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ACUTE AND CHRONIC HERPES ZOSTER INFECTION MEDICAL TREATMENT: STILL A ROLE FOR INNATE IMMUNITY MODULATION

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ABSTRACT

Background: The updated treatment of acute and chronic zoster infections encloses antiviral, painkillers neuropathic drugs for postherpetic neuralgia, anti-inflammatory compounds and vaccines. The persistent viability of the virus into the posterior horns of the medulla, makes its virulence highly resistant to the current protocols and suggests a reconsideration of an old bacteriotherapy enhancing the innate immunity and direct phagocytosis of the virus at any stage: the inactivated *Cutibacterium acnes*, formerly *Corynebacterium parvum*. The aim of this study is to counter Zoster neuropathic pain with Corynebacterium parvum.

Materials and Methods: A total of 300 patients were treated using C.parvum injection (2.5 cc of Parvulan added with 0.3 ml 1% lidocaine), in the subcutaneous deltoid area at any stage, affected by primary dermatomal zoster (64 cases), by genital Zoster (16 cases), by first infection (34 cases), by frequently relapsing primary zoster (14 cases), by Zoster neuralgia (46 cases), by Zoster infection in patients with cancer (32 cases), by cancer under chemotherapy [62 cases (46 men and 16 women)], by Zoster infection in severe immunodepression (28 cases), by Zoster encephalomyelitis (4 cases).

Results: Chronic neuralgia was successfully alleviated in most treated patients in a few weeks after the treatment.

Conclusion: in conclusion in the setting of zosteridae family side to the most updated pharmacology, we still recommend to take into account also an old historical weapon such as the C. parvum bacteriotherapy, because of its safe and effective direct antiviral activity.

***Keywords*:** Corynebacterium parvum, herpes zoster, innate immunity

INTRODUCTION

Nine human herpes viruses have hitherto been described.According to recently updated nomenclature[1], these are human alpha-herpes-virus 1 (herpes simplex virus type 1), human alpha-herpes-virus 2 (herpes simplex virus type 2), human alpha-herpes-virus 3 (varicella-zoster virus), human gamma-herpes-virus 4 (Epstein–Barr virus), human beta-herpes-virus 5 (human cytomegalovirus), human beta-herpes-virus 6A (human herpes-virus 6A), human beta-herpes-virus 6B (human herpes virus 6B), human beta-herpes-virus 7 (human herpes-virus 7), and human gamma-herpes-virus 8 (Kaposi's sarcoma herpes virus). Typical of this family is the property of long-term or life-long immunopathological colonization[2] in a non-replicative state. Immunosuppressive conditions, senescence medical or surgical interventions or infections by other viruses, can reverse the latency phase and awaken more aggressive infection. Triggered by the Covid-19 enclosed a huge amount of varicella-herpes zoster and simplex infection. Shanshal et Ahmed (2021) suggested a possible relationship between Covid-19 infection and primary Herpes simplex virus (HSV) infection or reactivation[3]; based on this report Covid-19 direct neuronal effect in addition to Covid-19-related psychological stress, fever, and immunological dysregulation could play a potential role in HSV reactivation or primary infection during Covid-19.

Almutairi and coworkers (2022) reviewed the literature reports about cases of herpes zoster (HZ) infection at the time of the current pandemic either in patients infected with COVID-19, whether with respiratory symptoms or not, but also with undetectable subclinical virus colonization and after COVID-19 vaccination[4]. The paper warns about the probable increased hazard of HZ during the pandemic and the need of preventive and curative interventions.

Due to the soaring of Herpes virus family epidemiology, during and after the current pandemics we have been stimulated to perform a therapeutic update especially on the Hesv 1 and 2, but also Vzv, whose acute infections are often dramatically painful, and followed by persistent pain for many years.

Herpes simplex virus type 1 (HSV-1; human herpes virus 1) and varicella-zoster virus (VZV; human herpes virus 3) infect the humans early in life. VZV infection is responsible of childhood chickenpox[5]. Primary HSV-1 infection is usually localized and may be asymptomatic, except for immunocompromised neonates and children[6]. The neurotropic invasion of both the strains goes back from the skin vesicles to neurons through axonal conduction but VZV spreads also via hematogenous route at the viraemic stage of infection using memory T-cells as trojan horses to colonize the dorsal root ganglia in a latent stage [7,8]; an infection "round trip stage" is also possible, running the virions from ganglia to skin lesions and then retrogradely invade new axons and ganglia cells. The clinical symptoms of HSV1 are usually the "cold labial sores", rarely external genital infections genital herpes, herpetic keratitis, and herpetic whitlow or eczema herpetic, more rarely encephalitis, and blindness[9]. VZV causes the "shingles", a painful vesicular dermatitis, with a vesicular-pustular appearance in 7 to 10 days, eventually evolving into a crust and healing 2, 3 weeks after the onset. Seventy to eighty percent of patients with herpes zoster complain of allodynia, prodromal symptoms, such as burning, shooting, stabbing, or throbbing pain in the dermatome(s)[10,11]. Very often followed by the post herpetic neuralgia (causing unexpected, excruciating burning or piercing pain flares, but also meningoencephalitis, paresis, and cranial nerve palsies, vasculopathies and eyes damages[12]. The latent phase of VZV infection is responsible of acute, subacute or chronic relapsing encephalitis, myelitis, Poly radiculitis[13]. In this ecliptic stage, the viruses viable into the ganglia cells undergo modification of the genome structure involving restriction viral gene transcription where the end of its' DNA strands are restricted and connected to form unit length epistomes and concatemers. The primary latency associated transcript (LAT) imprint specific RNAs spliced introns; similarly, in the VZV latency transcripts to 12 genes are reported. The mechanisms by which HSV-1 and VZV maintain latency into the human neurons, are quite different, being the former relapsing frequently, in younger people, while the latter relapse more rarely preferably in the elderly[14]. The estimated incidence is of Zoster infection is from 3 to 5 per 100.000 [increasing progressively with age[15] in, 10 to 34% of the infected patients develop postherpetic neuralgia (PHN), not subsiding after the skin resolution [16] classified in acute (30 days) sub-acute (60-120 days) and chronic, with a variably poor life quality, and daily activity duties impairment; the neuron inflammation[17] due to interleukin 6 release or/and immune reaction synchronous with the post-latency viral reactivation, are involved in the physiopathology of the neuropathic symptoms.

Post herp neuralgia occurs in 5-20% patients with zoster infection, increasing with age from 20 to 30% in 60 to 80 years old affected patients; the prodromic level of pain, (high intensity pain) with severe rush, severe immunodepression[18]. Pre-existence of lupus, diabetes and recent onset trauma are general predictors of post herpetic neuralgia[19]. The physiopathology of the syndrome in the previously scarred dermatome is due to an inflammatory cytokine mediated damage to the central neurons lowering the pain threshold with allodynia dysesthesias, paraesthesia, hyperalgesia chronic, burning aching pain with stabbing spikes [20].

The most common symptomatic treatments enclose:

Tricyclic antidepressants (TCAs) including nortriptyline, desipramine,

Calcium channel α2-δ ligands (gabapentin, gastroretentive gabapentin, gabapentin enacarbil and pregabalin);

Opioids and topical capsaicin patch or cream as second- or third-line treatment options; or combination therapies with different mechanisms of action, including lidocaine 5% patch[21-23].

Second and third line enclose opioid analgesics such as tramadol, oxycodone, morphine, and methadone. These drugs have been variously combined tailoring the treatment based on the severity of the symptoms.

Topical agents and creams enclose capsaicin 8% patch, capsaicin 0,075% cream, and zovirax,

Intrathecal glucocorticoids should be useful to control severe pain.

Cutibacterium acnes formerly Corynebacterium parvum, or Propionibacterium acnes, is a commensal of the human healthy skin probiotic, involved in the inflammatory process of acne; it' s physiologic role is the defence of epidermis integrity against pathogenic aggression by bacteria viruses and fungi, balancing especially the staphylococcus species and it' s damaging potential.

The inactivated bacterium was originally injected subcutaneously and dramatically enhanced the innate

immunity and macrophagic activity through lymph node stimulation; the challenge of the c.parvum triggered innate immunity cells against solid or lympho-haematological cancers with a strong oncolytic effect through mixed lympho-monocytic infiltrate of the cancer tissue and direct phagocytosis of cells clusters; the immunomodulant activity against cancer was industrially endorsed and marketed by the Wellcome-Burroughs company with the label: "Coryparv".

This drug plainly (without adjuvants) the phenol-killed bacterium Corynebacterium parvum, ATCC strain N°12930 to be subcutaneously injected with different protocols in a variety of cancers.

During the Coryparv oncological campaign, we became aware of its terrific potential against viral infections in cancerous patients, namely the varicella-herpes zoster and herpes simplex family, but also EBV, CMV, HPV etc[24].

Acute Herpes zoster infection with shingles and pain were defeated in 48-96 hours; in the latency phase of chronic herpetic neuralgia the symptoms were markedly improved or disappeared, with different treatment schedules: In most of the cases, with recent onset of the infection, a single dose is permanently effective to eradicate the neuralgia, in others, with longer clinical history the procedure has to be repeated twice or more times to achieve permanent remission: the drug labelled coryparv (Corynebacterium parvum) has been world-widely available in the pharmacies until 1983 when the Wellcome-Burroghs was bought by GLAXO, whose market policy was massively oriented to ranitidine in Gastroenterology rather than dealing with Oncological products; the registration was thus not renewed and we were able to find the identical injectable formula in Brazil with the brand name of Parvulan. The antiviral properties of this Brazilian formulation were unchanged, and in its drug leaflet instructions also for warts treatment are reported. The aim of this study is to counter Zoster neuropathic pain with Corynebacterium parvum.

2. MATERIALS AND METHODS

Our spontaneous anecdotical report encloses 300 cases (170 females and 130 males) aged 24 -95 (median age 55) years old applied between 2018 and 2023, to our "Second Opinion Medical Consulting Network" (Modena, Italy), looking for therapeutic solution to counteract the symptoms of Herpes Zoster for this anecdotal, spontaneous, and retrospective trial. The Second Opinion Medical

Network is a consultation referral web and Medical Office System enclosing a wide panel of specialists, to whom any patient with any illness or syndrome that is not adequately satisfied by the previous diagnosis or therapy can be applied for an individual telematic or front office clinical audit[25-27]. We have used C.parvum injection (2.5 cc of Parvulan added with 0.3 ml 1% lidocaine), in the subcutaneous deltoid area at any stage, affected by primary dermatomeric zoster (64 cases), by genital Zoster (16 cases), by first infection (34 cases), by frequently relapsing primary zoster (14 cases), by Zoster neuralgia (46 cases), by Zoster infection in patients with cancer (32 cases), by cancer under chemotherapy [62 cases (46 men and 16 women)], by Zoster infection in severe immunodepression (28 cases), by Zoster encephalomyelitis (4 cases).

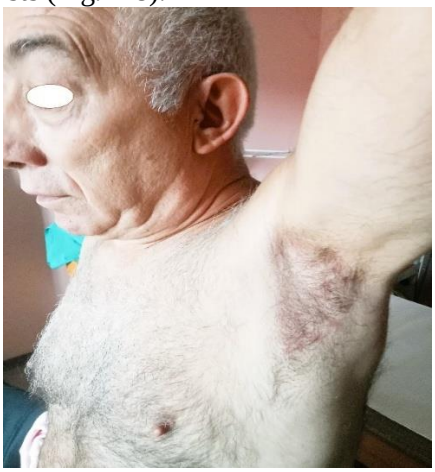
3. RESULTS

The successful clinical outcome of C.parvum administration in different clinical stages of Zoster infection, supposes this kind of bacteriotherapy, which is effective whatsoever the date of the primary virus replication into the host, or the latency symptomatic persistence in the Nervous ganglia or immune cells (Table 1).

Table 1. description of clinical cases

Time to stop the vesicles eruption	72-96 hours
Time to pain relieve	48-72 hours
Time to skin recovery	4-6 weeks
Herpetic neuralgia complete relieve	n.40 cases
Improvement more than 70%	n.3 cases
No improvement	n.1 case
Zoster Encephalomyelitis	n.4 cases: 1) 1 case complete relieve with a single injection shot; 2) 1 case complete relieve with 4 total injections 3) 1 case totally refractory to the treatment 4) 1 partially improved requiring drug therapy
Frequently relapsing HZ	The treatment has been repeated monthly for total 3 – 5 sessions
Untoward effects of the treatment	Not reported

Amazingly, in the viral vesicular skin phase the vesicles are dried in 72, 96 hours, and pain relieve is observed as well in a very short time. Chronic neuralgia is successfully counteracted in almost the treated patients in a few weeks after the treatment. Also, zoster encephalomyelitis has individual positive feedback with bacteriotherapy, but very often it requires integration with some psycho-neurotropic drugs enclosing PEA and CBD to definitely relieve the complexity of the brain circuits. As to the frequent relapsing skin infection, our treatment schedule is very effective to eradicate the virus, but the bacteriotherapy has to be modulated along the way on the basis of the frequency and intensity of the Zoster bursts (Fig. 1-8).



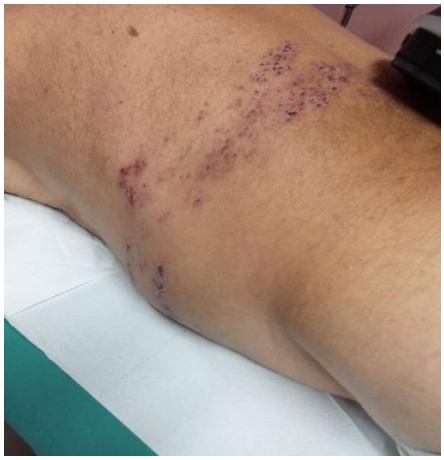
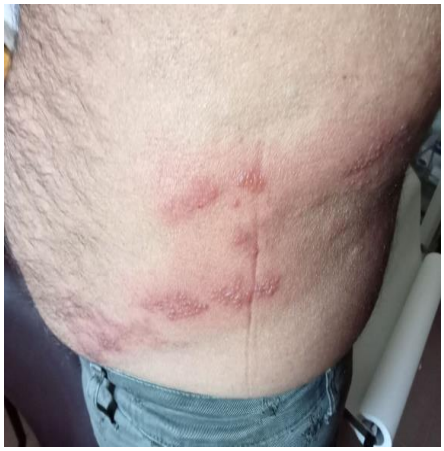


Figure 1. Sample of Herpes Zoster infection along single selected dermatomer
Figure 2. Sample of Herpes Zoster infection along single selected dermatomer



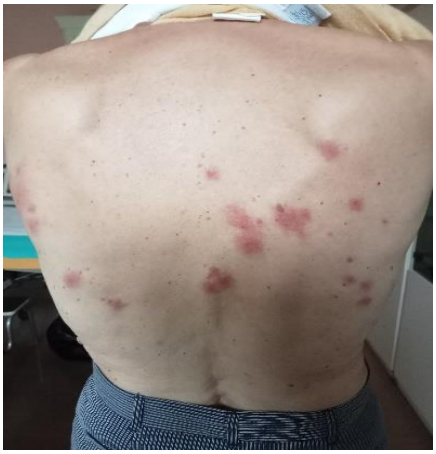
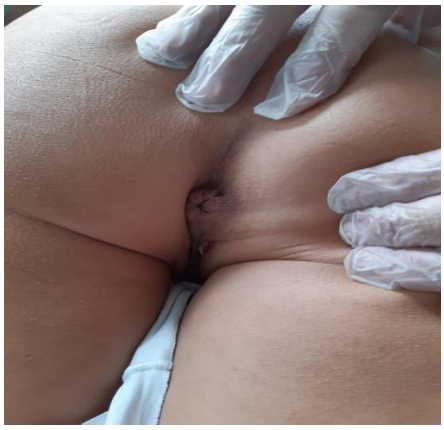


Figure 3. Sample of Herpes Zoster infection along multiple selected dermatomer
Figure 4. Sample of Herpes Zoster infection along multiple selected dermatomers.



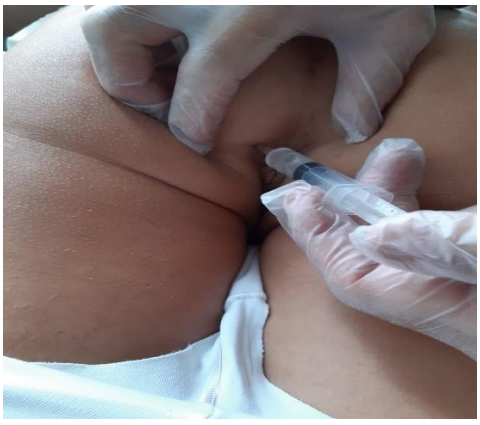


Figure 5. Perianal relapsing Zoster infection; Parvulan injection is performed preferably in the subcutaneous

involved area to achieve a stronger chemotactic T-cells induction against viruses.

Figure 6. Perianal relapsing Zoster infection; Parvulan injection is performed preferably in the subcutaneous involved area to achieve a stronger chemotactic T-cells induction against viruses.

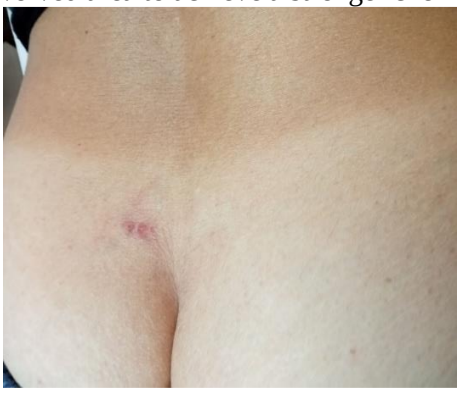


Figure 7. Early sacral Herpes Infection immediately counteracted by local Parvulan challenge

Figure 8. Early sacral Herpes Infection immediately counteracted by local Parvulan challenge

4. DISCUSSION

Our experience with C.Parvum bacteriotherapy against HZ infection started successfully a few years before Acyclovir registration and clinical use and was amazingly able to induce viral regression also in the chronically painful latency phase since the beginning of its use. Even if vaccination is the first class gold standard for the control of the infection, notwithstanding C.parvum is worth to be considered whenever the virus overwhelms the specific antibody response and suddenly outbursts.

Acyclovir and its derivatives act more slowly in comparison with the fast activation of innate immunity cells to directly phagocytize the virus, rather than interrupt its intracellular cycle, a more cumbersome duty to reach the same goal.

The advantage of C.parvum bacteriotherapy (CPBTH) is that the lympho-monocytes macrophages and dendritic/APC cells are permanently recruited, to fight the virus raids and prevent the post latency activation; therefore the infection relapse is prevented for months or years, as long as the cells are viable and their vital cycle is fulfilled.

In our cases the CPBTH re-administration is subjectively variable and depends, by the aggressivity of the viral strains, by the individual host immune function and other environmental cofactors.

The C.parvum injection is usually performed in the forearm subcutaneous tissue, but in case of relapse it can be successfully injected into the skin close to the lymph nodes draining the infected areas, or even, mixed with a small local anaesthetic amount, directly. Under the herpes vesicles to achieve a quicker and stronger chemotactic T cells recruitment.

We cannot thus standardize the C.parvum treatment schedule that have to be tailored accordingly with each clinical history on the basis of the goal of defeating postherpetic neuralgia is almost often achieved even in long standing histories of pain, achieving complete clearance of the viral particles in the posterior medullary horns by phagocytosis.

The CBPTH success in the chronic pain and even CNS-chronic neuropathy, cannot be assured neither by the antiviral treatments, nor by the vaccines, because the elicited antibodies are not supposed to be able to reach the intraneuronal hidden viruses and neutralize their nociceptive activity.

The CBPTH success in the chronic pain and even CNS-chronic neuropathy, cannot be assured neither by the antiviral treatments, nor by the vaccines, because the elicited antibodies are not supposed to be able to reach the intraneuronal hidden viruses and neutralize their nociceptive activity. Based on the literature update, the acne process physiopathology is the challenge of the innate immune system by Corynebacterium parvum (C. parvum) or Propionibacterium acnes (P. acnes), through a toll-like receptor 2 (TLR2)- dependent mechanism with activation of macrophages and monocytes to produce both interleukin 8 (IL-8), a neutrophil chemoattractant, and IL-12, a key regulator of type 1 T helper (Th1) cell responses[28,29]. T lymphocytes are then recruited to destroy the bacterial intrusion turning the innate immunity against the same pathogenic agent itself[30].

Thus, the antiviral activity of C. parvum can be considered a natural evolution and exploitation of its proinflammatory activity originally exerted in the sebaceous glands and keratinocytes with potentiation of their TLR receptors.

The antiviral activity of C. parvum can be considered a natural evolution and exploitation of its proinflammatory activity originally exerted in the sebaceous glands and keratinocytes with potentiation of their TLR receptors. This peculiar offence/defence relationship among C.parvum pathogenicity and environmental feedback

can clinically be adopted turning the innate immunity not against the viable C. parvum, like in the acnes model, rather to prevent the entrance and survival of pathogenic agents, viruses bacteria and fungi in any part of the body by means of subcutaneous injections of inactivated C.parvum at standardized concentrations, as a form of nonspecific surrogate vaccination[35].

CONCLUSION

in the setting of zosteridae family side to the most updated pharmacology, we still recommend to take into account also an old historical weapon such as the C.parvum bacteriotherapy, because of its safe and effective direct antiviral activity; a whole complex inactivated bacterium sometime is better and faster to elicit the viral clearance by direct phagocytosis than a chemical drug with an individual molecular target, or of a vaccine, with its multistep antibodies induction; the fast track activation of innate immunity cells and the short average time (48 -72 hours) of Zoster symptoms improvement, by C.parvum administration, renders it very appealing, and outmost safe especially when the other treatments fail and symptoms relapse.

6. Patents

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Informed Consent Statement:

Written informed consent has been obtained from the patient(s) to publish this paper.

Data Availability Statement. Data Availability Statements are available in the manuscript.

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Conflicts of Interest: The authors declare no conflicts of interest.

DECLARATIONS

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Competing Interests

The authors have no competing interests to declare.

Ethical Approval

The study was approved by the appropriate ethics committee and conducted according to relevant guidelines and regulations.

Informed Consent Not applicable.

REFERENCES

1. Walker, P.J.; Siddell, S.G.; Lefkowitz, E.J.; Mushegian, A.R.; Dempsey, D.M.; Dutilh, B.E.; Harrach, B.; Harrison, R.L.; Hendrickson, R.C.; Junglen, S.; et al. Changes to Virus Taxonomy and the International Code of Virus Classification and Nomenclature Ratified by the International Committee on Taxonomy of Viruses (2019). Arch. Virol. 2019, 164, 2417–2429, doi:10.1007/s00705-019-04306-w.

2. Weidner-Glunde, M.; Kruminis-Kaszkiel, E.; Savanagouder, M. Herpesviral Latency—Common Themes. Pathogens 2020, 9, 125, doi:10.3390/pathogens9020125.

3. Shanshal, M.; Ahmed, H.S. COVID-19 and Herpes Simplex Virus Infection: A Cross-Sectional Study. Cureus 2021, doi:10.7759/cureus.18022.

4. Almutairi, N.; Almutairi, A.N.; Almazyad, M.; Alwazzan, S. Herpes Zoster in the Era of COVID 19: A Prospective Observational Study to Probe the Association of Herpes Zoster with COVID 19 Infection and Vaccination. Dermatol. Ther. 2022, 35, doi:10.1111/dth.15521.

5. Ayoade, F.; Kumar, S. Varicella-Zoster Virus (Chickenpox). In StatPearls; StatPearls Publishing: Treasure Island (FL), 2023.

6. Arduino, P.G.; Porter, S.R. Herpes Simplex Virus Type 1 Infection: Overview on Relevant Clinico-pathological Features*. J. Oral Pathol. Med. 2008, 37, 107–121, doi:10.1111/j.1600-0714.2007.00586.x.

7. Smith, G. Herpesvirus Transport to the Nervous System and Back Again. Annu. Rev. Microbiol. 2012, 66, 153–176, doi:10.1146/annurev-micro-092611-150051.

8. Arvin, A.M.; Moffat, J.F.; Sommer, M.; Oliver, S.; Che, X.; Vleck, S.; Zerboni, L.; Ku, C.-C. Varicella-Zoster Virus T Cell Tropism and the Pathogenesis of Skin Infection. Curr. Top. Microbiol. Immunol. 2010, 342, 189–209, doi:10.1007/82_2010_29.

9. Saleh, D.; Yarrarapu, S.N.S.; Sharma, S. Herpes Simplex Type 1. In StatPearls; StatPearls Publishing: Treasure Island (FL), 2023.

10. Hama, Y.; Shiraki, K.; Yoshida, Y.; Maruyama, A.; Yasuda, M.; Tsuda, M.; Honda, M.; Takahashi, M.; Higuchi, H.; Takasaki, I.; et al. Antibody to Varicella-Zoster Virus Immediate-Early Protein 62 Augments Allodynia in Zoster via Brain-Derived Neurotrophic Factor. J. Virol. 2010, 84, 1616–1624, doi:10.1128/JVI.02061-09.

11. Dworkin, R.H.; Johnson, R.W.; Breuer, J.; Gnann, J.W.; Levin, M.J.; Backonja, M.; Betts, R.F.; Gershon, A.A.; Haanpää, M.L.; McKendrick, M.W.; et al. Recommendations for the Management of Herpes Zoster. Clin. Infect. Dis. 2007, 44, S1–S26, doi:10.1086/510206.

12. Gilden, D.; Nagel, M.A.; Cohrs, R.J.; Mahalingam, R. The Variegate Neurological Manifestations of Varicella Zoster Virus Infection.

Curr. Neurol. Neurosci. Rep. 2013, 13, 374, doi:10.1007/s11910-013-0374-z.
13. Gnann, Jr., J.W. Varicella-Zoster Virus: Atypical Presentations and Unusual Complications. J. Infect. Dis. 2002, 186, S91–S98, doi:10.1086/342963.
14. Ou, Y.; Davis, K.A.; Traina-Dorge, V.; Gray, W.L. Simian Varicella Virus Expresses a Latency-Associated Transcript That Is Antisense to Open Reading Frame 61 (ICP0) mRNA in Neural Ganglia of Latently Infected Monkeys. J. Virol. 2007, 81, 8149–8156, doi:10.1128/JVI.00407-07.
15. Marra, F.; Parhar, K.; Huang, B.; Vadlamudi, N. Risk Factors for Herpes Zoster Infection: A Meta-Analysis. Open Forum Infect. Dis. 2020, 7, ofaa005, doi:10.1093/ofid/ofaa005.
16. Christo, P.J.; Hobelmann, G.; Maine, D.N. Post-Herpetic Neuralgia in Older Adults: Evidence-Based Approaches to Clinical Management. Drugs Aging 2007, 24, 1–19, doi:10.2165/00002512-200724010-00001.
17. Zhu, J.; Cao, D.; Guo, C.; Liu, M.; Tao, Y.; Zhou, J.; Wang, F.; Zhao, Y.; Wei, J.; Zhang, Y.; et al. Berberine Facilitates Angiogenesis Against Ischemic Stroke Through Modulating Microglial Polarization via AMPK Signaling. Cell. Mol. Neurobiol. 2019,39,751–768
18. Johnson, R.W.; McElhaney, J. Postherpetic Neuralgia in the Elderly. Int. J. Clin. Pract. 2009, 63, 1386–1391, doi:10.1111/j.1742-1241.2009.02089.x.
19. Forbes, H.J.; Thomas, S.L.; Smeeth, L.; Clayton, T.; Farmer, R.; Bhaskaran, K.; Langan, S.M. A Systematic Review and Meta-Analysis of Risk Factors for Postherpetic Neuralgia. Pain 2016, 157, 30–54, doi:10.1097/j.pain.0000000000000307.
20. PNS 2022 Abstract Supplement. J. Peripher. Nerv. Syst. 2022, 27, doi:10.1111/jns.12496.
21. Fashner, J.; Bell, A.L. Herpes Zoster and Postherpetic Neuralgia: Prevention and Management. Am. Fam. Physician 2011, 83, 1432–1437.
22. Nalamachu, S.; Morley-Forster, P. Diagnosing and Managing Postherpetic Neuralgia. Drugs Aging 2012, 29, 863–869, doi:10.1007/s40266-012-0014-3.
23. Dworkin, R.H.; O'Connor, A.B.; Backonja, M.; Farrar, J.T.; Finnerup, N.B.; Jensen, T.S.; Kalso, E.A.; Loeser, J.D.; Miaskowski, C.; Nurmikko, T.J.; et al. Pharmacologic Management of Neuropathic Pain: Evidence-Based Recommendations. Pain 2007, 132, 237–251, doi:10.1016/j.pain.2007.08.033.
24. Palmieri, B.; Vadalà, M.; Roncati, L.; Garelli, A.; Scandone, F.; Bondi, M.; Cermelli, C. The Long-standing History of Corynebacterium Parvum, Immunity, and Viruses. J. Med. Virol. 2020, 92, 2429–2439, doi:10.1002/jmv.26100.
25. Palmieri, B.; Iannitti, T. The Web Babel Syndrome. Patient Educ. Couns. 2011, 85, 331–333, doi:10.1016/j.pec.2011.02.019.
26. Palmieri, B.; Iannitti, T.; Capone, S.; Fistetto, G.; Arisi, E. [Second opinion clinic: is the Web Babel Syndrome treatable?]. Clin. Ter. 2011, 162, 575–583.
27. Palmieri; Vadalà The "Second Opinion Medical Network." Int J Pathol Clin Res.
28. Leyden, J.J.; McGinley, K.J.; Mills, O.H.; Kligman, A.M. Propionibacterium Levels In Patients With And Without Acne Vulgaris. J. Invest. Dermatol. 1975,65,382–384, doi:10.1111/1523-1747.ep12607634.
29. Kamisango, K.; Saiki, I.; Tanio, Y.; Okumura, H.; Araki, Y.; Sekikawa, I.; Azuma, I.; Yamamura, Y. Structures and Biological Activities of Peptidoglycans of Listeria Monocytogenes and Propionibacterium Acnes. J. Biochem. (Tokyo) 1982, 92, 23–33, doi:10.1093/oxfordjournals.jbchem.a133918.
30. Kim, S.B.; Corapcioglu, M.Y. Vertical Transport of Cryptosporidium Parvum Oocysts Through Sediments. Environ. Technol. 2002, 23, 1435–1446, doi:10.1080/09593332508618448.
31. Vowels, B.R.; Yang, S.; Leyden, J.J. Induction of Proinflammatory Cytokines by a Soluble Factor of Propionibacterium Acnes: Implications for Chronic Inflammatory Acne. Infect. Immun. 1995, 63, 3158–3165, doi:10.1128/iai.63.8.3158-3165.1995.
32. Liu, J.; Enomoto, S.; Lancto, C.A.; Abrahamsen, M.S.; Rutherford, M.S. Inhibition of Apoptosis in Cryptosporidium Parvum Infected Intestinal Epithelial Cells Is Dependent on Survivin. Infect. Immun. 2008, 76, 3784–3792, doi:10.1128/IAI.00308-08.
33. McInturff, J.E.; Wang, S.-J.; Machleidt, T.; Richard Lin, T.; Oren, A.; Hertz, C.J.; Krutzik, S.R.; Hart, S.; Zeh, K.; Anderson, D.H.; et al. Granulysin-Derived Peptides Demonstrate Antimicrobial and Anti-Inflammatory Effects Against Propionibacterium Acnes. J. Invest. Dermatol. 2005, 125, 256–263,
34. Zhang, B.; Choi, Y.M.; Lee, J.; An, I.S.; Li, L.; He, C.; Dong, Y.; Bae, S.; Meng, H. Toll-like Receptor 2 Plays a Critical Role in Pathogenesis of Acne Vulgaris. Biomed. Dermatol. 2019, 3, 4,
35. Palmieri, B.; Vadalà, M. Letter to the Editor: Corynebacterium Parvum(Propionibacterium Acnes): Cytokines Cells, Innate Immunity, and Putative Antiviral Adoption. J. Interferon Cytokine Res. 2021, 41, 132–136