ATYPICAL GIANOTTI-CROSTI SYNDROME ASSOCIATED WITH EPSTEIN-BARR VIRUS IN A 3-MONTH-OLD INFANT: A CASE REPORT AND SYSTEMATIC LITERATURE REVIEW

Mehmet Yeral, Ekin Altinbas, Emine Tugba Alatas, Ceyda Tetik Aydogdu, Suzan Demir Pektas
Mugla Training and Research Hospital, Mugla, Turkey

Keywords Gianotti-Crosti syndrome, acrodermatitis, hepatitis B virus, Epstein-Barr virus, infant.

Abbreviations **EBV =** Epstein-Barr virus; **GCS** = Gianotti-Crosti syndrome.

Case report. A three-month-old male infant of Turkish descent presented with a 3-day history of a progressive, widespread skin eruption. One week prior, the patient had experienced mild rhinitis. Physical examination revealed numerous erythematous vesicles and bullae, ranging from 0.5 to 1.5 cm in diameter, clustered in the genitocrural region and extending to the trunk and distal extremities, with complete sparing of the face (Fig. 1). Despite the extensive eruption, the infant remained afebrile, hemodynamically stable, and continued to feed normally. No lymphadenopathy was observed.

Initial laboratory evaluation revealed mildly elevated C-reactive protein (5.69 mg/L; reference range < 5.00). Epstein-Barr virus viral capsid antigen (EBV VCA) IgM antibodies were positive (index 0.58; reference range < 0.11), while both EBV VCA IgG and EBV nuclear antigen (EBNA) IgG were negative, a pattern consistent with acute primary EBV infection. Serologic tests for CMV, HSV-1/2, HBV, HCV, and HIV were all negative. Complete blood count was within normal limits, and liver function tests were normal. The clinical and laboratory data led to the diagnosis of Gianotti-Crosti syndrome.

The patient was managed conservatively with the application of emollients and parental reassurance. At the one-month follow-up, all lesions had completely resolved without scarring. Follow-up serology confirmed definitive seroconversion: EBV VCA IgM had turned negative, whereas both EBV VCA IgG and EBNA IgG were positive.

Materials and methods. A systematic review of the literature was conducted in accordance with the PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to identify published reports of EBV-associated Gianotti-Crosti syndrome (GCS). Electronic literature searches were performed in PubMed, Scopus, Web of Science, and the Cochrane Library, including publications from January 1983 to March 2026. Due to a lack of institutional access to Embase, systematic searching of this database was not possible. The PubMed search strategy utilized the following Boolean syntax: ("Gianotti-Crosti" OR "papular acrodermatitis") AND ("Epstein-Barr" OR "EBV"). Equivalent search concepts, adapted to the specific indexing of each database, were applied in Scopus and Web of Science.

Eligible studies were required to report patients with a clinical diagnosis of GCS confirmed by laboratory evidence consistent with primary EBV infection. Acceptable diagnostic confirmation included serological positivity for EBV VCA IgM, documented seroconversion of VCA IgG and/or EBNA IgG, or detection of EBV-DNA by PCR. Studies were excluded if they described cases attributable to alternative infectious etiologies, lacked sufficient patient-level clinical details, represented duplicate reports, were published in languages other than English, or were unavailable in full text. Narrative reviews, editorials, and commentaries without original case data were also excluded.

Manual backward citation tracking of the reference lists of eligible publications was performed to identify studies missed by database searches. For each included case, the following variables were

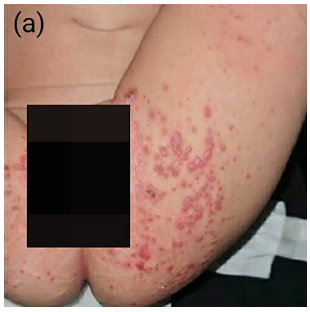

Fig. 1a

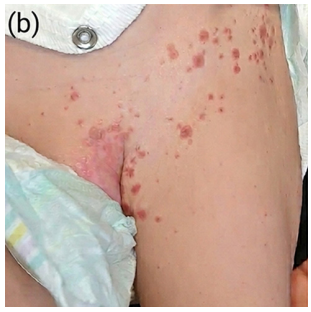

Fig. 1b

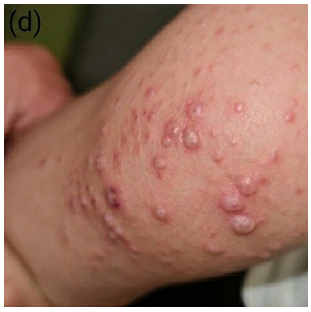

Fig. 1c

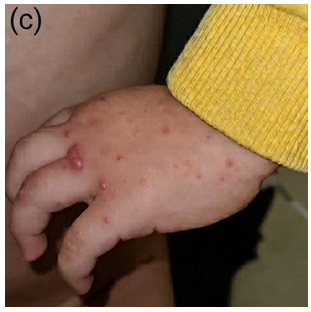

Fig. 1d

Figs. 1a, b, c, d: Bullous Gianotti-Crosti syndrome in a 3-month-old infant.

extracted when available: patient age, sex, lesion morphology, anatomical distribution, EBV serological findings, extracutaneous manifestations, hepatic involvement, treatment, and clinical outcome. The present index case was incorporated into the overall descriptive analysis. Statistical analysis was performed using Microsoft Excel, with continuous variables summarized as means and ranges, and categorical variables presented as frequencies and percentages.

Results. *Study selection*. The literature search and study selection process are summarized in the PRISMA flow diagram. A total of 109 articles were identified through database searches: 45 from PubMed, 36 from Scopus, 28 from Web of Science, and 0 from the Cochrane Library. Following duplicate removal and application of the predefined eligibility criteria, 20 studies from the database search met the inclusion criteria. Manual citation tracking identified four additional eligible publications, bringing the literature-derived dataset to 24 studies. With the inclusion of the present index case, the final synthesis comprised 25 sources, representing a cumulative cohort of 45 laboratory-confirmed cases of EBV-associated GCS (see Supplementary Table S1 for a full case-by-case summary).

Demographic characteristics. Among the 42 patients with available age data, the mean age was approximately 3.5 years (range: 1.5 months to 26 years). The majority (81%, n = 34/42) were between 1 and 6 years of age; 93% (n = 39/42) were 12 years of age or younger. Infants under one year of age represented 10% (n = 4/42), while adult cases accounted for 7% (n = 3/42). Among the 42 patients with available sex data, 24 were male and 18 were female. The youngest patient reported was the 1.5-month-old infant described by Sarma et al. (15), whereas the oldest was the 26-year-old adult reported by Mempel et al. (22).

Clinical characteristics. The majority of included patients presented with monomorphic papular or papulovesicular eruptions. Vesiculobullous morphology was identified in only two cases: the present patient and the case reported by Marcassi et al. (10). Purpuric or hemorrhagic variants were described by Sarma et al. (15), Lowe et al. (26), and Kim et al. (29). All cases demonstrated acral involvement. Trunk extension was documented in approximately 35% of included cases, including the present patient and those in the studies by Manduca et al. (6), Afonso et al. (8), Marcassi et al. (10), Sears et al. (14), Lowe et al. (26), and selected patients by Hofmann et al. and Taïeb et al. (21, 28). Exclusive facial involvement was described by Yoshida et al. (17).

Extracutaneous and laboratory findings. Lymphadenopathy was the most frequently reported extracutaneous finding, observed in 52% (n = 25) of cases, while hepatosplenomegaly was documented in 19% (n = 9). Elevated liver enzymes or biochemical evidence of hepatic involvement were identified in 56% (n = 27) of patients. The present patient demonstrated EBV VCA IgM positivity during the acute phase, followed by seroconversion confirming primary infection; no biochemical liver abnormalities were identified.

Table S1. Systematic compendium of laboratory-confirmed EBV-associated Gianotti-Crosti syndrome cases (n = 25 sources; 45 cases; ordered chronologically from most recent to oldest). The present index case is listed first. Treatment not specifically reported in source publications is listed as "Conservative (symptomatic)" in keeping with the universally self-limiting natural history of GCS. Intermediate screening numbers in the PRISMA diagram should be verified against the author's screening log.

Study (author, year)	*Age*	*Sex*	*Lesion morphology*	*Anatomical distribution*	*EBV serology*	*Extracutaneous findings*	*Treatment*	*Clinical outcome/ follow-up*
Present case: Yeral, 2026	3 months	M	Vesiculobullous; bullae 0.5-1.5 cm	Genitocrural, trunk, extremities; facial sparing	VCA IgM+ —> seroconversion (VCA IgG+, EBNA IgG+) at 1 month	None; normal LFTs	Emollient; parental reassurance	Complete resolution; no scarring; 1 month
Manduca et al., 2026	13 years	M	Papular, verrucous	Face, extremities, trunk	VCA IgM+	Lymphadenopathy; vasculitis on histology	Not specifically reported	Resolution
Chin & Liy-Wong, 2023	3 years	F	Papular	Bilateral cheeks, extremities	VCA IgM+, VCA IgG+	None	Not specifically reported	Chronic course >20 months; eventual resolution
Afonso et al., 2021	13 years	M	Papular	Face, forearms, legs, trunk	VCA IgM+, VCA IgG+, EBNA IgG+	Hepatosplenomegaly; elevated LFTs	Conservative (symptomatic)	Resolution
Ciccarese et al., 2020	26 years	M	Papular	Acrolocated (classical)	EBV IgM+	Lymphadenopathy; elevated LFTs	Conservative	Resolution
Marcassi et al., 2018	Not stated	F	Vesiculobullous	Acrolocated + truncal	VCA IgM+	Not reported	Conservative	Resolution
Bassi et al., 2016	2.5 years	F	Papular	Face, extremities	VCA IgM+	Lymphadenopathy; erythema nodosum	Conservative	Resolution
Al Dhaheri et al., 2016	14 months	M	Papular	Acrolocated	VCA IgM+	Lymphadenopathy	Conservative	Resolution
Tekin et al., 2014	4 months	M	Papular	Acrolocated	VCA IgM+ —> seroconversion	None	Conservative	Resolution
Sears et al., 2014	12 months	M	Papular	Face, extremities, trunk	VCA IgM+	Lymphadenopathy	Conservative	Resolution
Sarma et al., 2013	1.5 months	M	Haemorrhagic papular	Acrolocated	EBV IgM+	None	Conservative	Resolution
Gumuş et al., 2008	2 years	M	Papular	Face, extremities (classical)	VCA IgM+	Lymphadenopathy	Conservative	Resolution
Yoshida et al., 2004 n = 5	6m, 1y, 5y, 6y, 10y	4M, 1F	Papular (all)	Facial/acral; localised facial in some	VCA IgM+ (all 5)	Lymphadenopathy (4/5); elevated LFTs (3/5)	Conservative (all)	Spontaneous resolution (all)

Terasaki et al., 2003	3 years	M	Papular	Acrolocated	EBV DNA+ (PCR); EBV reactivation	Lymphadenopathy; elevated LFTs	Conservative	Resolution
Smith & Skelton, 2000, n = 3	15m, 4y, 5y	3M	Papular (all)	Acrolocated (all)	VCA IgM+ (all 3)	Lymphadenopathy (1/3); normal LFTs	Conservative (all)	Spontaneous resolution (all)
Maeda et al., 1999	3 years	M	Papular	Acrolocated	VCA IgM+ —> seroconversion	Lymphadenopathy; Bell's palsy	Conservative	Cutaneous and Bell's palsy resolved
Hofmann et al., 1997 n = 5	1.5-5 years	3M, 2F	Papular (all)	Acrolocated (all)	VCA IgM+ (all 5)	Lymphadenopathy (3/5); elevated LFTs (4/5)	Conservative (all)	Spontaneous resolution (all)
Mempel et al., 1996	26 years	M	Papular	Acrolocated (classical)	VCA IgM+	Lymphadenopathy; splenomegaly; elevated LFTs	Conservative	Resolution
Lacour & Harms, 1995	8 months	F	Papular	Face, extremities	VCA IgM+ (post-vaccination)	Fever; hepatosplenomegaly; elevated LFTs	Conservative	Resolution
Baldari et al., 1994 n = 5	~1 year ×5	N/R	Papular, epidemic	Acrolocated (all)	EBV serology+ (all 5)	Hepatomegaly (3/5); elevated LFTs (4/5)	Conservative (all)	Spontaneous resolution (all)
Essink et al., 1994	18 months	M	Papular	Acrolocated	VCA IgM+	Lymphadenopathy; elevated LFTs	Conservative	Resolution within 8 weeks
Lowe et al., 1989	8 months	M	Haemorrhagic papular	Face, extremities, trunk	VCA IgM+	None; normal LFTs	Conservative	Resolution
Schmidt et al. (1986)	10 months	N/R	Papulovesicular	Acrolocated	VCA IgM+	Elevated LFTs	Conservative	Resolution
Taieb et al., 1986, n = 5	2-6 years	N/R	Papular (all)	Acrolocated (all)	EBV serology+ (all 5)	Lymphadenopathy (all 5); elevated LFTs (5/5)	Conservative (all)	Spontaneous resolution (all)
Kim et al., 1983	4 months	M	Haemorrhagic papular	Acrolocated	VCA IgM+	None	Conservative	Resolution
Harpey & Lenoir, 1983	5 months	N/R	Papular	Acrolocated	VCA IgM+	None	Conservative	Resolution
Iosub et al., 1984	6 months	N/R	Papular	Acrolocated	VCA IgM+	None	Conservative	Resolution

Abbreviations: EBNA = Epstein-Barr Nuclear Antigen; EBV = Epstein-Barr Virus; F = Female; GCS = Gianotti-Crosti Syndrome; LFTs = Liver Function Tests; M = Male; N/R = Not Reported; PCR = Polymerase Chain Reaction; VCA = Viral Capsid Antigen.

Discussion. *Age spectrum and immunological considerations*. Gianotti-Crosti syndrome (GCS) is classically recognized as a pediatric exanthem, with peak incidence occurring between 1 and 6 years of age (1, 3). This updated overall analysis supports this established epidemiological pattern; however, the occurrence of cases outside the conventional age distribution indicates that EBV-associated GCS should not be regarded as strictly confined to this demographic group. Primary EBV infection during early infancy is generally considered infrequent or clinically attenuated, largely due to maternally transferred antibodies (5, 23). The appearance of EBV-associated GCS in very young infants is therefore clinically remarkable. The youngest patient reported was the 1.5-month-old infant described by Sarma et al. (15), while additional infantile cases described by Lowe et al. (26), Harpey et al. (30), Iosub et al. (31), and Tekin et al. (13) further demonstrate that clinically overt, EBV-triggered cutaneous disease can occur despite passive immune protection in early life.

A particularly noteworthy feature of the present case is the coexistence of an extensive vesiculobullous morphology with complete spontaneous resolution within one month. This presentation raises the possibility that immune responses to primary EBV infection in early infancy may differ qualitatively from those observed in older children: immunological immaturity might alter the balance between antiviral immune regulation and hypersensitivity-mediated cutaneous inflammation, permitting marked skin manifestations alongside relatively rapid resolution. Given the limited number of infantile cases in the literature, this interpretation remains speculative. Adult-onset presentations in the reviewed cohort, including cases by Ciccarese et al. (9) and Mempel et al. (22), suggest that delayed primary EBV infection may occasionally present with a clinicomorphological picture consistent with GCS.

Clinicomorphological spectrum and diagnostic implications. GCS is traditionally described as a self-limiting eruption of monomorphic erythematous papules or papulovesicles symmetrically distributed over acral regions (1). Although this pattern remains predominant in the reviewed cohort, the body of evidence indicates that EBV-associated disease can exhibit a broader clinicomorphological spectrum. Vesiculobullous morphology appears to be particularly rare, with only the current case and that described by Marcassi et al. (10) clearly fitting this pattern. Similarly, the hemorrhagic or purpuric variants described by Sarma et al. (15), Lowe et al. (26), and Kim et al. (29) indicate that intense inflammatory cutaneous responses can occur within the recognized disease spectrum.

Anatomical distribution also demonstrated variability. Although acral involvement remained a defining feature in all included cases, trunk extension was documented in multiple reports (6, 8, 10, 14, 26, 28). Rigid adherence to classic diagnostic expectations—such as obligatory sparing of the trunk—may therefore lead to non-recognition of atypical EBV-associated cases. In infants and children, bullous, hemorrhagic, or widespread eruptions may initially raise suspicion for autoimmune bullous diseases, infectious vesiculobullous eruptions, or drug reactions. However, when the eruption displays a compatible, symmetrical acral distribution accompanied by supportive EBV serology, EBV-associated GCS should be considered.

Systemic findings and clinical relevance. Although GCS is frequently approached primarily as a cutaneous eruption, the reviewed cases indicate that EBV-associated disease often occurs in the context of broader systemic viral involvement. Hepatic involvement emerged as one of the most clinically relevant systemic findings: elevated liver enzymes were identified in 56% of evaluated cases, indicating that hepatic participation is a relatively frequent feature of EBV-associated GCS, despite its complete absence in the present patient. This variability suggests that hepatic involvement may support the diagnosis when present, but should not be regarded as a mandatory diagnostic criterion. Baseline assessment of liver function may be a reasonable consideration in suspected cases of EBV-associated GCS, particularly when patients present with widespread cutaneous disease, systemic symptoms, or hepatosplenomegaly. Taken together, these findings support the interpretation of EBV-associated GCS as a systemic viral reaction with prominent cutaneous expression, rather than an eruption strictly limited to the skin.

Study limitations. This study has several limitations. The rarity of laboratory-confirmed EBV-associated GCS entails an inherent risk of publication bias, as unusual or diagnostically challenging presentations are more likely to be reported than typical, self-limiting cases. The available literature consisted predominantly of case reports and small case series, leading to heterogeneity in reporting quality and diagnostic workup. Although multiple major databases were systematically searched, Embase was not comprehensively evaluated due to a lack of institutional access, and the restriction to English-language publications may have excluded eligible reports in other languages. This systematic review was not prospectively registered on PROSPERO; given the predominantly case-based nature of the available literature, prospective protocol registration was deemed not applicable, and the methodology was instead defined a priori and rigorously applied throughout the selection process. Finally, given the retrospective and descriptive nature of single-case literature, a formal meta-analytic synthesis was not feasible; the results must therefore be interpreted as a descriptive characterization of the currently reported spectrum.

Conclusions. This case and the updated systematic review expand the recognized clinical spectrum of Epstein-Barr virus-associated Gianotti-Crosti syndrome, demonstrating that the condition can occur in early infancy and present with atypical features, including vesiculobullous morphology and trunk involvement. Atypical presentations should not rule out the diagnosis, particularly when the eruption displays a compatible, symmetrical acral distribution alongside supportive EBV serological findings. Recognition of these variants can help avoid unnecessary diagnostic workups and support appropriate conservative management.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Address to:
Dr. Ekin Altinbas
Marmaris Street no 48 Mugla Turkey 48000
e-mail: altinbasekin@gmail.com

References

1) Brandt O, Abeck D, Gianotti R, Burgdorf W. Gianotti-Crosti syndrome. *J Am Acad Dermatol*. 2006;54(1):136-45. doi 10.1016/j.jaad.2005.09.033
2) Crosti A. *G Ital Dermatol Minerva Dermatol* 1966;107(5):399-412. Italian. PMID 4869128
3) Leung AKC, Sergi CM, Lam JM, Leong KF. Gianotti-Crosti syndrome (papular acrodermatitis of childhood) in the era of a viral recrudescence and vaccine opposition. *World J Pediatr*. 2019;15(6):521-7. doi 10.1007/s12519-019-00269-9
4) Chuh A, Zawar V, Sciallis GF, et al. Pityriasis rosea, Gianotti-Crosti syndrome, asymmetrical periflexural exanthem, popular purpuric gloves and socks syndrome, eruptive pseudoangiomatosis, and eruptive hypomelanosis: do their epidemiological data substantiate infectious etiologies. *Infect Dis Rep*. 2016;8(1):6418. doi 10.4081/idr.2016.6418
5) Cohen JI. Epstein-Barr virus infection. *N Engl J Med*. 2000;343(7):481-92. doi 10.1056/NEJM200008173430707
6) Manduca S, Truong C, Flamm A, Orlow SJ. Cutaneous manifestations of Epstein-Barr virus: a unique case of Gianotti-Crosti syndrome. *J Cutan Pathol*. 2026;53(7):581-5. doi 10.1111/cup.70080
7) Chin LD, Liy-Wong C. A rare case of chronic Gianotti-Crosti syndrome. *SAGE Open Med Case Rep*. 2023;11:2050313X231164250. doi 10.1177/2050313X231164250
8) Afonso A, Cachao J, Pinto Junior VL, Gouveia T. Gianotti-Crosti syndrome: a challenging exanthema. *BMJ Case Rep*. 2021;14(4):e240747. doi 10.1136/bcr-2020-240747

9) Ciccarese G, Trave I, Herzum A, et al. Dermatological manifestations of Epstein-Barr virus systemic infection: a case report and literature review. *Int J Dermatol*. 2020;59(10):1202-9. doi 10.1111/ijd.14887
10) Marcassi AP, de Donato Piazza CA, de Meio Paiva Seize MB, da Costa Pereira Cestari S. Atypical Gianotti-Crosti syndrome. *An Bras Dermatol*. 2018;93(2):265-7. doi 10.1590/abd1806-4841.20186726
11) Bassi A, Venturini E, Montagnani C, et al. Gianotti-Crosti syndrome and erythema nodosum. *J Pediatr*. 2016;172:217. doi 10.1016/j.jpeds.2016.01.050
12) Al Dhaheri HS, Al Kaabi A, Kara Hamo Y, et al. Gianotti Crosti syndrome following Epstein-Barr virus infection. *Case Rep Dermatol Med*. 2016;2016:1017524. doi 10.1155/2016/1017524
13) Tekin M, Halmis H, Ozarsian K, Gulyuz A. Gianotti-Crosti syndrome following Epstein-Barr virus in-fection: a case report. *J Pediatr Res*. 2014;1(4):236-8. doi 10.4274/jpr.25744
14) Sears W, Hodge B, Jones B, et al. Visual diagnosis: 12-month-old boy with persistent rash and lymphadenopathy. *Pediatr Rev*. 2014;35(10):452-5. doi 10.1542/pir.35-10-452
15) Sarma N, Sarkar UK, Das MK, Das S. Hemorrhagic Gianotti-Crosti syndrome in a one and half month old infant: an extremely unusual presentation. *Indian J Dermatol*. 2013;58(1):65-7. doi 10.4103/0019-5154.105313
16) Gumus P, Teksam O, Akinci H, et al. Gianotti-Crosti syndrome as the only manifestation of primary Epstein-Barr virus infection: a case report. *Turk J Pediatr*. 2008;50(3):302-4. PMID: 18773682
17) Yoshida M, Tsuda N, Morihata T, et al. Five patients with localized facial eruptions associated with Gianotti-Crosti syndrome caused by primary Epstein-Barr virus infection. *J Pediatr*. 2004;145(6):843-4. doi 10.1016/j.jpeds.2004.08.006
18) Terasaki K, Koura S, Tachikura T, Kanzaki T. Gianotti-Crosti syndrome associated with endogenous reactivation of Epstein-Barr virus. *Dermatology*. 2003;207(1):68-71. doi 10.1159/000070947
19) Smith KJ, Skelton H. Histopathologic features in Gianotti-Crosti syndrome secondary to Epstein-Barr virus. *J Am Acad Dermatol*. 2000;43(6):1076-9. doi 10.1067/mjd.2000.109289
20) Maeda S, Tsuda H, Haruki S, Mitsuto I. Atypical Epstein-Barr virus infection associated with Gianotti-Crosti syndrome and Bell's palsy. *Pediatr Int*. 1999;41(3):315-7. doi 10.1046/j.1442-200x.1999.01054.x
21) Hofmann B, Schuppe HC, Adams O, et al. Gianotti-Crosti syndrome associated with Epstein-Barr virus infection. *Pediatr Dermatol*. 1997;14(4):273-7. doi 10.1111/j.1525-1470.1997.tb00955.x
22) Mempel M, Abeck D, Bye-Hansen T, Ring J. Gianotti-Crosti syndrome in an adult following Epstein-Barr virus infection. *Acta Derm Venereol*. 1996;76(6):502-3. doi 10.2340/0001555576502503
23) Lacour M, Harms M. Gianotti-Crosti syndrome as a result of vaccination and Epstein-Barr virus infection. *Eur J Pediatr*. 1995;154(8):688-9. doi 10.1007/BF02079084
24) Baldari U, Monti A, Righini MG. An epidemic of infantile papular acrodermatitis due to Epstein-Barr virus. *Dermatology*. 1994;188(3):203-4. doi 10.1159/000247139
25) Essink AH, Winterberg DH, Hulsebosch HJ. Viral papular acrodermatitis: Gianotti-Crosti syndrome caused by Epstein-Barr virus infection in a child. *Ned Tijdschr Geneeskd*. 1994;138(35):1775-7. Dutch. PMID 7935898
26) Lowe L, Hebert AA, Duvic M. Gianotti-Crosti syndrome associated with Epstein-Barr virus infection. *J Am Acad Dermatol*. 1989;20(2 Pt 2):336-8. doi 10.1016/s0190-9622(89)70041-4
27) Schmidt KU, Hoting E, Mensing H, Nasemann T. Infantile acro-localized papulovesicular syndrome. *Dtsch Med Wochenschr*. 1986;111(39):1482-4. *German*. doi 10.1055/s-2008-1068656
28) Taieb A, Plantin P, Du Pasquier P, et al. Gianotti-Crosti syndrome: a study of 26 cases. *Br J Dermatol*. 1986;115(1):49-59. doi 10.1111/j.1365-2133.1986.tb06219.x
29) Kim YJ, Way BH, Garcia RR. Gianotti-Crosti syndrome in an infant with Epstein-Barr virus infection. *Pediatr Infect Dis*. 1983;2(3):239-43. doi 10.1097/00006454-198305000-00015
30) Harpey JP, Lenoir G. Epstein-Barr virus and infantile papular acrodermatitis. *J Pediatr*. 1983;102(1):167-8. doi 10.1016/s0022-3476(83)80336-9
31) Iosub S, Santos C, Gromisch DS. Papular acrodermatitis with Epstein-Barr virus infection. Clin Pediatr (Phila). 1984;23(1):33-4. doi 10.1177/000992288402300106