ORIGINAL ARTICLE

# Odontogenic cervical necrotizing fasciitis, etiological aspects

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## Abstract

**Introduction:** Cervical necrotizing fasciitis is a rare but very severe infection that affects the soft-tissues of the cephalic extremity. Cervical necrotizing fasciitis most frequently occurs secondarily to inflammatory odontogenic disorders and represents the most severe infection of maxillofacial spaces, with a high lethal potential.

**Materials and Methods:** In this study, we selected 55 patients with confirmed cervical necrotizing fasciitis of odontogenic origin, treated in the Clinic of Oral and Maxillofacial Surgery in Cluj-Napoca during January 1996-December 2012.

**Results:** In the majority of cases, the disease evolved without the presence of associated systemic disorders (60% [45.49-72.69]), the rest of the patients having 1-4 types of systemic disorders; type 2 diabetes mellitus was the most frequent type of underlying systemic disorder. From the appearance of the first symptoms until the presentation for treatment, a time interval of 2-30 days elapsed. During this time period, 78.18% (95% confidence interval [CI] [65.49-89.06]) of the patients received antibiotic treatment, but without results. Mandibular molars were the most frequent starting point of the disease, and the submandibular space was the first affected by the disease, 47.27% (95% CI [32.76-61.79]). Bacteriological exams showed that facultatively aerobic/anaerobic G + bacteria were the most frequently identified (72.22% [58.21-83.60]).

**Conclusion:** The odontogenic lesions of the lower molars, complicated by submandibular space infections, are the most frequent starting point of odontogenic cervicofacial necrotizing fasciitis. Delayed surgical treatment and strict antibiotic therapy play an important role in favoring the development of odontogenic necrotizing fasciitis.

**Key words:** Head and neck infections, necrotizing fasciitis, odontogenic infection

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## Introduction

Necrotizing fasciitis is a severe bacterial infection with rapid evolution along the fascial planes, with the involvement of adjacent tissues, accompanied by systemic toxico-septic phenomena.[1,2] The term of necrotizing fasciitis was introduced by Wilson in 1952[3] and is considered as the most adequate for describing this disease.[1] Necrotizing fasciitis most frequently affects the lower extremity of the body, its presence in the cephalic extremity being an exception.[4,5] Cervical necrotizing fasciitis most frequently occurs secondarily to inflammatory odontogenic disorders and represents the most severe infection of maxillofacial spaces, with a high lethal potential.[6,7] Although the lethal risk of maxillofacial infections has decreased due to the development of antibiotics, over the past 10-15 years there has been an increase in bacterial resistance to antibiotics, with an aggravation of the severity of odontogenic infections.[8,9]

A multitude of aerobic and anaerobic bacterial species with a synergistic action are incriminated in the development of necrotizing fasciitis.[10,11] At the same time, an important role in the occurrence of this disease is attributed to the background on which it evolves, the most frequently

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incriminated disorders being diabetes mellitus, obesity, immune deficiencies, chronic alcoholism, or hepatic deficit.[5,7,8,10,11] The literature reports the infection of the partially erupted third mandibular molar as the main starting point of odontogenic necrotizing fasciitis.[12-15] From this level, the bacterial flora initially affects the submandibular space and subsequently extends to the fascial system, with the development of necrotizing fasciitis.[12-15] The limitations of these reports regarding the etiology of the development of necrotizing fasciitis should be noted. These limitations are due to the relatively small number of cases included in studies, or even if data on a significant number of cases have been collected, these are predominantly limited odontogenic infections, not necrotizing fasciitis cases. It can be noted that the majority of the authors who evaluate necrotizing fasciitis of the head and neck do not take into account its starting point, although this can be decisive in terms of the characteristics of the bacterial flora involved.[5,14,16]

The aim of this study was a detailed assessment of more than 40 cases in order to obtain a statistically significant evaluation of the way in which strictly odontogenic necrotizing fasciitis develops.

## Materials and Methods

For this study, the patients hospitalized and treated for necrotizing fasciitis of the head and neck in the period January 1996-December 2012 were available. Data on each case were obtained through the study of the clinical observation records and of the paraclinical investigation results (imaging interpretation results, bacteriological examinations, laboratory investigations).

### Study inclusion criteria

The presence of intraoperatively confirmed necrotizing fasciitis, the odontogenic origin of infection. Following the analysis of the clinical observation records and paraclinical investigations, 3489 cases of odontogenic infections of head and neck soft tissues were detected in the host department; of these, 55 patients had necrotizing fasciitis.

The following were analyzed in the patients included in the study: General data (age, sex, environment of origin), data-related to the starting point of necrotizing fasciitis, bacterial flora involved, treatment received until the presentation to the specialized service (presence of previous treatment, type of treatment, characteristics of antibacterial therapy received), data related to the clinical manifestations of the disorder until the presentation for specialized treatment. In parallel, the background on which the disorder developed (presence of systemic disorders, their degree of compensation) was evaluated.

The study protocol was approved by the Ethics Committee of the "Iuliu Ha□ieganu" University of Medicine and Pharmacy on May 16, 2014, approval number 173. All the patients included in the study signed an informed consent and agreed to participate in scientific studies.

For contingency tables, the Microsoft Excel software was used. Quantitative data were summarized as absolute and relative frequency. To allow generalizability, 95% confidence intervals (CI) for relative frequencies were computed using an optimized formula.[17,18]

The comparison of two proportions was conducted with the Z-test. A $P < 0.05$ was considered as statistically significant. The association in contingency tables was quantified using the contingency coefficient whenever data were nominal, or the gamma coefficient when data were ordinal at a significance level of 5%.

Statistical analysis was performed using the IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.

## Results

The study inclusion criteria were met by a number of 55 patients, 31 males and 24 females ($Z = -1.9143$, $P = 0.0556$). The incidence of odontogenic necrotizing fasciitis cases remained constant during the analyzed years, with a minimum of 1 case, a maximum of 7 cases and an average of 3.23 cases/year.

The age of the patients included in the study ranged between 17 and 78 years, with a mean of 41.03 years and a maximum incidence in the third decade of life. The male sex was the most frequently affected by this disorder (56.36%), but without a significant difference compared to the female sex. The distribution of patients depending on their environment of origin was balanced between rural areas (28 patients) and urban areas (27 patients).

In the majority of the patients, the disease evolved without the presence of associated systemic disorders (60% [45.49-72.69]), the rest of the patients having 1-4 types of systemic disorders; type 2 diabetes mellitus was the most frequent type of underlying systemic disorder [Table 1]. In the majority of the patients who had systemic disorders, these were decompensated at the time of presentation for specialized treatment (statistical $Z = -2.5749$, $P = 0.01$). Decompensation was found in 56.25% of patients with a single associated systemic disorder compared to 83.33% of patients with more than one associated systemic disorder (statistical $Z = -1.3795$, $P = 0.1677$).

The group of teeth that was the most frequent starting point of necrotizing fasciitis was represented by mandibular molars, of which the most frequent starting point was the right third mandibular molar, 16.36% (95% CI [7.31-29.06]) of the cases, followed by the right first mandibular molar, 14.54% (95% CI [5.49-27.24]) of the cases. The first

area affected by the septic process was the submandibular gland space in the case of 47.27% of the patients (95% CI [32.76-61.79]), followed by the cheek region in 20% of the cases (95% CI [10.94-32.69]), and the sublingual gland space in 12.73% of the cases ([5.49-23.60]).

From the onset of the disorder to the presentation of patients for specialized treatment, a time period varying between 2 and 30 days elapsed, with a median of 5 days and an interquartile range from 3.5 to 7 days. During this time interval, the majority of the patients received antibiotic treatment, 78.18% (95% CI [65.49-89.06]) [Figure 1].

In most cases, patients received antibiotic treatment with a single type of antibiotic (81.40% [69.12-90.88]), but there were cases when two types (13.95% [5.49-27.24]) or even three types (4.65% [1.86-14.51]) of antibiotics were administered. The predominant route of administration of antibiotics was p.o., 76.74% [63.67-87.24], the rest being administered intravenous (i.v.) (23.26% [11.68-39.48]). The proportion of patients who had oral antibiotic administration was statistically significantly higher compared to the proportion of patients with i.v. administration (statistical $Z = -8.3006$, $P < 0.0001$). In the studied sample, there were 14 anti-biotherapy schemes [Table 2]. There was a wide variety of administered antibiotics, ampicillin being the most frequently used (32.56% [20.03-47.24]), followed by amoxicillin (16.28% [7.31-29.06]).

From the septic focus, biological samples were taken and bacteriological examination was performed in 67.27%

**Table 1: Associated systemic pathology and its type (compensated/decompensated)**

| Associated pathology | Total | Compensation | | |
|---|---|---|---|---|
| | | Yes | No | *Z* (*P*) |
| Cardiac | 2 (9.09 [0.21-27.07]) | 2 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Leukemia | 1 (4.55 [0.21-22.52]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Syphilis | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| DM 1* | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| DM 2** | 7 (31.82 [13.84-54.34]) | 1 (14.29 [2.04-55.1]) | 6 (85.71 [44.9-97.96]) | 5.40 (<0.0001) |
| Malignant | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| Hepatic | 1 (4.55 [0.21-22.52]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Chronic malnutrition | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 0 (n.a.) | n.a. |
| Adrenocortical insufficiency | 1 (4.55 [0.21-22.52]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Cardiac + DM 2** | 3 (13.64 [4.75-36.16]) | 1 (33.33 [11.1-88.9]) | 2 (66.67 [11.1-88.9]) | 1.25 (0.2104) |
| Cardiac + DM 2**+ neoplasia+hepatic | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| Cardiac + DM 2**+ COBP | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| DM II**+ hepatic | 1 (4.55 [0.21-22.52]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| Total | 22 | 7 | 13 | |

*Diabetes mellitus type 1, **Diabetes mellitus type 2. DM=Diabetes mellitus; COBP=Chronic obstructive bronchopneumonia

**Table 2: Distribution of patients depending on the type of antibiotic treatment received prior to the presentation for specialized treatment and its form of administration**

| Antibiotic | Total (percentage [95%CI]) | Type of administration | | |
|---|---|---|---|---|
| | | Intravenous (percentage [95%CI]) | Per os (percentage [95% CI]) | Z (*P*) |
| Amp | 14 (32.56 [18.66-48.78]) | 1 (7.14 [0.51-35.2]) | 13 (92.86 [64.8-99.49]) | 12.46 (<0.0001) |
| Pen | 4 (9.30 [2.38-20.88]) | 1 (25.00 [6.25-68.75]) | 3 (75.00 [31.25-93.75]) | 2.31 (0.0209) |
| Aug | 3 (6.98 [2.38-18.55]) | 0 (n.a.) | 3 (100 [n.a.]) | n.a. |
| Oxa | 3 (6.98 [2.38-18.55]) | 0 (n.a.) | 3 (100 [n.a.]) | n.a. |
| Cli | 2 (4.65 [0.05-16.23]) | 1 (50.00 [25.00-75.00]) | 1 (50.00 [25.00-75.00]) | n.a. |
| Dox | 2 (4.65 [0.05-16.23]) | 0 (n.a.) | 2 (100 [n.a.]) | n.a. |
| Amo | 7 (16.28 [7.03-30.18]) | 1 (14.29 [2.04-55.1]) | 6 (85.71 [44.90-97.96]) | 5.40 (<0.0001) |
| Amp + dox | 1 (2.33 [0.05-11.57]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Amp + gen | 2 (4.65 [0.05-16.23]) | 1 (50.00 [25.00-75.00]) | 1 (50.00 [25.00-75.00]) | n.a. |
| Amp + gen + met | 1 (2.33 [0.05-11.57]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Pen + gen | 1 (2.33 [0.05-11.57]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Aug + ceph | 1 (2.33 [0.05-11.57]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Dox + met | 1 (2.33 [0.05-11.57]) | 0 (n.a.) | 1 (100 [n.a.]) | n.a. |
| Gen + amo + met | 1 (2.33 [0.05-11.57]) | 1 (100 [n.a.]) | 0 (n.a.) | n.a. |
| Total | 43 | 10 | 33 | |

Amp=Ampicillin; Pen=Penicillin; Aug=Augmentin; Oxa=Oxacillin; Cli=Clindamycin; Dox=Doxycycline; Amo=Amoxicillin; Gen=Gentamicin; Met=Metronidazole; Ceph=Cephalosporin; 95% CI=95% confidence interval

**Table 3: Identified bacterial strains**

| Bacterial flora (number of strains × number of subjects) | Facultatively aerobic G− | Facultatively aerobic G+ | Anaerobic G− | Facultatively aerobic G− and facultatively aerobic G+ | Total number of subjects |
|---|---|---|---|---|---|
| *B. fragilis* (1×1) | 0 | 0 | 1 | 0 | 1 |
| *G + cocci + G − cocci* (*n*×1) | 0 | 0 | 0 | 1 | 1 |
| *E. cloacae* (1×1) | 1 | 0 | 0 | 0 | 1 |
| *E. faecalis + S. epidermidis + A. baumannii* (3×1) | 1 | 2 | 0 | 0 | 1 |
| *E. faecium* (1×1) | 0 | 1 | 0 | 0 | 1 |
| *E. coli* (1×6) | 6 | 0 | 0 | 0 | 6 |
| MRSA* (1×3) | 0 | 3 | 0 | 0 | 3 |
| MSSA** (1×8) | 0 | 8 | 0 | 0 | 8 |
| MSSA + *S. pyogenes* (2×2) | 0 | 1 | 0 | 0 | 2 |
| *P. aeruginosa + K. pneumoniae + E. faecalis* (3×1) | 0 | 0 | 0 | 1 | 1 |
| α hemolytic *Staphylococcus* species (*n*×1) | 0 | 1 | 0 | 0 | 1 |
| *S. epidermidis* (1×1) | 0 | 1 | 0 | 0 | 1 |
| *S. mitis* (1×1) | 0 | 1 | 0 | 0 | 1 |
| Group A β-hemolytic *streptococcus* (1×3) | 0 | 3 | 0 | 0 | 2 |
| *S. pyogenes* (1×3) | 0 | 3 | 0 | 0 | 3 |
| *S. sanguis* (1×1) | 0 | 1 | 0 | 0 | 1 |
| *S. viridans* (1×1) | 0 | 1 | 0 | 0 | 1 |
| Total | 7 | 26 | 1 | 2 | 36 |

*MRSA=Methicillin-resistant *Staphylococcus aureus*; **MSSA=Methicillin-sensitive *Staphylococcus aureus*. *E. faecalis=Enterococcus faecalis; S. epidermidis=Staphylococcus epidermidis; B. fragilis=Bacteroides fragilis; E. cloacae=Enterobacter cloacae; A. baumannii=Acinetobacter baumannii; E. faecium=Enterococcus faecium; E. coli=Escherichia coli; S. pyogenes=Streptococcus pyogenes; P. aeruginosa=Pseudomonas aeruginosa; K. pneumonia=Klebsiella pneumonia; S. mitis=Staphylococcus mitis; S. sanguis=Staphylococcus sanguis; S. viridans=Staphylococcus viridans*

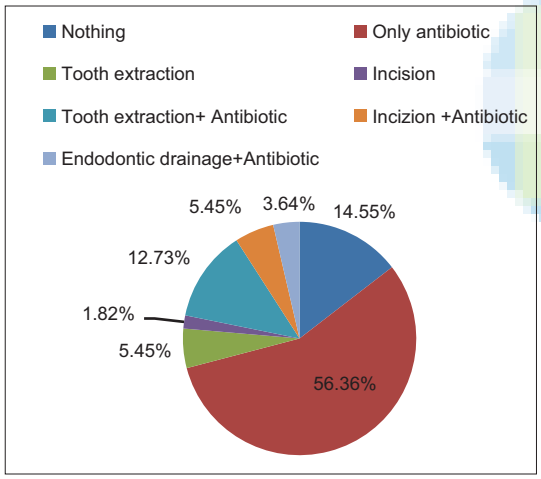


**Figure 1:** Distribution of patients depending on the type of treatment received prior to the presentation for specialized treatment

of the patients (95% CI [52.76-79.97]); one bacterial strain was identified in 86.11% of the patients (95% CI [72.76-94.51]), 2 bacterial strains were identified in 5.56% of the patients (95% CI [1.85-14.51]), 3 bacterial strains were found in 5.56% of the patients (95% CI [1.85-14.51]), and in one patient, several bacterial strains were described (95% CI [0.03-9.058]). Facultatively aerobic/anaerobic G+bacteria were the most frequently identified (72.22% [58.21-83.60]), followed by facultatively aerobic/anaerobic G−bacteria (19.44% [10.94-32.69]), and the association of facultatively aerobic/anaerobic G+and G−bacteria (5.56% [1.85-14.51]). Anaerobic G− bacteria were discovered in 2.78% (95% CI [0.03-12.69]) of the cases. The distribution of the main categories of microorganisms depending on the number of identified strains and the number of subjects is shown in Table 3.

## Discussion

The results obtained in this study show the fact that necrotizing fasciitis affects individuals regardless of their age, sex or environment of origin. Thus, the highest incidence of odontogenic necrotizing fasciitis cases was found in the third decade of life, without statistical difference between the two sexes. This result is in accordance with the results obtained by other studies evaluating odontogenic soft tissue infections, which report the highest incidence in young adults, regardless of sex.[8,12] However, it is different from those of other studies evaluating cervicofacial necrotizing fasciitis, which report the highest incidence of this disorder in adults aged 40-50 years.[14,16] This difference can be explained by the fact that in these studies, the disease occurs in the majority of the cases on an immunosuppressed background secondarily to chronic disorders such as diabetes mellitus or HIV infection, which have a long evolution.[14,16,19] However, in this study, the incidence of immunosuppressive comorbidities in patients with necrotizing fasciitis is also higher compared to the incidence found in patients with limited odontogenic infections of head and neck soft-tissues.[8,12] Type 2 diabetes mellitus is the most frequently found comorbidity as a single disorder or associated with other comorbidities, and an aggravating factor is the fact that only in one case the disorder was

medically compensated. These results are similar to those obtained by other authors, who report a frequent presence of diabetes mellitus in patients with necrotizing fasciitis.[14,16,19] In a study carried out by Juncar *et al*.,[7] it was shown that the degree of compensation, as well as the duration of diabetes mellitus, were particularly important in the evaluation of its impact on the immune system.

In this study, the odontogenic starting point of necrotizing fasciitis and the first location of the infection in the soft-tissues were evaluated in detail. The analysis of odontogenic infections made by other author's evidences that the main starting point for odontogenic infections in general and odontogenic necrotizing fasciitis is represented by mandibular molars.[1,12,14,20] A similar result was obtained in this study, which indicates mandibular molars, particularly the third molar, as the main starting point of necrotizing fasciitis. The fact that the submandibular space is in close proximity to the mandibular molars can explain that this was first affected by the septic process, and from this level, the infection extended to the fascial level. Thus, it results that early effective treatment of odontogenic submandibular infections is particularly important in the prevention and treatment of necrotizing fasciitis.[16,20,21]

The literature reports highly variable time periods from the onset of the first symptoms to the presentation of the patient with necrotizing fasciitis for emergency treatment. Some authors evidence clinical cases in which necrotizing fasciitis developed very quickly, in 2 or 3 days.[13] Other authors report longer time periods, up to 14 days elapsed from the first symptoms to the patient's presentation for specialized treatment.[1,20] Specialized treatment was given to the patients included in this study about 5 days after the onset of the disorder, the cases with an extremely rapid or an extremely slow onset being rare. In fact, this time interval is similar to that found in the case of patients with limited odontogenic suppurations.[7] The majority of the patients had antibiotic treatment prior to their presentation for specialized treatment (56.36%), but this treatment could not limit the extension of the septic process. The fact that patients with necrotizing fasciitis had in most of the cases, before their presentation for specialized treatment, antibiotic therapy alone is also reported by other studies.[1,8,14,22] These studies show that antibiotic therapy alone, without surgical treatment, cannot effectively treat odontogenic soft tissue infections.[1,8,14] The data available in this study do not allow for a correct analysis of the effectiveness of antibiotics as a single treatment in odontogenic soft tissue infections. However, we can support the fact that for patients with necrotizing fasciitis, they were ineffective, despite the association of up to three types of antibiotics in certain cases.

The delayed presentation for specialized treatment is more frequently found in patients with comorbidities, although these show a higher incidence of severe odontogenic infections.[7] The literature incriminates comorbidities as the main factor in the development of severe odontogenic infections.[1,4,5,8,14,16] It is obvious, based on the results of this study, which the presence of immunosuppressive disorders favors the occurrence of necrotizing fasciitis, but this factor cannot be demonstrated to be the main causal factor. It can be noted that the majority of the patients with necrotizing fasciitis have no comorbidities. The data obtained indicate that delayed surgery and antibiotic therapy as a single treatment are much more important in the development of cervical necrotizing fasciitis of odontogenic origin. A limitation of the study is the fact that it does not analyze a group of patients with acute odontogenic disorders who receive strict antibiotic treatment, in order to see how many of these develop necrotizing fasciitis. Such a study is not possible for human and ethical reasons, but it is known from current medical practice that the majority of the patients receive only antibiotic treatment during the first stages of odontogenic inflammation.

In most of the necrotizing fasciitis cases, a single bacterial strain was identified as being responsible for the development of necrotizing fasciitis. This is in contradiction with other studies that most frequently show the presence of multiple bacterial strains involved in the development of necrotizing fasciitis.[15,23] There are two possible explanations for these different results. Antibiotic treatment, administered prior to biological sample collection, might have eliminated the antibiotic-sensitive bacterial flora, which allowed for the excessive development of a single bacterial strain. Another explanation may result from the fact that, in general, bacteria that are easy to identify were discovered, which would indicate a deficiency in the identification of all present bacterial species. However, the first statement might be more plausible because the bacterial species discovered in patients with necrotizing fasciitis are similar to those discovered by most literature studies.[5,16,15,23,24] Surprisingly, the most frequent species found at the level of simple odontogenic abscesses or endodontic or periodontal lesions, where the *Streptococcus* strains are dominant,[12,25] were not detected in necrotizing fasciitis areas. Both methicillin-resistant and methicillin-sensitive *Staphylococcus aureus* strains were most frequently discovered in septic necrotic areas, in this as well as other studies.[5,16,15,23,24] These differences in bacterial flora between limited odontogenic abscesses and odontogenic necrotizing fasciitis can explain the different clinical and evolutive features between the two types of infections. Since an important number of odontogenic necrotizing fasciitis cases start from relatively common odontogenic abscesses, it can be inferred that at a given moment, there is a significant change in the bacterial flora present in the septic focus. Further studies are necessary in order to determine precisely the way in which this changes, but as shown by this study, single antibiotic therapy and the delayed drainage of septic areas favor this bacterial imbalance.

## Conclusion

The odontogenic lesions of the lower molars, complicated by submandibular space infections, are the most frequent starting point of odontogenic cervicofacial necrotizing fasciitis.

Delayed surgical treatment and strict antibiotic therapy play an important role in favoring the development of odontogenic necrotizing fasciitis. The combination of these two factors can be considered as important as the presence of immunosuppressive comorbidities in the etiology of necrotizing fasciitis.

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