

FOAR – facet joint osteoarthritis with radiculopathy – a case series and a hypothesis explaining spinal nerve irritation in the absence of osteodiskal compression

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Abstract

Isolated facet joint osteoarthritis is not widely recognized as the cause for neurologic symptoms in radiological MR imaging reports.

Purpose: To describe imaging findings of patients suffering from facet osteoarthritis with radiculopathy (FOAR).

Methods: Consecutive open case series in an out-patient environment with patients referred to MRI because of radicular pain or paraesthesia. 4 males and 4 women were found with activated degenerative zygoapophyseal joint disease by T2 fat suppressed sequences adjacent to the affected spinal nerve.

Results: Frequent localization of FOAR (n = 5) was the upper and middle cervical spine (C 1/2 to C 4/5) with one patient having occipital neuralgia, followed by the lower cervical spine (n = 2, both C 5/6). One lumbar case (L 5/S 1) was observed. Axial T2 weighted MRI sequences showed tissue with low-signal surrounding the facet joint obscuring the perineural structures within the intervertebral foramen. Compression of the spinal nerve by intervertebral disks or bony structures was absent.

Conclusions: We suggest periarticular and thus perineural inflammatory tissue as the reason for spinal nerve affection, which could be easily detected by MRI.

Keywords: Zygoapophyseal Joint – Osteoarthritis – Radiculopathy – Magnetic Resonance Imaging – Spinal Nerve

Introduction

Osteoarthritis is defined as “A progressive, degenerative joint disease, the most common form of arthritis, especially in older persons. The disease is thought to result not from the aging process but from biochemical changes and biomechanical stresses affecting articular cartilage. In the foreign literature it is often called osteoarthrosis deformans.” (National Library of Medicine – Medical Subject Headings, 2013).

Pain attributable to degenerative changes of the zygapophysial (or facet) joints of the spine is a common observation in out-patient clinical practice. The prevalence of facet related chronic neck pain has been estimated about 42% [1], or more than 50% [2]. However, there is an overweight reporting degenerative intervertebral disk and vertebral endplate changes in clinical imaging, presumably because of tradition, experience and focus on disk extrusion due to the large arsenal of interventional and surgical treatment. There is also a lack of primary literature as well as substantial passages in textbooks of radiology and neuroradiology concerning spinal nerve affection in isolated osteoarthritis of facet joints. Central spinal and recessal stenosis, as well as spinal nerve compression, are generally recognized, but the reporting radiologist may be at risk missing cases with activated degenerative facet joint disease with synovitis and periarticular inflammatory tissue with the ability to affect a segmental spinal nerve – which we call FOAR (*f*acet joint *o*steo*a*rthritis with *r*adiculopathy). This an open case series of imaging findings in patients with FOAR, to be proven by future controlled trials

Material and Methods

Setting: Urban out-patient environment with patients referred to magnetic resonance imaging (MRI) from the treating physician (usually a neurologist, n = 3, or an orthopedic specialist, n = 3) to a non-hospital imaging center. This open case series is the result of observational findings from December 2010 to January 2012 and have not been the subject for a controlled clinical trial or an institutional review board.

Patients: Eight patients (age 44 – 78 years, median 62 years, 4 males and 4 females) with pain or paraesthesia attributable to the affected segmental nerve. None had pre-treatment, relevant preceding diagnostic imaging or known systemic inflammatory joint disease or disease of the nervous system (Table 1). Occasional tests for rheumatoid arthritis and related diseases were negative. Within the study period, both authors examined 1337 spinal MRIs, whereof 448 were cervical.

Magnetic resonance imaging: Imaging was done on 1.5 or 3 Tesla clinical MRI systems (Siemens Magnetom Espree or Skyra, Erlangen, Germany). In all cases, sagittal T1 and T2 fast spin-echo sequences with slice thickness of 3 mm and a field-of-view (FOV) 280 or 320 mm were applied. All patients were scanned with a T2 weighted, fat suppressed fast spin-echo inversion recovery sequence (TIRM = turbo-inversion recovery-magnitude) in sagittal (n = 7) and / or coronar plane (n = 3). Axial imaging was parallel to the intervertebral disks in all cases with a T2 weighted fast spin-echo sequence, slice thickness of 3 mm, FOV 200 mm and in one case by a fat suppressed T2 TIRM sequence. Additionally, in three patients axial T1 weighted sequences were done with / without contrast media and fat suppression, two with a fat suppressed / non-fat suppressed T1 3D VIBE (= volume interpolated breath hold examination) sequence with slice thickness of 1 mm.

Clinical investigation and assessment of complaints and symptoms was done by the referring physician and detailed on the written referral. Additionally, the neuroradiologist or radiologist interviewed all patients before MRI. Treatment was done by the referring physicians, specific information was acquired by telephone interview or the patients file. Follow-up assessment was by telephone interview after various time points (up to 6 month) or personal observation.

Results

Patient characteristics, location of FOAR and clinical information are summarized in Table 1; two patients were lost for follow-up. All patients had erosive changes of the articular processes of the zygoapophyseal (facet) joints with subchondral bone marrow edema on spinal MRI and some hypertrophic changes of the articular processes. Effusion and bone marrow edema was shown by high signal adjacent to the articular surface on T2 fat suppressed sequences (Fig 1 a). Contrast enhancement of the facet joint capsule (Fig 2 a) and intraarticular spaces indicated synovitis. The joint capsules and the ligamenta flava were enlarged and the periarticular structures obscured: On axial T2 weighted images, low signal surrounding the joint and contrast enhancement was interpreted as periarticular inflammatory tissue involving the adjacent spinal nerve (Figs. 1 b, 2 b). The normal perineural structures like fat and blood vessels within the intervertebral foramen were not longer detectable (Fig 2); however, gross compression by intervertebral disks and bony structures was absent. In one case, the distal nerve showed slight signal increase that was interpreted as nerve edema or axonal damage (patient 7). He was treated by periradicular therapy and improved quickly.

Most frequently, the upper and middle cervical spine C 1/2 to 4/5 (n = 5) was involved. One patient, a 78 y old lady (patient 3), presented with long-lasting neuralgiform pain radiating into the right occipital region, classified as occipital neuralgia. The activated reactive changes of the right C 1/2 facet with some joint effusion clearly involved the epidural c2 spinal nerve (Fig 3). She didn't tolerate oral ibuprofen because of gastrointestinal side effects and more aggressive treatment was not considered, her clinical status was unchanged after 6 month. Two elder patients (4 and 5) were also affected at a higher cervical level: both had FOAR at level C 2/3 on the right side.

The only case with involvement of the lower spine (patient 2) was at the facet joint L 5 / S 1 on the right with extensive periarticular and bone marrow activation (Fig 4). Here, a segmental anomaly with residual S 1 / S 2 facets and a rudimental intervertebral disk at this level could have some implication with increased mechanical stress in the lumbosacral junction. Intramuscular cortisone injections over two weeks and oral diclofenac improved the radiating leg pain. Patient 8, a 49 year old male, had some pain and prickling paraesthesia attributable to the right c5 spinal nerve but improved spontaneously.

None of the patients showed relevant intervertebral disk degeneration or protrusion at the affected spinal level and no hypertrophic vertebral endplate changes were present. On follow-up, none developed fever or other signs of systemic or pyogenic arthritis and occasional tests for rheumatoid arthritis were negative.

Discussion

We presented an open case series of 8 patients with radicular pain and occasional paraesthesia from spinal nerve involvement in isolated facet joint osteoarthritis. Incidence of FOAR was

about 0.6 % and 1.8 % concerning the cervical spine. The most frequent location (5 of 8) was the upper and middle cervical spine C 1/2 to 4/5, in accordance with the prevalence of degenerative cervical facet changes in a cadaveric population [3]. None of the patients with FOAR had motor deficits indicating that gross compression with Wallerian demyelination didn't occur and only one patient showed signal increase of the distal spinal nerve. Instead, we propose spinal nerve irritation due to the inflammatory changes within and around the zygoapophyseal joint affecting the perineural tissue and the nerve itself as the cause for the symptoms. The clinical course was benign, if systemic or local anti-inflammatory therapy with cortisone and / or non-steroidal anti-inflammatory drugs (e.g. diclofenac) were prescribed. However, treatment could fail and improvement be delayed, if these drugs weren't tolerated or discontinued. Physical therapy stabilizing the paraspinal muscles had accompanying positive effect. Maybe local application of cortisone by periradicular injection could prove beneficial, but sufficient experience is still lacking.

Synovitis causing radicular symptoms in the cervical spine was not recognized until two patients reported in 1994 [4], who were imaged by contrast enhanced CT and treated surgically. Until now, to our knowledge, FOAR has very rarely been identified with one report including histological finding of hypertrophic synovial tissue [5]. In the absence of a systemic inflammatory joint disease, an inflammatory reaction and activation due to biomechanical stress within the facet joint could be mediated by cytokines [6, 7] and induce radiculopathy, a concept suggested by animal experiments [8].

Despite a prevalence of facet related pain of up to 50% [1, 2], activated degenerative facet changes are much less frequently reported in daily clinical work, because the focus is directed to spinal stenosis and disk herniation, as well as spinal nerve compression in the lateral recessus and intervertebral foramen. These pathologies are often treated surgically, whereas

facet osteoarthritis and degeneration is a domain of conservative and interventional pain therapy [2, 9]. The lack of surgical ambition and the high prevalence of about an overwhelming 50% of facet related pain [1] and an even higher prevalence of facet degeneration [3, 10, 11] may account for this. Furthermore, there may not be a correlation with imaging findings and therapeutic interventions like facet blocks [12]. In our case series, therapy was very conservative with only one of eight patients (patient 7) treated by combined cortisone and local anesthetics injection. Maybe the lady with occipital neuralgia (patient 3) would have benefited from local therapy, as she didn't tolerate ibuprofen due to gastrointestinal adverse effects. But there was no consent for more aggressive therapy, despite there are even surgical options for occipital neuralgia and atlantoaxial osteoarthritis applying screw fixation [13, 14].

The imaging findings in FOAR were uniform with erosive and somewhat hypertrophic changes of the zygoapophyseal joints with bone marrow edema, joint effusion and most strikingly inflammatory synovial tissue adjacent to the spinal nerve within the intervertebral foramen. Bone marrow edema and effusion is easily seen with T2 weighted fat suppressed MR sequences [15] and contrast enhancement could give additional evidence for active inflammation. After notation of these changes, most useful for detection of direct affection of the spinal nerve were axial T2 weighted sequences with low signal changes of the joint capsule and annihilation of the high signal surrounding the spinal nerve representing perineural fat. Low signal on T2 indicates tissue with considerable amount of cells, which is consistent with inflammatory infiltration and thus contrast enhancing on T1. Additional CT imaging or radiography is judged to be dispensable [16]. Three Tesla clinical MR imaging with good shimming to provide homogeneous fat suppression and high resolution has facilitated detection of inflammatory joint reaction with spinal nerve involvement, but with sufficient alertness FOAR is easily found with 1.5 T systems, too. Interestingly, in case of

FOAR relevant disk degeneration, protrusion and endplate changes were absent in the affected but usually present in the adjacent spinal segments, possibly indicating elevated biomechanical stress at the neighboring facet joints.

Limitations: We do not have a histological confirmation of our observations and the assessment of radiculopathy is not verified by objective tests others than simple physical investigation. Clinical follow-up is somewhat incomplete and follow-up imaging is usually not intended as the degenerative joint changes are commonly not reversible or rapidly progressive. Due to rational and pragmatic diagnosis and therapy in an out-patient setting, this case sample doesn't qualify for a controlled clinical trial but presents observational findings in a high volume imaging center.

Today, we cannot rule out, that the symptoms arise from the facet joint only. A large controlled trial with both, osteoarthritis with and without involvement of perineural tissue and thorough correlation with clinical symptoms could give further evidence for our hypothesis.

Conclusion: Facet joint osteoarthritis with radiculopathy may be more common than usually reported and is easily detected by fat suppressed T2 sequences, preferably supported by T1 contrast enhancement with fat suppression. The clue is osteoarthritis with annihilation of high signal surrounding the spinal nerve representing perineural fat on T2 weighting without gross compression by intervertebral disks or bony structures in the intervertebral foramen.

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Table 1: Patient Characteristics, Symptoms, Treatment and Follow-up

Patient	Age [y]	Sex	Level	Side	Complaints	Treatment	Follow-up
1	48	f	C 5/6	R	radicular pain c6	iv cortisone	improved
2	61	m	L 5/ S 1	R	radicular pain l5 / s1	im cortisone, diclofenac	symptom-free
3	78	f	C 1/2	R	occipital neuralgia c2	ibuprofen, discontinued	not improved
4	75	m	C 2/3	R	radicular pain c3, paraesthesia, vertigo	n.d.	n.d.
5	67	f	C 2/3	R	radicular pain c3, paraesthesia	n.d.	n.d.
6	44	f	C 3/4	L	radicular pain c4, snapping phenomena	diclofenac, physiotherapy	improved
7	62	m	C 5/6	R	radicular pain c6	periradicular therapy	improved
8	49	m	C 4/5	R	paraesthesia c5	non	improved

Figure Captions

Fig 1 (a) Sagittal T2 TIRM fat suppressed image of the cervical spine in a 44 y old female patient (patient 6) with left sided pain since 4 weeks, radiating to the shoulder. Long arrows point at edematous subchondral bone marrow in the left C 3/4 facet articular processes. On axial T2 weighted images (b) there is enlargement of the facet joint capsule without effusion but low signal tissue surrounding the capsule (short arrows). The spinal nerve c4 is obscured in the intervertebral foramen indicating involvement into the inflammatory changes (striking difference to the right c4 nerve). The patient was treated with relative low dose diclofenac for 6 weeks and physical therapy. She improved significantly over a period of twelve month

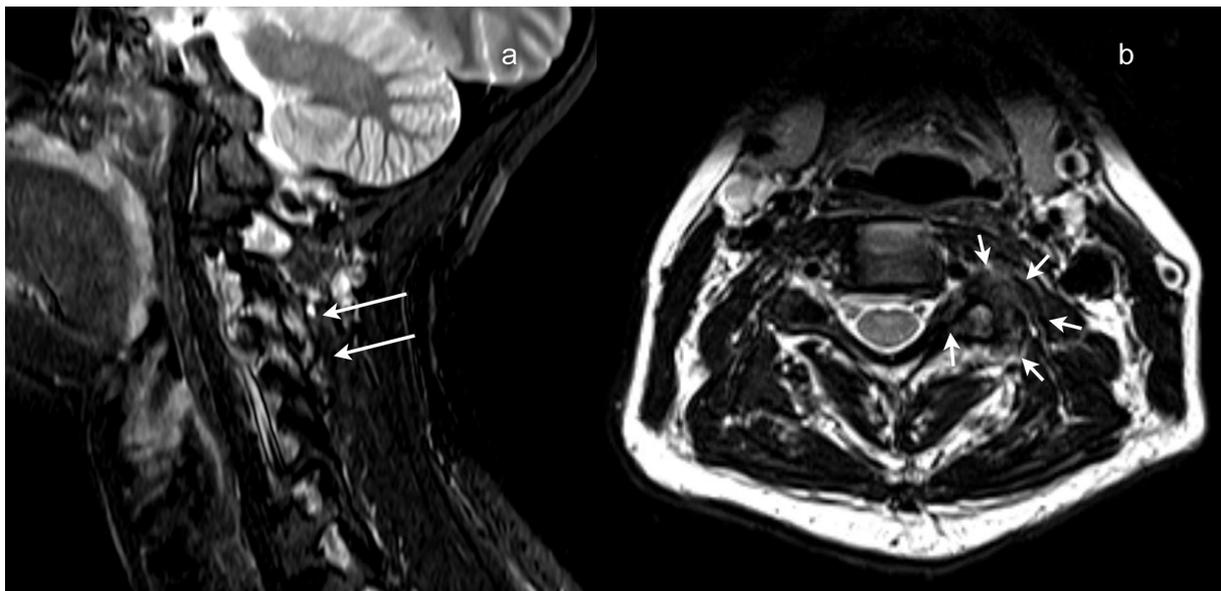


Fig 2 (a) Axial T1 VIBE fat suppressed image after contrast enhancement (patient 1). The C 5/6 facet joint on the right side shows slight hypertrophic apophyseal bony changes, a small subchondral cyst und low to intermediate signal (b) of the joint capsule and the surrounding tissue involving the intervertebral foramen c6. The spinal nerve is visible after contrast enhancement in the intervertebral foramen as a thin low signal structure adjacent to the

swollen joint capsule in (a) but completely obscured on T2 - in comparison with the normal intraforaminal structures on the left side (b)

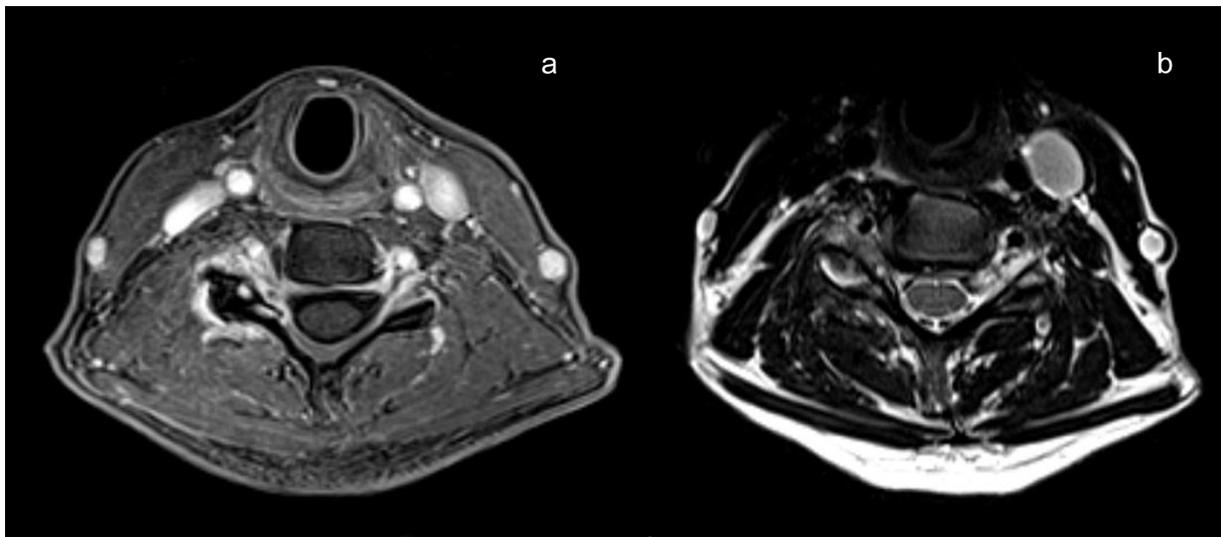


Fig 3 Coronar T2 fat suppressed TIRM in patient 3, a 78 year old lady (patient 3) with right-sided occipital neuralgia (a). The right apophyseal joint C1/2 (*) shows bone marrow edema and effusion. The subchondral endplates are slightly enlarged, thus the dens appear eccentric (which isn't really true in relation to the lateral masses of the atlas). (b) Axial T1 fat suppressed VIBE sequence after contrast injection at level C1/2 shows intense signal increase of the joint capsule and synovial reaction, even within the epidural space at the level of spinal nerve c2 (*). Arrow on the left side: c2 spinal nerve penetrating the normal epidural space, while on the right side, the spinal nerve is obscured by presumed inflammatory tissue

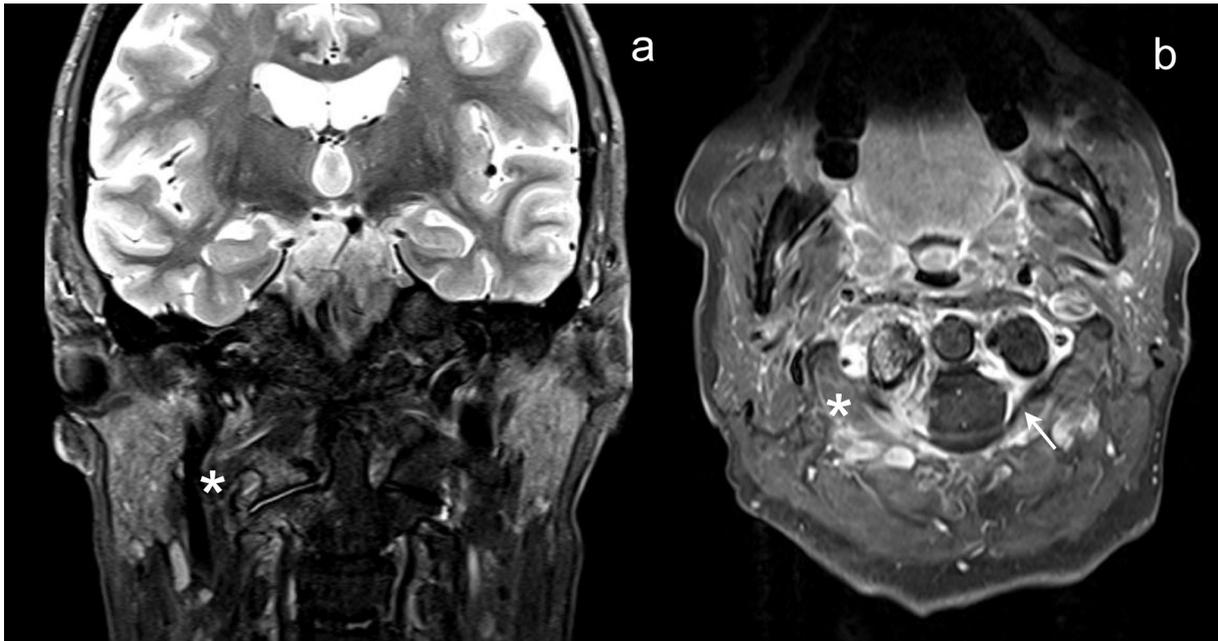
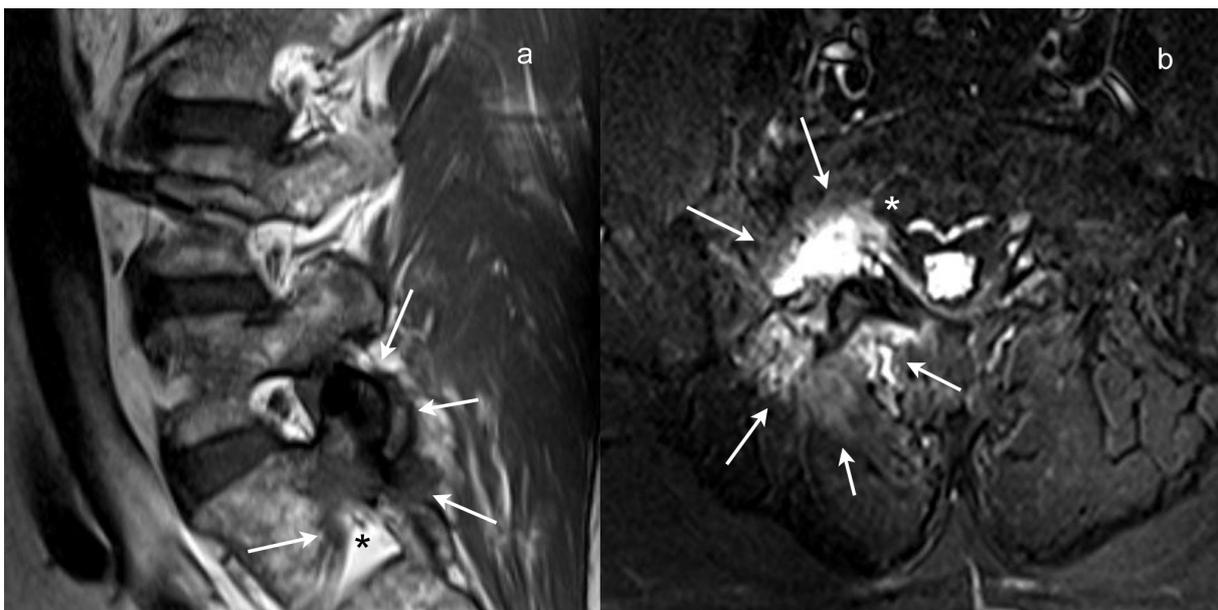


Fig 4 The lumbar case, a 61 y old male (patient 2). (a) Slight joint hypertrophy, irregular articular facets and periarticular tissue L 5/S 1 on sagittal T1 with reaction in the paraspinal muscles (arrows) as well in the bone marrow of the sacrum (b), T2 fat suppressed axial sequence. The star (*) indicates the s1 spinal nerve in the foramen on both images, which is directly involved. There is a segmental anomaly as there is a rudimentary S 1/2 facet joint right to the star in (a)



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