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Long-term consequences of Herpesvirus infection and chronic inflammation of connective tissue

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Narrative: Cyst-like formations in various tissues of the body may reflect a common mechanism of their development: uniform pressure of the contents (blood, cerebrospinal fluid, synovial fluid, glandular secretions, etc.) under conditions of connective tissue weakness leads to the formation of spherical structures. The association between these viruses and connective tissue may serve as a unifying factor linking diverse clinical conditions and structural abnormalities.

Given the widespread distribution of connective tissue and its derivatives in the body, structural abnormalities potentially associated with herpesvirus infection may be expected to occur almost ubiquitously. The author has not identified references to this in the available literature. Further studies aimed at a more detailed investigation of this phenomenon may allow prediction of cyst development and assessment of connective tissue status, and in many cases may support a shift from surgical treatment toward more conservative and less invasive therapeutic approaches.

Indexing Terms: Herpesvirus; cyst; fibrosis; viral burden; immune-mediated inflammation; connective tissue; Tarlov cyst.

Introduction

Spinal and joint disorders (including intervertebral disc herniation, coxarthrosis, cystic changes, joint instability, and chronic pain syndromes) are traditionally regarded as consequences of mechanical overload and degenerative processes, without sufficient consideration of muscle function and other contributing factors.

'... The clinical diversity of manifestations associated with chronic consequences of exposure to these viruses suggests a profound level of viral impact at the level of fibroblasts and during the formation of different tissues ...'

However, clinical practice demonstrates that surgical and manual interventions do not lead to sustained improvement in all patients. A proportion of patients return after surgical treatment with recurrent symptoms and persistent complaints, despite technically successful procedures.

This suggests that there is still an incomplete understanding of the causes,

contributing factors, and underlying mechanisms involved in the development of chronic inflammation and fibrosis.

One of such factors may be persistent herpesvirus infection (including cytomegalovirus [CMV], Epstein–Barr virus [EBV], varicella-zoster virus [VZV], human herpesvirus 6 [HHV-6], herpes simplex virus [HSV], among others), which can maintain immune response imbalance even in the absence of active viral replication and exert a destructive effect on connective tissue.

Observations

The authors are the first to suggest a potential role of CMV and EBV in the development of long-term destructive changes in connective tissue. Using the example of facet joint cysts, a conceptual framework for the aetiology and mechanisms of cyst-like formations is proposed, along with therapeutic approaches for their correction.

Based on empirical observations, the authors noted the presence of IgG antibodies to CMV and EBV in a group of patients presenting with cysts, hemangiomas, connective tissue weakness, lipomas, and related conditions. Diseases traditionally associated with CMV infection include lymphomas, inflammatory bowel diseases, nasopharyngeal carcinoma, gastric cancer, and multiple sclerosis.

To date, the authors have not identified reports in the available literature describing a relationship between the sequelae of this viral infection and cystic formations.

Epstein–Barr virus (EBV) (Fig. 1), cytomegalovirus (CMV) (Fig. 2), and varicella-zoster virus (VZV) are herpesviruses typically acquired in childhood that establish persistent, latent infection and may influence the development of the immune system.

EBV, in particular, is an extremely prevalent herpesvirus: by adulthood, more than 90% of individuals demonstrate serological evidence of prior infection (IgG antibodies), and in many populations, seroprevalence reaches 90–95%. Infection usually occurs during childhood or adolescence and results in lifelong latency.

EBV should therefore be regarded as a near-universal infectious agent in the adult population, with its clinical significance determined not by the mere presence of infection, but by the phase of viral activity and the host immune status. (1 - 3)

Fig 1: Epstein–Barr virus (EBV). Schematic representation.

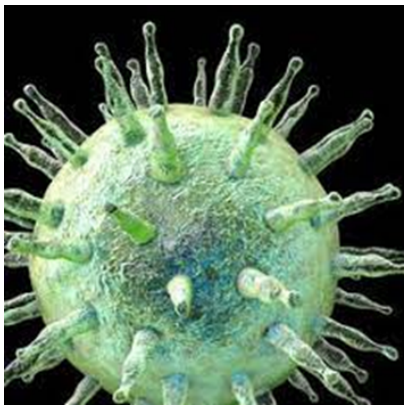
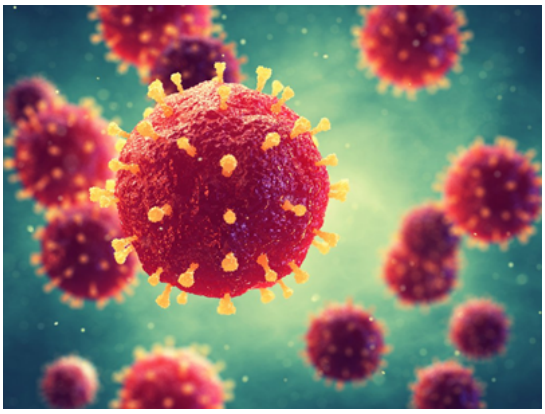


Fig 2: Cytomegalovirus (CMV). Schematic representation.



However, insufficient attention has been given to the latent and subclinical consequences of exposure to this group of viruses. Clinically significant lesions and conditions, such as hemangiomas, lipomas, cystic changes, and connective tissue weakness at various anatomical sites, are typically interpreted as degenerative processes. However, in our opinion, they may also have a causal relationship with herpesvirus infection.

Observations of patients with such changes suggest this association, supported both by laboratory findings and by the positive effects of targeted antihomotoxic therapy. Note: Homotoxicology is a branch of biological (regulatory) medicine developed in the 1950s by the German physician Hans-Heinrich Reckeweg. It is based on the concept that disease represents a manifestation of the body's response to homotoxins, endogenous and exogenous toxins, inflammatory mediators, infectious agents, and metabolic byproducts. The primary aim of therapy is not to suppress symptoms, but to support the body's self-regulatory capacity, enhance detoxification and regenerative mechanisms, and facilitate the restoration of functional equilibrium.

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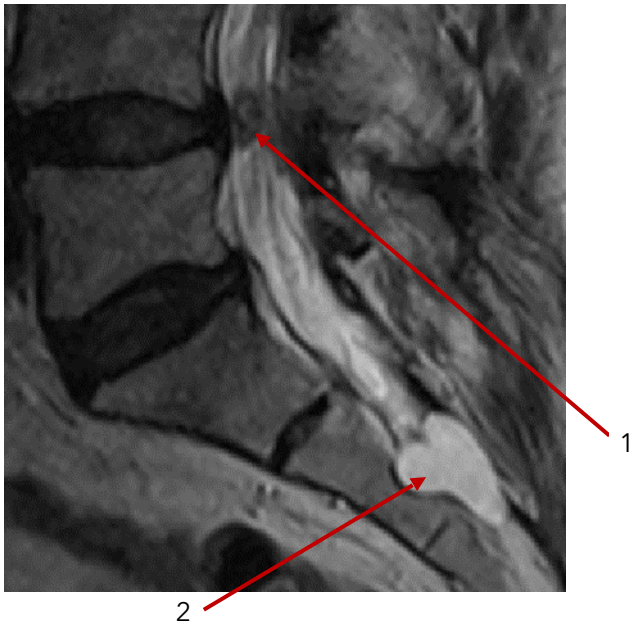
Synovial and facet joint cysts of the spine are generally regarded as degenerative formations. In some cases, histological examination reveals pseudocystic degenerative changes of the ligamentum flavum, accompanied by characteristic microcalcifications and a foreign body-type giant cell reaction. According to the literature, these cystic formations are most often not associated with an infectious process and are attributed to degenerative changes and increased segmental mobility of the spine. (4 - 8)

Tarlov cysts

Perineural cysts (Tarlov cysts) are described in clinical and morphological literature as meningeal dilatations of the spinal nerve root sheaths filled with cerebrospinal fluid (CSF). The cyst wall is typically composed of fibrous tissue with elements of the leptomeningeal (arachnoid) membrane, often including nerve fibres within its structure (Fig. 3).

Fig 3: Magnetic resonance imaging (MRI):
1 – facet joint cyst; 2 – Tarlov cyst



Their origin has been associated with congenital factors, disturbances of cerebrospinal fluid (CSF) dynamics, a valve-like mechanism, or post-traumatic changes. Although their aetiology remains incompletely understood, unlike a number of other pathological processes, perineural cysts do not demonstrate features of infectious or viral involvement. (9 - 13)

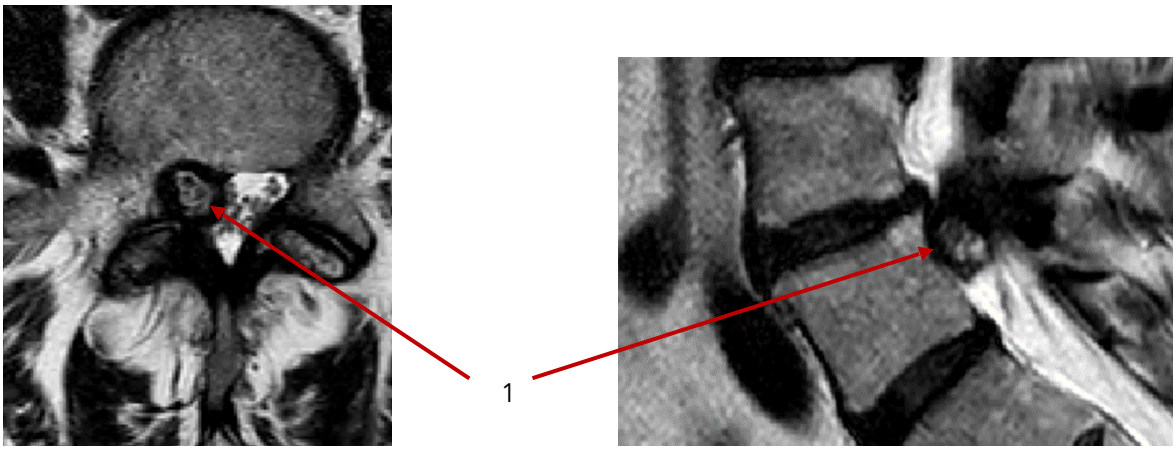
A rare complication following microdiscectomy includes discal, annular, and hemorrhagic cysts of the facet joints (Fig. 3, 1), as well as fibrocystic cavities lacking a true synovial lining. (14 - 20) Morphological examination has not revealed specific cytomegalovirus inclusion bodies or viral cytopathic effects.

Synovial cysts are abnormal fluid-filled sacs within the spinal joints and are relatively common in patients with spinal spondylosis. These cysts are benign and resemble cystic formations in other anatomical locations, such as wrist ganglion cysts. They are generally considered to develop as a result of age-related degenerative changes and are most frequently observed in patients over 65 years of age. Although they may occur throughout the spine, they are most commonly found in the lumbar region. For spine surgeons, the presence of a facet joint cyst is indicative of segmental instability at the corresponding spinal level. (41 - 45)

Various conservative treatment approaches for these cysts have been described; however, in cases of chronic pain or neurological deficit, surgical intervention is required. (46, 47)

Clinical manifestations in patients with synovial cysts requiring surgical treatment are typically associated with nerve root compression and spinal instability at the level of the cysts (Figs. 4, 5).

Figs 4, 5: Magnetic resonance imaging (MRI):
(1) facet joint synovial cyst causing nerve compression and requiring surgical removal



In addition to synovial cysts of the facet joints, patients in this group also presented with hemangiomas (Fig. 6), cystic lesions of internal organs (Figs. 7, 8), cystic formations in other joints (Fig. 9), as well as aneurysms.

Elevated IgG levels against CMV, HSV, and HHV-6 indicate prior infection, i.e., viral carriage. This is typical for the majority of adult patients and does not, in itself, establish a causal relationship with cyst formation or disc herniation.

Fig 6: Magnetic resonance imaging (MRI): (1) hemangioma of a thoracic vertebra

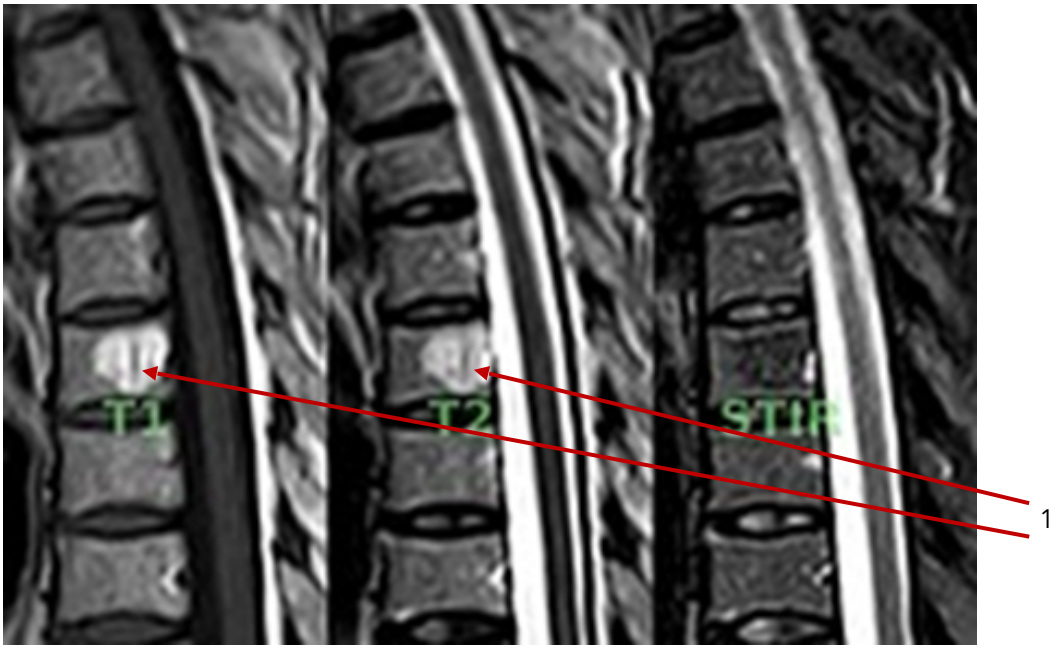


Fig 7: Ultrasound examination (US):(1) renal cyst

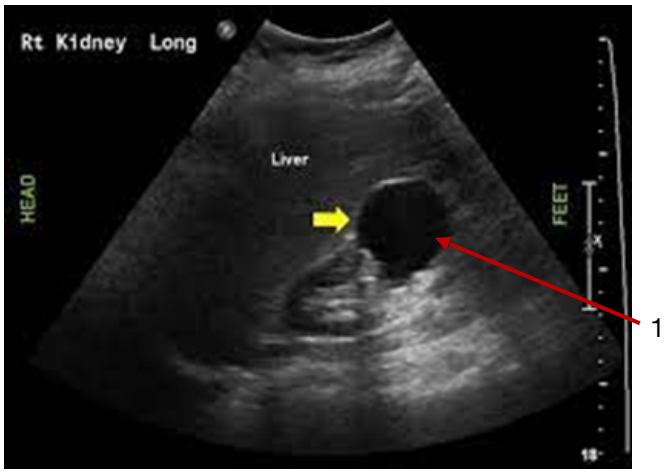


Fig 8: Ultrasound examination (US):(1) renal cyst

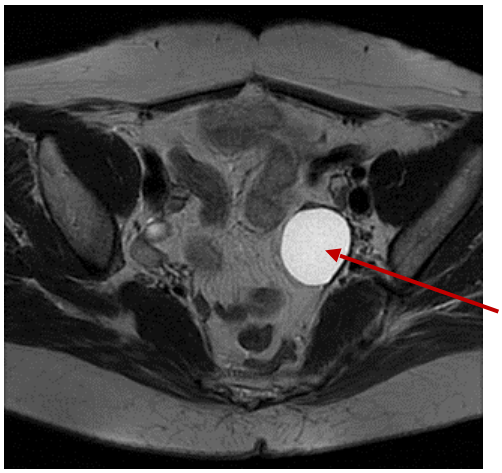
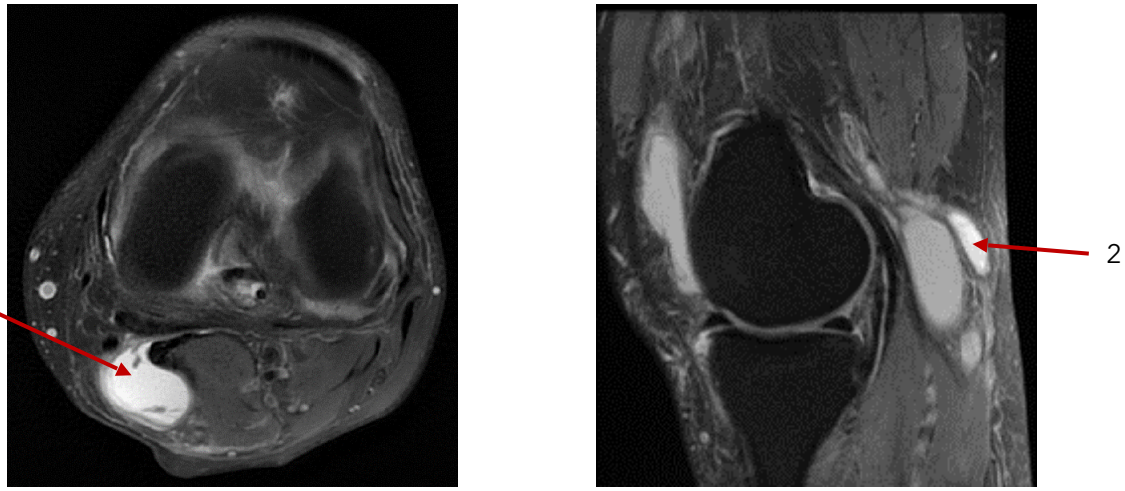


Fig 9: Magnetic resonance imaging (MRI): (1, 2) Baker's cyst



Demonstration of viral activity requires polymerase chain reaction (PCR) testing (in blood, cerebrospinal fluid, or affected tissue) in conjunction with clinical data; IgG avidity testing is used to determine the timing of CMV infection. (21 - 24) In our opinion, alterations of connective tissue manifested as volumetric, rounded formations, together with elevated IgG titers, may suggest the presence of such an association.

Following primary infection, herpesviruses (including CMV, HHV-6, and HSV) persist in the host for life. Under conditions of stress or immunosuppression, they may undergo reactivation. Even in the absence of detectable viral DNA in the analysed sample, herpesvirus persistence, particularly CMV, may be associated with prolonged immune activation. (25 - 29)

Table 1: Avidity changes in the course of infection

Infection stage	IgG avidity*	Interpretation
Early (acute) stage, first 2–4 weeks:	low	Recent infection
Subacute / transitional stage:	moderate	Infection occurred recently
Chronic / past stage:	high	Past infection / immunity
Reactivation (e.g., EBV, CMV):	may decrease	Possible reactivation
* IgG avidity refers to the overall strength of binding between IgG antibodies and their specific antigens, taking into account multiple binding sites. It is used to help determine the timing of an infection, as low avidity indicates a recent infection while high avidity suggests a past infection.		

Macrophages normally perform a protective function (M1 phenotype); however, under conditions of chronic viral stimulation, they may shift toward an M2-like phenotype. Such a shift is associated with reduced antiviral immune responses and increased production of profibrotic cytokines. TGF-β, IL-13, IL-17, and IL-10). Resulting in fibroblast activation and collagen accumulation. Note: Cytokines are low-molecular-weight signalling proteins that mediate intercellular communication within the immune system and regulate inflammation, immune responses, and tissue remodelling processes.

A stiffer and less elastic connective tissue is formed, clinically manifested by pain, stiffness, and a tendency toward recurrence following physical нагрузки or surgical interventions; scar tissue may also develop, including in tendons, as an example of a localised fibrotic process (e.g., Dupuytren's contracture). (30–40) The author observed softening of scar tissue following therapeutic correction with antihomotoxic agents.

Table 2: Key cytokines and their clinical significance

Cytokine	Source	Effect on connective tissue	Clinical significance
TGF-β1:	M2 macrophages, fibroblasts	Activation of myofibroblasts, ↑ collagen	Primary driver of fibrosis
IL-13, IL-4:	Th2 lymphocytes	Stimulate extracellular matrix production, reduce elasticity	Sclerosis of joint capsules, joints, and ligaments
IL-17A/F:	Th17 cells	Enhance chronic inflammation and remodeling	Transition from inflammation to fibrosis
TNF-α, IL-1β, IL-6:	M1 macrophages, monocytes	Support inflammation, activate fibroblasts	Pain, edema, chronic inflammation
CCL2 (MCP-1):	Macrophages, endothelium	Recruits monocytes, maintains inflammatory infiltrate	Chronic cellular infiltrate

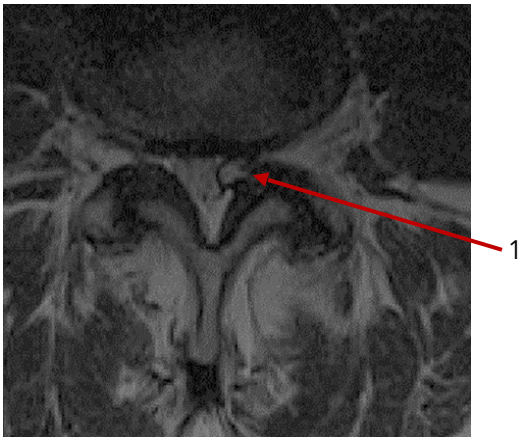
Clinical case 1

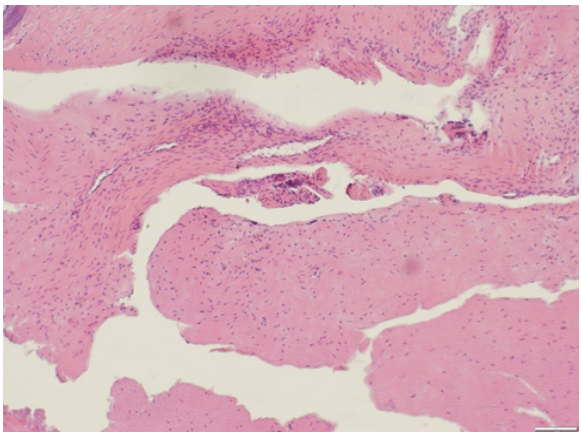
Patient B., 56 years old

Complaints of pain in the left thigh, aggravated by movement. The clinical presentation was consistent with left-sided L5 nerve root compression. Instrumental examination revealed a facet joint cyst (Figs. 10, 11). The patient also had a Tarlov cyst. Laboratory testing demonstrated elevated IgG antibodies to CMV and EBV (Fig. 12).

Surgical treatment was performed, consisting of removal of the facet joint cyst. In the postoperative period, resolution of pain in the left leg was observed.

Figs: 10, 11: Magnetic resonance imaging (MRI): (1)





Clinical case 2

Patient T., 71 years old

Complaints of severe lower back pain radiating to the right lower extremity, with sensory disturbance in the right foot. Instrumental examination revealed a facet joint cyst (Fig. 13).

Patient T. underwent conservative treatment using high-dilution integrated phytotherapeutic preparations produced by Alfa-Omega. Drainage, symptomatic, detoxification, and preparations containing information related to EBV and CMV infection were selected. The selection was based on manual muscle testing (MMT). (48)

As a result of the treatment, a reduction in cyst size and resolution of nerve compression were observed (Fig. 14); subjectively, there was complete resolution of pain and restoration of sensation.

Fig. 13. Magnetic resonance imaging (MRI): (1) facet joint cyst before treatment 12/06/2024

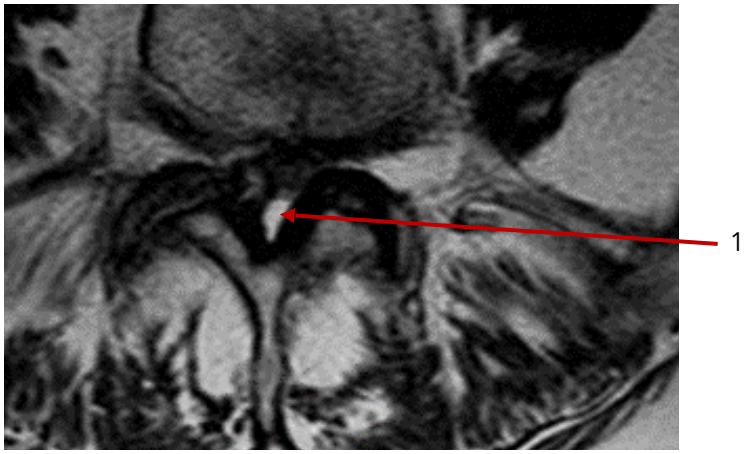


Fig. 14. Magnetic resonance imaging (MRI):(1) facet joint cyst after treatment, after 4 months, on 24/10/2024

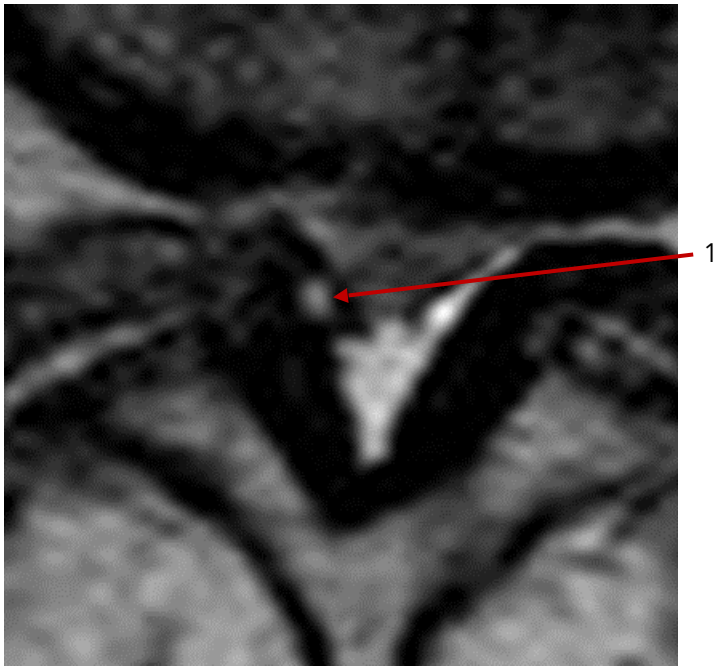
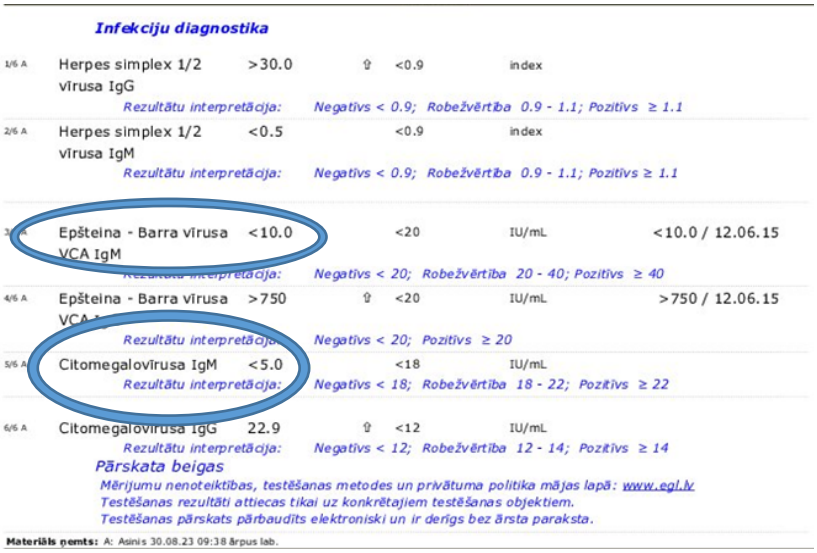


Fig.12: Laboratory findings of patient B.



Infekciju diagnostika

1/6 A	Herpes simplex 1/2 vīrusa IgG	>30.0	⇧	<0.9	index	
	Rezultātu interpretācija:	*Negatīvs < 0.9; Robežvērtība 0.9 - 1.1; Pozitīvs ≥ 1.1*				
2/6 A	Herpes simplex 1/2 vīrusa IgM	<0.5		<0.9	index	
	Rezultātu interpretācija:	*Negatīvs < 0.9; Robežvērtība 0.9 - 1.1; Pozitīvs ≥ 1.1*				
3/6 A	Epšteina - Barra vīrusa VCA IgM	<10.0		<20	IU/mL	<10.0 / 12.06.15
	Rezultātu interpretācija:	*Negatīvs < 20; Robežvērtība 20 - 40; Pozitīvs ≥ 40*				
4/6 A	Epšteina - Barra vīrusa VCA	>750	⇧	<20	IU/mL	>750 / 12.06.15
	Rezultātu interpretācija:	*Negatīvs < 20; Pozitīvs ≥ 20*				
5/6 A	Citomegalovīrusa IgM	<5.0		<18	IU/mL	
	Rezultātu interpretācija:	*Negatīvs < 18; Robežvērtība 18 - 22; Pozitīvs ≥ 22*				
6/6 A	Citomegalovīrusa IgG	22.9	⇧	<12	IU/mL	
	Rezultātu interpretācija:	*Negatīvs < 12; Robežvērtība 12 - 14; Pozitīvs ≥ 14*				

Pārskata beigas

Mērījumu nenoteiktības, testēšanas metodes un privātuma politika mājas lapā: www.egl.lv
Testēšanas rezultāti attiecas tikai uz konkrētajiem testēšanas objektiem.
Testēšanas pārskats pārbaudīts elektroniski un ir derīgs bez ārsta paraksta.

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Empirical observations

The use of complex antihomotoxic therapy preparations aimed at suppressing herpesvirus infection leads to a reduction or even disappearance of cysts (Fig. 15) and resolution of synovitis. In several cases, lipomatosis, cystic-fibrotic formations in the breast, and ovarian cysts were resolved.

A significant reduction of fibrosis in Dupuytren's contracture was also observed. Patients reported disappearance of tendon thickening in stenosing ligamentitis. Resolution of excess interstitial fluid and improvement of connective tissue weakness and hypermobility were also noted. Cases of arrest in the progression of bone cysts and dural cysts were documented.

Fig. 15. Magnetic resonance imaging (MRI):
(1) facet joint cyst before treatment; (2) the same facet joint cyst after treatment

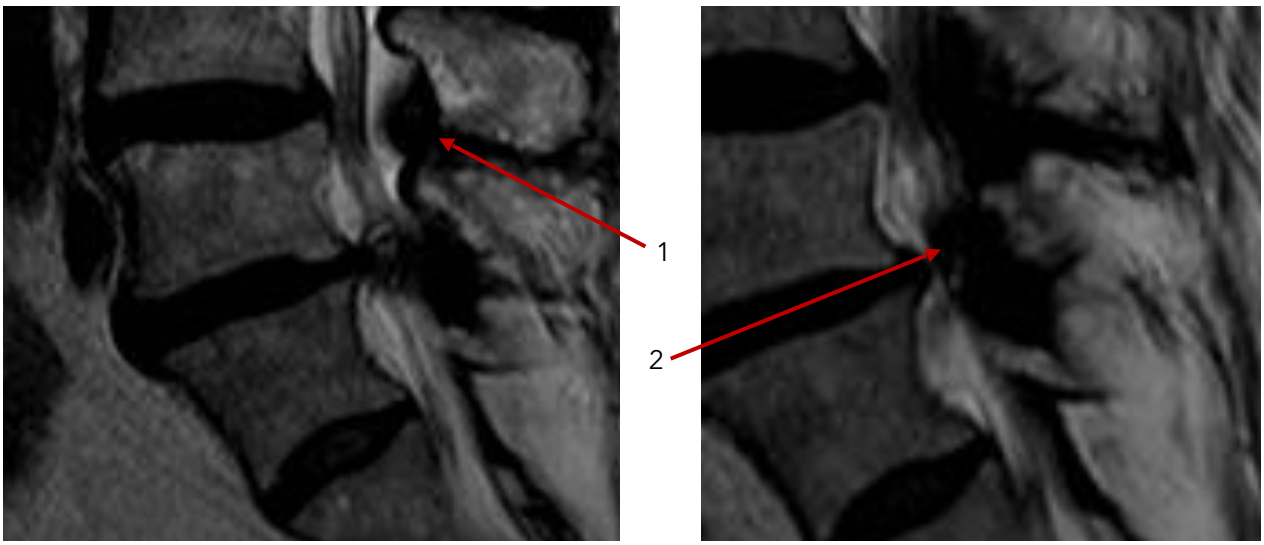
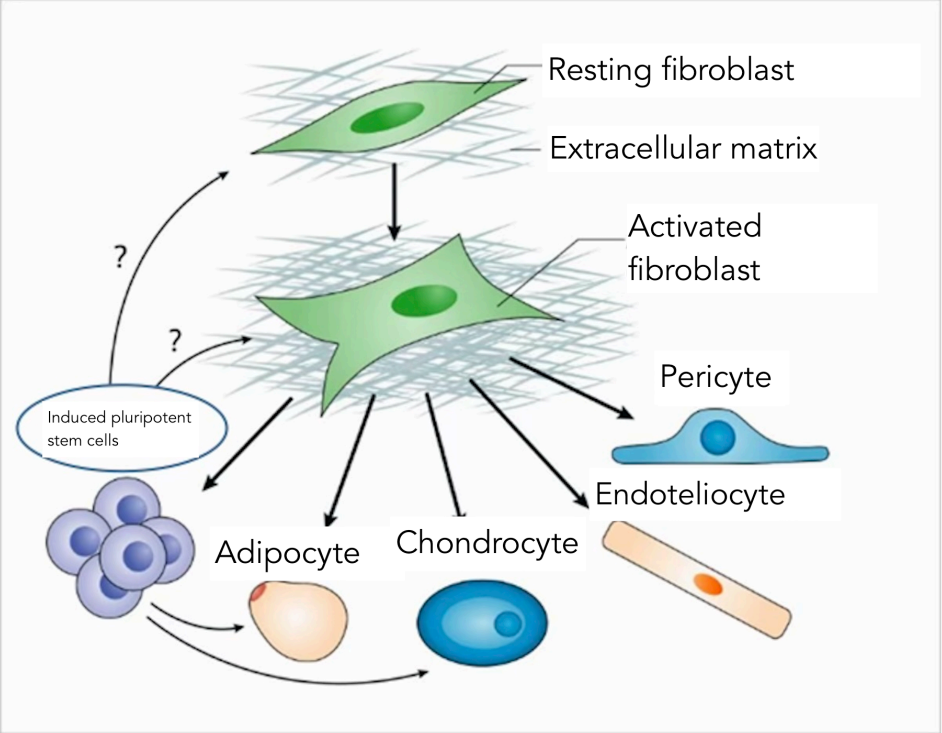


Figure 16 illustrates how fibroblasts, connective tissue cells, give rise to other specialised cells that form the structures of the body. If impairment of fibroblast function under the influence of herpesviruses is assumed to lead to alterations in multiple tissues, this provides a logical explanation for how CMV and EBV may initiate cystic formations in various anatomical locations.

The clinical diversity of manifestations associated with chronic consequences of exposure to these viruses suggests a profound level of viral impact at the level of fibroblasts and during the formation of different tissues.

Fig. 16. Differentiation of fibroblasts into other cell types of the body.



Cyst-like formations in various tissues of the body may reflect a common mechanism of their development: uniform pressure of the contents (blood, cerebrospinal fluid, synovial fluid, glandular secretions, etc.) under conditions of connective tissue weakness leads to the formation of spherical structures. The association between these viruses and connective tissue may serve as a unifying factor linking diverse clinical conditions and structural abnormalities.

Given the widespread distribution of connective tissue and its derivatives in the body, structural abnormalities potentially associated with herpesvirus infection may be expected to occur almost ubiquitously. The author has not identified references to this in the available literature. Further studies aimed at a more detailed investigation of this phenomenon may allow prediction of cyst development and assessment of connective tissue status, and in many cases may support a shift from surgical treatment toward more conservative and less invasive therapeutic approaches.

Conclusions

1. Morphologically, cysts have a degenerative-fibrotic nature; no direct evidence of herpesvirus cytopathic effects has been identified within them.
2. High seropositivity for herpesviruses (IgG) reflects prior infection and does not constitute evidence of an etiological relationship. This issue requires further investigation.

3. Elevated IgG titers are observed in all patients with cystic formations, indicating the need for further study of the chronic consequences of CMV and EBV infection.
4. Chronic viral burden maintains immune-mediated inflammation, promotes the shift of macrophages from the M1 to the M2 phenotype, and leads to excessive production of profibrotic cytokines. Consequences include chronic pain, stiffness, soft tissue fibrosis, risk of symptom recurrence after interventions, and cystic formations in various tissues.
5. As a rule, patients present with cystic formations in multiple tissues rather than localised involvement.
6. The presence of cystic formations indicates connective tissue weakness, which may be used as a prognostic marker in injury prevention in both athletes and other populations.
7. The use of specific phytotherapeutic, homeopathic, and antihomotoxic preparations (containing information about the nature of the pathogen) may eliminate or, at least, reduce the manifestations of chronic viral impact, thereby suggesting CMV and EBV as factors contributing to significant changes in the body
8. Herpesvirus carriage can only conditionally be considered asymptomatic. The presence of the virus in the body leads to continuous effects on connective tissue, with consequences affecting various structures of the organism.
9. Patients with cysts of various localisations should undergo laboratory diagnostics aimed at detecting herpesvirus infection.
10. The use of manual muscle testing (MMT) allows for diagnosis and individualised selection of antihomotoxic therapy regimens aimed at suppressing herpesvirus infection.
11. The presented data are based on analysis of the literature and clinical observations. The mention of complementary therapeutic approaches does not imply a causal relationship from the perspective of evidence-based medicine and does not replace standard methods of diagnosis and treatment.

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