treatments—demonstrate high rates of visible lesio4n clearance, recurrence remains frequent due to viral persistence in infected tissues and the variable immune response of individual patients [1-3].

The cellular immune response plays a fundamental role in controlling and clearing HPV infection. Various studies have shown that states of immu-

nosuppression are associated with increased susceptibility to infection, viral persistence, and lesion recurrence. In this context, modulating the immune response has emerged as a complementary therapeutic strategy to improve clinical outcomes in patients with HPV infection and recurrent genital warts [2,4].

In vivo and in vitro studies show that the immunomodulatory activity of Pidotimod targets both innate and adaptive immunity. Pidotimod induces dendritic cell (DC) maturation, increases the expression of HLA-DR and co-stimulatory molecules, stimulates DCs to release pro-inflammatory molecules that drive T-cell proliferation and differentiation toward a Th1 phenotype, enhances natural killer (NK) cell function, and promotes phagocytosis; these mechanisms of action also regulate the production of cytokines involved in the antiviral response. These properties have sparked interest in its potential utility as an adjuvant treatment for persistent viral diseases, including HPV-associated lesions [4].

Available clinical evidence regarding the use of Pidotimod for HPV-associated genital lesions is limited but encouraging. A randomized, placebo-controlled clinical trial involving women with vulvar papillomatosis showed a higher rate of complete lesion regression in patients treated with Pidotimod compared to those receiving a placebo, suggesting a potential benefit derived from the stimulation of the cellular immune response [5].

Furthermore, subsequent studies have indicated that the efficacy of Pidotimod against HPV-related genital lesions may be comparable to that observed with other immunomodulators, yet with a favorable safety profile [4].

Similarly, a prospective study evaluated the use of Pidotimod combined with vitamin C following laser vaporization in women with genital warts. The results showed a trend toward a higher clinical remission rate and lower lesion recurrence compared to surgical treatment alone, although the differences did not reach statistical significance due to the limited sample size. Nevertheless, these findings suggest that boosting the immune response via immunomodulators could contribute to infection control and the reduction of recurrences [2,4].

Given the high recurrence rate of genital warts and the importance of the immune response in clearing HPV, the evaluation of immunomodulatory therapies such as Pidotimod represents a promising area of research. Its potential to promote lesion regression, decrease viral persistence, and reduce recurrences could serve as a useful complementary strategy within the comprehensive management of women with HPV infection and genital warts.

Objective

To describe the clinical course of Mexican women with genital warts associated with Human Papillomavirus (HPV) who were treated with Pidotimod—either as monotherapy or in combination with other treatments—in specialized private practice settings.

MATERIALS AND METHODS

Study Design: A retrospective, observational, longitudinal, descriptive, and multicenter study conducted via a review of medical records at four private practice centers. These included two centers in Mexico City (one specializing in Gynecology and Obstetrics with a sub-specialty in Gynecologic Oncology and advanced training in Colposcopy, and one general medicine center with training in Colposcopy) and two centers in Toluca, State of Mexico (specializing in Gynecology and Obstetrics with advanced training in Colposcopy). Patients presented voluntarily to these specialized HPV care centers.

The study protocol involved reviewing medical records from 2007 to 2025 (conducted between December 1, 2025, and May 1, 2026) for patients diagnosed with HPV who had been prescribed Pidotimod as part of their treatment—either as monotherapy or in combination, as determined by the specialist—and who met the inclusion criteria.

To assemble the sample using the census method, the following criteria were strictly applied to the clinical records reviewed:

Inclusion Criteria; Gender and Age: Female patients aged 18 to 60 years at the time of diagnosis.

Diagnostic Certainty: Confirmed diagnosis of Human Papillomavirus (HPV) infection via: Presence of clinically visible genital warts located on the vulva, vagina, cervix, perineum, and/or perianal region; histopathological confirmation via colposcopy-guided biopsy and/or genomic identification via Polymerase Chain Reaction (PCR).

Cytological Findings: Concomitant cytology report of low-grade squamous intraepithelial lesion (LSIL / CIN I).

Therapeutic Regimen: Patients who received systemic treatment with Pidotimod, either as monotherapy or in combination with other treatments prescribed in private practice.

Follow-up and Adherence: Clinical record of a minimum 12-month follow-up, documented by at least two follow-up visits spaced 4 to 6 months apart within that period.

Data Quality: Records with complete clinical documentation explicitly including: sociodemographic variables, life-cycle stage (reproductive stage, climacteric, or postmenopause), morphological characteristics of the lesions, temporal progression, and records of prior or concomitant combination therapies.

Exclusion Criteria: Coexisting Immunocompromise: Patients with a history or active diagnosis of conditions that substantially alter the immune response (e.g., HIV infection, chronic use of systemic corticosteroids, immunosuppressive therapies, chemotherapy, or active extra-pelvic malignancies).

Uncontrolled Chronic Comorbidities: Patients with uncontrolled metabolic or organic diseases (e.g., uncontrolled diabetes mellitus with HbA1c > 7%, severe liver or kidney disease) that could interfere with the natural course of the infection or the pharmacokinetics of the immunomodulator.

Concomitant/Overlapping Immunomodulatory Treatment: Having received other systemic immunomodulators or HPV vaccines within the 2 weeks prior to starting Pidotimod or during the course of the evaluated therapeutic cycle.

Pregnancy or Breastfeeding: Patients who are pregnant or actively breastfeeding during treatment, due to physiological immunological and endocrine changes that alter the behavior of condylomata acuminata.

Loss to Follow-up: Patient records for those who did not complete the scheduled 12-month clinical evaluations or who discontinued treatment before completing the prescribed minimum regimen.

Definitions considered

Condyloma acuminatum, also known as genital warts, is a benign lesion caused by Human Papillomavirus (HPV) infection—primarily subtypes 6 and 11—characterized by exophytic lesions (papillomatous or warty growths on the skin or genital mucosa). These lesions are located on the vulva, vagina, cervix, perineum, and perianal region; they may be solitary or multiple, flat or raised, with a rough surface and a pinkish or grayish color. Associated symptoms include itching, burning, mild bleeding, and

discomfort during sexual intercourse.

Clinical remission of genital warts: The complete disappearance of visible lesions in the genital region (condylomata acuminata). Absence of recurrence during 12 months of follow-up, with resolution of associated symptoms such as itching, burning, bleeding, or local pain. Clinical confirmation via physical examination.

Genital wart recurrence: the reappearance of visible wart-like lesions in the genital region after achieving initial clinical remission.

Reappearance of new lesions: warts at the same site or in nearby areas.

New infection: appearance of warts due to acquisition of a different HPV strain.

Persistence: lesions that never completely disappeared after initial treatment.

Sample design: Census of "all available patients meeting inclusion criteria."

Statistical analysis: Performed using the Statistical Package for the Social Sciences (SPSS 26.0) software. Quantitative variables are described using mean and standard deviation or median and interquartile range, depending on their distribution. Categorical variables are expressed as frequencies and percentages.

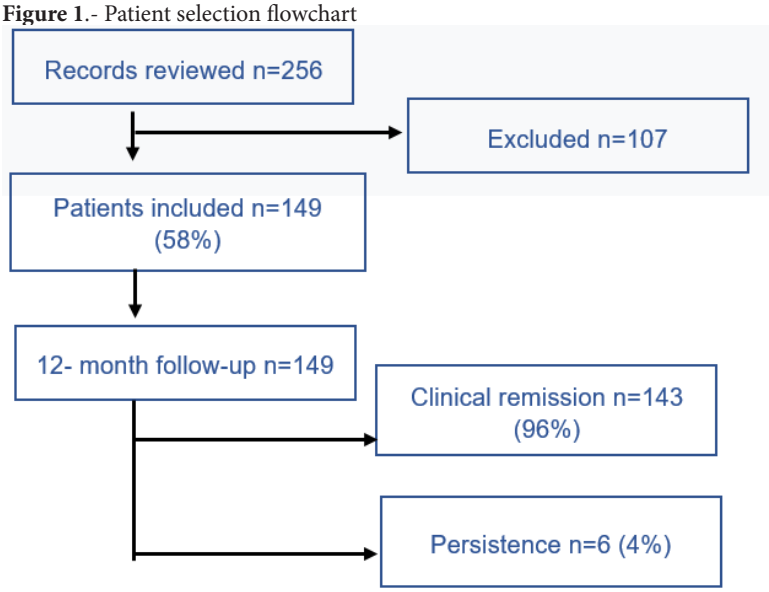
Comparisons between groups were made using the Chi-square test or Fisher's exact test for categorical variables and the Mann-Whitney U test for non-parametric continuous variables. Statistical significance was defined as a p-value <0.05.

RESULTS

To structure the study population by age group, classification was based on the epidemiological behavior of HPV infection across the following age ranges: 18–24, 25–34, 35–44, 45–54, and 55–60 years.

A total of 256 clinical records from 2007 to 2025 were reviewed; all patients presented with visible condylomata. Of these, 149 (58%) met the inclusion criteria and were included in the study analysis. Patients presented voluntarily at private clinics—two in Mexico City and two in Toluca, State of Mexico—staffed by two specialists in obstetrics, gynecology, and colposcopy, one general practitioner trained in colposcopy, and one gynecologic oncologist.

Figure 1.- Patient selection flowchart



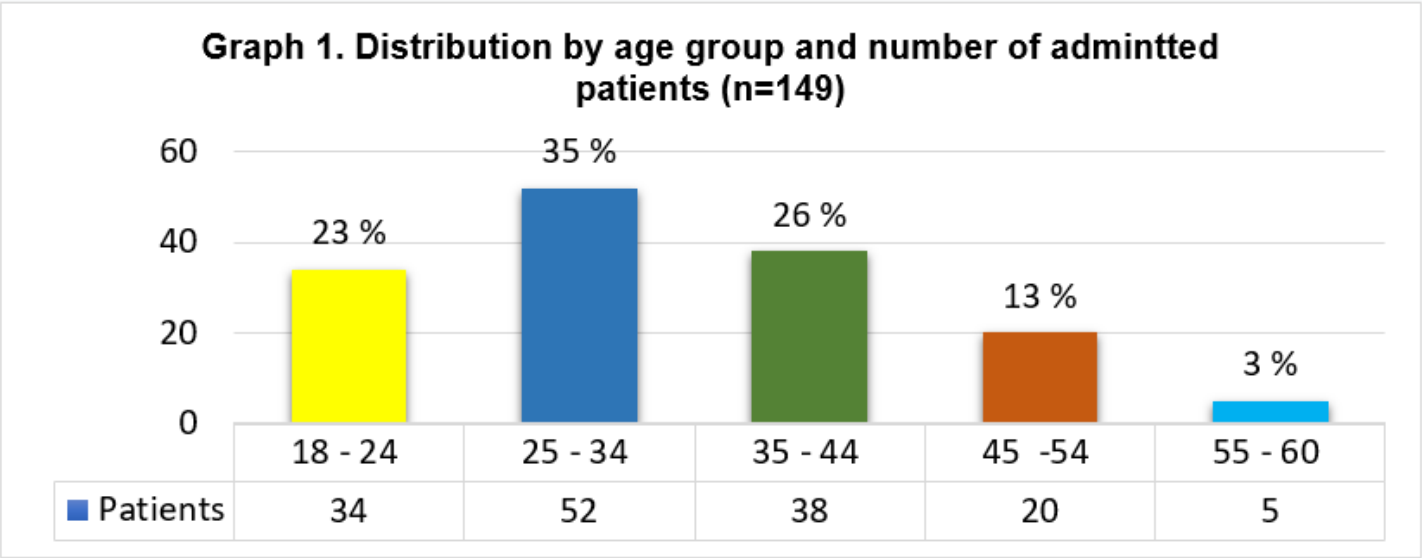
33 (22%) of the patients were treated in 2023, followed by 30 (20%) in 2024 (Table 1).

Table 1. ADMISSION DATES BY YEAR

YEAR	NUMBER OF CASES	PORCENTAGE
2007	1	1
2011	1	1
2012	1	1
2015	1	1
2016	1	1
2018	4	3
2020	13	9
2021	16	11
2022	22	15
2023	33	22
2024	30	20
2025	26	17
total	149	100

Source: Clinical records from participating centers

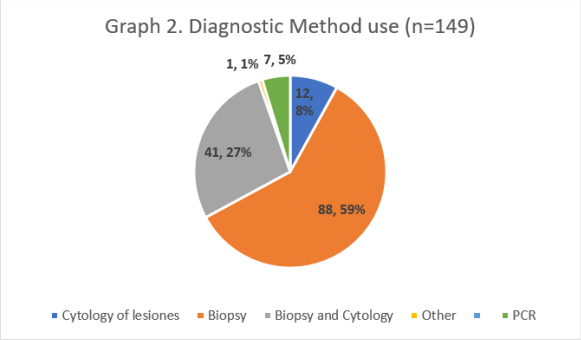
The mean age of the total sample was 33 years (range: 18–60 years); all participants were Mexican women, with 76 (51%) located in Toluca, State of Mexico, and the remainder in Mexico City. The most affected age group consisted of 52 women (35%) aged 25–34 years, followed by 38 (26%) aged 35–45 years (Graph 1).



Source: Clinical records from participating centers

149 (100%) were female and immunocompetent.
11 (7%) had undergone prior treatment for low-grade squamous intraepithelial lesions (LSIL).

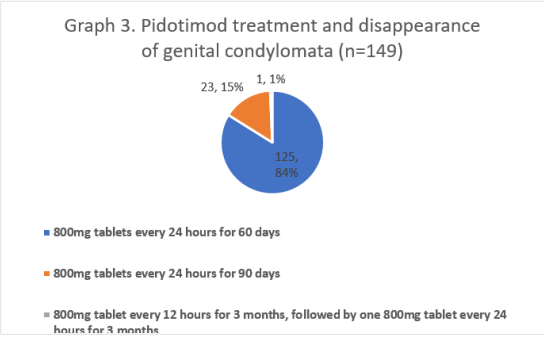
Regarding the HPV diagnostic method, the virus was identified via biopsy in 88 cases (59%) and via biopsy and cytology in 41 cases (27.5%); regarding the identified genotype, 135 (91%) were not typed, while 12 samples (8%) were high-risk (HPV 16, 18, 31) (Graph 2).



Source: Clinical records from participating centers

131 (88%) were of reproductive age—with a mean menstrual cycle phase of 17 days (range: 5–30 days)—while 12 (8%) were postmenopausal and 4 (3%) were in the climacteric period.

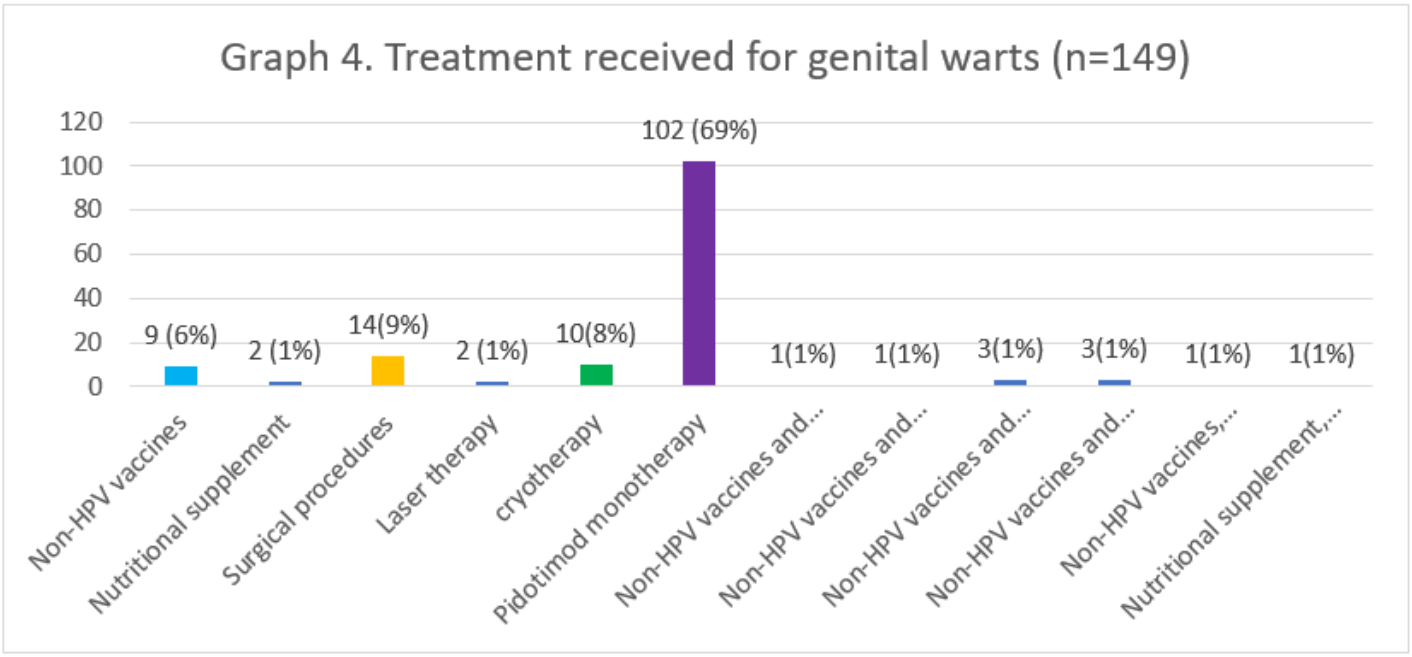
Regarding the treatment administered for genital condylomata, 125 patients (84%) took 800 mg Pidotimod tablets (one tablet every 24 hours for 60 days), followed by 23 patients (15%) prescribed one 800 mg tablet every 24 hours for 90 days, to be taken two hours before or after meals (Graph 3).



Source: Clinical records from participating centers

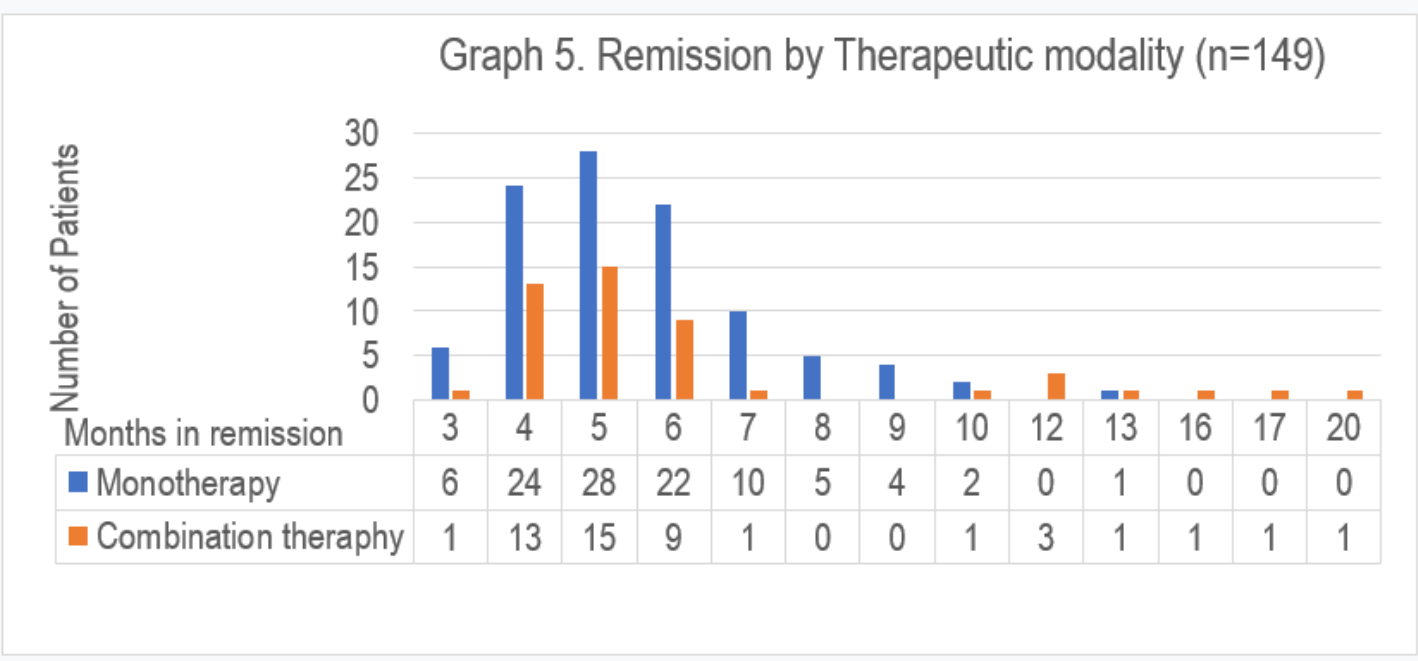
141 (95%) received only one cycle of Pidotimod.

Regarding the treatment received for genital warts, 102 women (69%) underwent monotherapy with Pidotimod, and 47 women (31.5%) received combination therapy with Pidotimod (Graph 4).



Source: Clinical records from participating centers

Regarding the resolution of genital warts—comparing Pidotimod monotherapy versus combination therapy and the rate of disappearance—118 of the 149 women (79%) experienced regression within 6 months, and the warts in 129 of the 149 women had disappeared by the seventh month (Graph 5).



Months in remission	3	4	5	6	7	8	9	10	12	13	16	17	20
Monotherapy	6	24	28	22	10	5	4	2	0	1	0	0	0
Combination theraphy	1	13	15	9	1	0	0	1	3	1	1	1	1

Source: Clinical records from participating centers

The resolution of genital warts within 6 months was most rapid in the 25–34 age group (51 patients; 34.28%), followed by women aged 18–24 (31 patients; 20%) (Table 2).

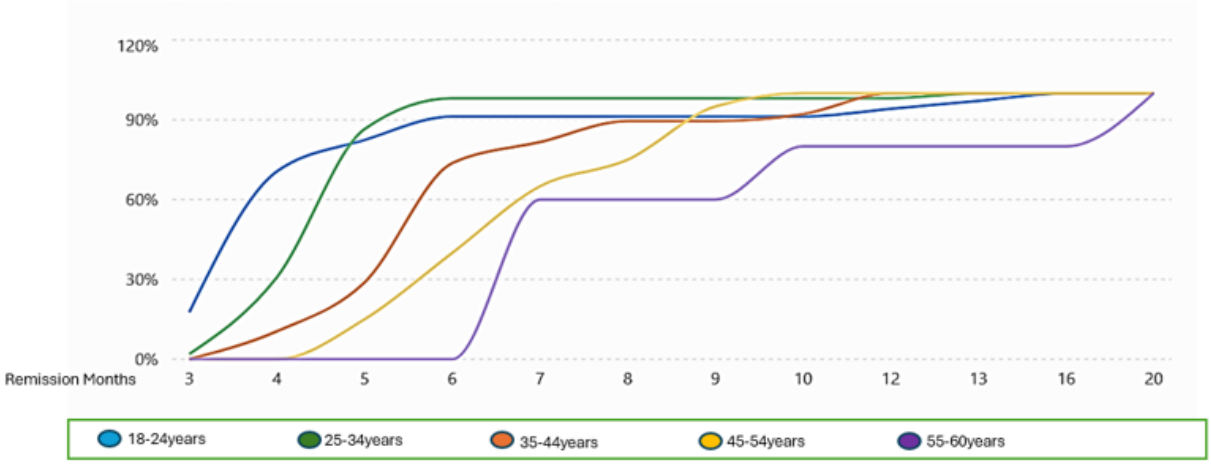
Table 2. Time in Months from the Start of Pidotimod Treatment to the Disappearance of Genital Warts by group (n=149)

Agee (years)	18-24		25-34		35-44		45-54		55-60		Total		%Total
Number of patients included	34(23%)	RA	25(34%)	RA	38(26%)	RA	20 (13%)	RA	5(3%)	RA	149	RA	100
Time in months from the start of Pidotimod treatment to the disappearance of genital warts													
3	6	17.6	1	1.9	0	0	0	0	0	0	7	4.7	5
4	18	70.6	15	30.8	4	10.5	0	0	0	0	37	29.5	25
5	4	82.4	29	86.5	7	28.9	3	15	0	0	43	58.4	29
6	3	91.2	6	98.1	17	73.7	5	40	0	0	31	79.2	21
7	0	91.2	0	98.1	3	81.6	5	65	3	60	11	86.6	7
8	0	91.2	0	98.1	3	89.5	2	75	0	60	5	89.9	3
9	0	91.2	0	98.1	2	94.7	2	85	0	60	4	92.6	2
10	0	91.2	0	98.1	1	97.4	1	90	1	80	3	94.6	2
11	0	91.2	0	98.1	1	100	0	90	0	80	1	95.3	1
12	1	94.1	0	98.1	0	100	1	95	0	80	2	96.6	1
13	1	97.1	1	100	0	100	0	95	0	80	2	98	1
16	1	100	0	100	0	100	0	95	0	80	1	98.7	1
17	0	100	0	100	0	100	1	100	0	80	1	99.3	1
20	0	100	0	100	0	100	0	100	1	100	1	100	1

RA= Accumulated remission **Source:** Clinical records from participating centers

6-month milestone; short-term cumulative remission rate: by the sixth month, 79.2% of the total population under treatment had already achieved complete remission. However, a clear difference based on age is observed: while remission exceeded 90% among patients aged 18 to 34, it barely reached 40% in the 45-to-54 age group (Graph 6).

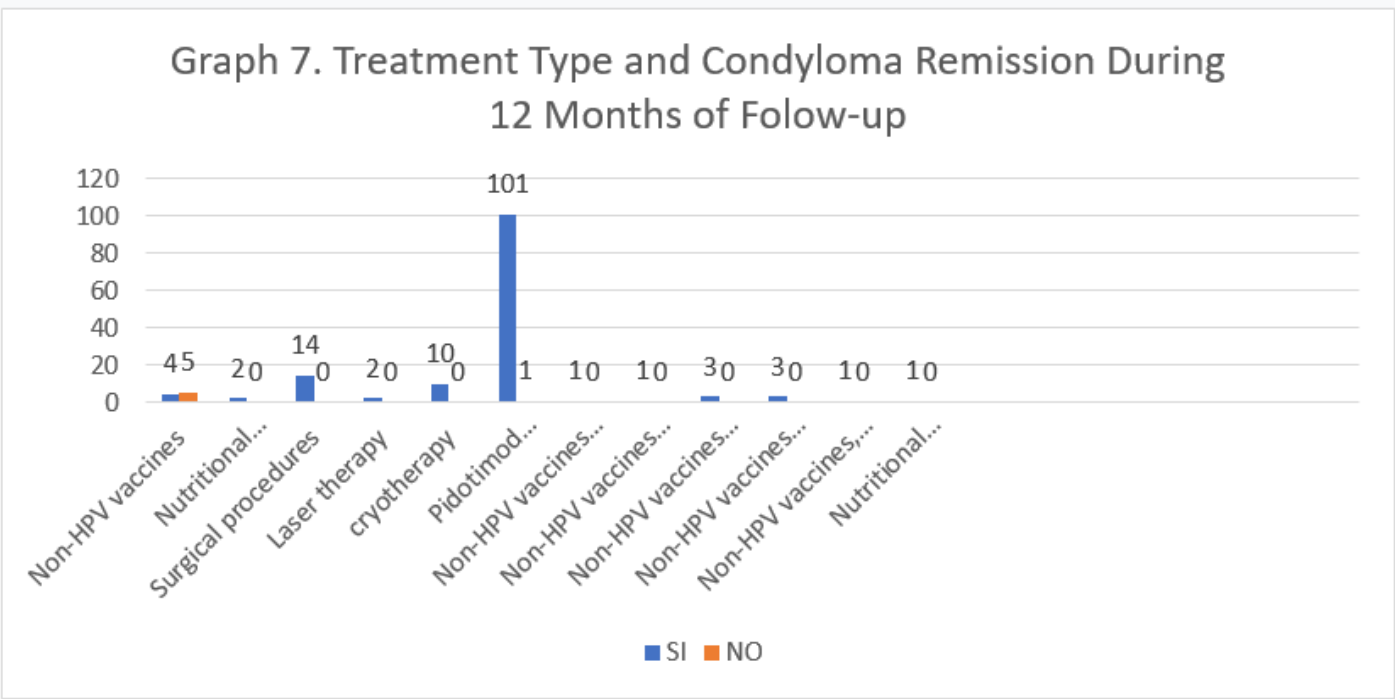
Graph 6. Cumulative Remission of Genital Warts According to age group. Inverse Kaplan- Meier curves illustrating the cumulative clearance of lesions after Treatment with Pidotimod (n=149)



Source: Clinical records from participating centers

During the 12-month follow-up, 143 patients (96%) showed improvement, and no recurrences were reported.

Regarding follow-up, 142 patients (95%) attended regular check-ups, with an average of 2.6 visits (range: 1 to 5) over the 12-month period. No adverse events were reported during treatment (Graph 7).



Source: Clinical records from participating centers

ANALYSIS OF RESULTS

Demographic Characteristics and Clinical Profile of the Sample

A total of 149 records of female patients meeting the inclusion criteria were analyzed, representing 58.2% of the total records reviewed (N=256) between 2007 and 2025. Epidemiological patterns by age group revealed that the most affected age range was 25 to 34 years (52 cases; 35%), followed by the 35-to-45-year-old group (38 cases; 26%). This distribution aligns with international literature, which indicates that the peak incidence of condylomata acuminata occurs during the decades of greatest sexual and reproductive activity [6,7].

In our cohort, 131 patients (88%) were of reproductive age. Notably, 100% of the sample was classified as immunocompetent and free of chronic comorbidities (such as uncontrolled diabetes or autoimmune diseases) that could directly bias viral clearance or treatment response. A definitive diagnosis of HPV infection was confirmed via biopsy in 88 cases (59%), followed by 41 cases (27%) confirmed through both biopsy and cytology. Regarding genotyping, 135 samples (91%) were not typed, whereas high-oncogenic-risk genotypes (HPV 16, 18, 31) were identified in 12 samples (8%); this finding was consistent with reports by Ochoa-Carrillo et al. (2015)1 for southern Mexico City, underscoring the importance of a comprehensive approach, given that coinfection with high-risk strains increases the risk of progression to high-grade squamous intraepithelial lesions [6,8].

Pidotimod Therapeutic Regimen

The Pidotimod dosage prescribed to the patients in our cohort was determined by each of the experts based on their daily clinical experience.

Pidotimod was predominantly administered at a dose of 800 mg every 24 hours for 60 days to 125 (84%) of the patients, while 23 (15%) received the same regimen extended to 90 days, administered two hours before or after meals to optimize bioavailability. In contrast, Guerra B (1998) reported evidence from a double-blind study using pidotimod to treat vulvar papillomatosis; the study design involved administering 800 mg of pidotimod daily for 90 days, demonstrating the drug's efficacy—comparable to the action of IFN-α and β—in treating the condition. Additionally, a study by Zervoudis S et al. (2010) concluded that pidotimod therapy (800 mg every 24 hours plus vitamin C for 15 days), following laser vaporization of genital warts, could be effective in reducing recurrence rates for HPV-related conditions.

The vast majority of the study population—141 patients (95%)—required only a single treatment cycle. Regarding the therapeutic modality, pidotimod was prescribed as monotherapy for 102 women (69%) and in combination with other conventional therapies for 47 patients (31.5%).

Recent studies highlight pidotimod as a therapeutic tool for managing HPV infection, given its mechanism of action—which targets both innate and adaptive immune responses—as it is a synthetic, non-specific immunomodulator that induces dendritic cell maturation and increases the expression of antigen molecules human leukocyte antigen subtype DR (HLA-DR) and other co-stimulatory molecules (CD83 and CD86); it drives T-cell differentiation toward the Th1 type (via the release of pro-inflammatory molecules through dendritic cell stimulation), increases NK cell activity, inhibits thymocyte apoptosis, promotes phagocytosis, increases salivary immunoglobulin A (IgA) levels, and regulates TLR-1 and TLR-2 signaling pathways in the epithelium. It also stimulates cellular immunity through the activation of macrophages and T lymphocytes, thereby enhancing the innate and adaptive response against HPV [2,4,5,9-15].

Evaluation of Clinical Remission and Regression Kinetics

The primary efficacy endpoint showed that 118 out of 149 (79.2%) patients in the total population achieved complete clinical remission (total disappearance of visible exophytic lesions) before completing 6 months of treatment. By the seventh month, the cumulative success rate had risen to 129 out of 149 (86.5%). When the rate of lesion disappearance was broken down by age group, a statistically significant difference was observed in the Kaplan-Meier curves. Remission at 6 months exceeded 90% in the cohort of young patients aged 18 to 34 years; the 25-to-34-year-old group showed the fastest response, comprising 51 patients (34.28%) a result very similar to that reported by Stein et al. (2026) [16], while women aged 18 to 24 years constituted the second group, with 31 patients (20%). The results observed in our cohort are consistent with those reported by Guerra et al. (1998)5, who evaluated 49 women with HPV-associated vulvar papillomatosis in a randomized, double-blind, placebo-controlled clinical trial. The authors found complete regression of lesions in 66.7% of patients treated with Pidotimod compared to 31.8% of those who received a placebo, demonstrating a significant reduction in the lesion area at the end of treatment. These findings support the potential immunomodulatory role of Pidotimod as an adjuvant therapy in patients with HPV infection.

In contrast, within the group undergoing the menopausal transition (aged 45 to 54), the cumulative remission rate at 6 months barely reached 40%. This marked chronological disparity suggests that, despite the patients being immunocompetent, physiological immunosenescence and age-related hormonal fluctuations alter local immune response kinetics and slow down drug-mediated cellular clearance [1,17].

Long-Term Follow-up: Recurrence and Persistence

During the 12-month period of strict clinical follow-up, 142 patients (95%) maintained consistent adherence to their scheduled visits (every 4 to 6 months). Long-term results revealed that 143 patients (96%) in the total sample experienced sustained clinical improvement without recurrence (defined as the reappearance of warts following initial remission). The remaining cases involved either treatment failure due to the persistence of initial lesions or events of recurrence/new infection; this represents a marginal percentage compared to the recurrence rates reported in the literature for conventional ablative treatments alone (such as cryotherapy or electrosurgery), which typically range from 20% to 30%. These findings statistically validate that the inclusion of Pidotimod strengthens local immunological memory, drastically reducing the risk of latent viral reactivation in the perilesional basal epithelium [18].

DISCUSSION

The main finding of this multicenter study demonstrates that the use of Pidotimod predominantly as monotherapy (69%), represents a highly effective clinical strategy for inducing macroscopic remission of vulvar, vaginal, cervical, and perianal condylomata acuminata. It achieved cumulative clearance rates of 79.2% at 6 months and 86.5% at 7 months, complemented by robust long-term protection, as evidenced by 96% of patients remaining recurrence-free at the 12-month follow-up. These results are of great clinical significance when compared with conventional local ablative or destructive therapeutic options such as cryotherapy, electrosurgery, or trichloroacetic acid [18]. Although these traditional treatments achieve rapid removal of visible exophytic tissue, they are historically associated with recurrence rates ranging from 20% to 30% during the first few months post-treatment18. This therapeutic failure stems from the fact that ablative procedures eliminate the physical lesion but do not eradicate the latent HPV viral load persisting in the keratinocytes of the basal layer of macroscopically healthy perilesional epithelium; this reservoir serves as the primary substrate for subsequent recurrences in the context of a deficient cellular immune response [8]. The low recurrence rate observed in our cohort (4%) after one year validates the hypothesis that systemic immunomodulation is key to breaking the cycle of HPV recurrence. Pidotimod, a synthetic dipeptide (3-L-pyroglutamyl-L-thiazolidine-4-carboxamide), acts directly by optimizing the innate and adaptive immune responses through dendritic cell maturation, macrophage stimulation, and Toll-like receptor (TLR) expression, as well as by promoting a T-helper type 1 (Th1) effector response and increasing Natural Killer (NK) cell activity [19].

This immunological cascade promotes the release of proinflammatory cytokines such as interferon-gamma (IFN-γ) and interleukin-2 (IL-2), critical biological elements for the host to achieve clearance of HPV-infected keratinocytes [15].

Thus, while commonly used topical immunomodulators such as 5% imiquimod are limited to inducing a localized inflammatory response frequently accompanied by local adverse effects such as erosion, erythema, and intense pain that compromise therapeutic adherence [18].

Oral pidotimod provides a generalized and well-tolerated immunological optimization that acts systemically throughout the anogenital epithelium.

A critical analytical finding in our research is the marked disparity in remission kinetics when stratifying the population by age ranges using Kaplan-Meier curves. It was observed that younger patients (18 to 34 years old) responded significantly more rapidly, exceeding 90% clinical remission at 6 months of treatment. Conversely, the 45 to 54year old group exhibited a markedly lower cumulative remission rate (40%) over the same period. This phenomenon can be explained by the physiological process of immunosenescence and the hormonal decline of menopause. With advancing age, there is progressive thymic involution and an alteration in T-cell homeostasis, characterized by a reduction in the repertoire of naïve T cells and a decreased capacity for clonal proliferation in response to novel or persistent viral antigens [20]. Furthermore, the transition to the postmenopausal state involves a hypoestrogenism induces epithelial atrophy, alters the vaginal microbiota, and modifies the local mucosal immune response; these factors slow the rate at which the immune system even when stimulated by Pidotimod destroys tumor or infected cells [20]. This behavior underscores the need to individualize clearance expectations in daily clinical practice, suggesting that older patients might justifiably require the extended 90 day regimen evaluated in 15% of our sample or combination therapies from the outset.

We acknowledge that the study's main limitation lies in its retrospective design based on a review of medical records and the absence of a concurrent control group receiving a placebo or destructive therapy alone. Furthermore, as molecular HPV typing was not performed in 91% of cases due to the private practice setting, it was not possible to correlate remission or persistence kinetics with specific low- or high-oncogenic-risk genotypes. Nevertheless, the research's strengths lie in its multicenter, longitudinal nature; the use of a complete census with strict 12-month follow-up based on standardized colposcopic and gynecological evaluation criteria applied by specialists; and the analyzed sample size (n=149), which represents one of the largest clinical series reported on the off-label use of oral Pidotimod for this specific indication in a Latin American population.

CONCLUSIONS

1. The use of orally administered Pidotimod (Adimod) constitutes a highly effective and safe therapeutic strategy for the comprehensive management of immunocompetent women with genital warts (condylomata acuminata) associated with Human Papillomavirus (HPV).
2. The drug demonstrates a remarkable capacity to induce complete, macroscopic clinical remission of exophytic lesions in the short term, with the vast majority of the study population achieving resolution before completing six months of treatment.
3. The regression kinetics of genital warts are significantly influenced

by the patient's age. Women in younger age groups (18 to 34 years) exhibit accelerated clearance rates compared to those in peri- and postmenopausal stages (45 to 54 years); this suggests that factors associated with local physiological immunosenescence slow the response rate, justifying longer or combined treatment regimens for this subgroup.

4. The incorporation of Pidotimod drastically reduces the long-term clinical recurrence rate (12-month follow-up). This validates its clinical benefit regarding cellular immunological memory—as opposed to conventional destructive or ablative therapies used in isolation, which often show high recurrence rates due to the persistence of the latent virus in healthy perilesional epithelium.
5. Findings from this multicenter study in private practice support the recommendation to consider systemic immunomodulation—either as monotherapy or as a cornerstone of combination regimens—within current clinical algorithms to optimize the clearance of HPV lesions in the Mexican female population.

FINAL CONCLUSION

This study provides relevant clinical evidence positioning Pidotimod as a safe, effective therapeutic option with excellent patient adherence for the comprehensive management of genital warts. Its ability to induce sustained remission and drastically reduce recurrence rates supports its use both as monotherapy for patients with newly developed lesions and within combination regimens to optimize local biological clearance. Large-scale, randomized, double-blind, controlled clinical trials are recommended to consolidate these findings and formally incorporate this immunomodulator into international clinical practice guidelines for the management of anogenital HPV.

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CONFLICTS OF INTEREST

The authors declare having received financial compensation for the time dedicated to reviewing and extracting data from clinical records at their respective centers. Apart from this support for the study's operational phase, the authors declare no commercial, financial, personal, or academic conflicts of interest regarding the manufacturer of Pidotimod or any other biological or pharmaceutical entity that could have influenced the study design, statistical analysis, or interpretation of the presented results.

ETHICAL CONSIDERATIONS

1. This research protocol was designed, structured, and executed in strict compliance with the ethical principles for medical research involving human subjects established in the World Medical Association's Declaration of Helsinki [21], and its subsequent amendments. Furthermore, the study adhered to the provisions of the Regulations of the General Health Law on Health Research currently in force in Mexico; it was classified pursuant to Article 17—as "no-risk research," given that it was a retrospective study based exclusively on the review of medical records, involving no deliberate intervention or modification of biological, psychological, or social variables in the patients [22].

To ensure the confidentiality and privacy of the information, and in accordance with current personal data protection regulations, all records were rigorously anonymized at the source. Data extraction was performed using coded numerical identifiers, permanently removing any personally identifiable information (names, social security numbers, identity card numbers, or patient photographs) from the databases analyzed in the statistical software.

Given the purely retrospective nature of the analysis and the fact that data collection took place years after the patients had completed their routine clinical follow-up in private practice (records from the 2007–2025 period), the requirement for written informed consent was waived. Nevertheless, it was ensured that the information was used strictly for scientific and continuing medical education purposes, with the data securely safeguarded under the custody of the principal investigators.

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