

Література

1. Бондаренко А.Н. Особенности патогенеза и терапии парентеральных вирусных гепатитов у больных, употребляющих наркотики: дисс. ... доктора мед. наук / А.Н. Бондаренко. – Киев, 2005. – 351 с.
2. Зайцев И.А. Лечение больных хроническим вирусным гепатитом С, не ответивших на противовирусную терапию / И.А. Зайцев // Сучасні інфекції. – 2007. – № 4. – С. 41-50.
3. Майер К.-П. Гепатит и последствия гепатита / К.-П. Майер. – М.: Гэотар-Мед, 2001. – 424 с.
4. A functional SNP of interferon-gamma gene is important for interferon-alpha-induced and spontaneous recovery from hepatitis C virus infection / [Y. Huang, H. Yang, B. B. Borg et al.] // Proc. Natl. Acad. Sci. (PNAS) USA. – 2007. – Vol. 104, N 3. – P. 985-990.
5. Polymorphisms in interferon-induced genes and the outcome of hepatitis C virus infection: roles of MxA, OAS-1 and PKR / [S. Knapp, L.J. Yee, A.J. Frodsham et al.] // Genes Immun. – 2003. – N 4. – P. 411-419.
6. Selzner N. Can genetic variations predict HCV treatment outcomes? / N. Selzner, I. McGilvray // J. Hepatology. – 2008. – Vol. 49. – P. 548-556.
7. Бондаренко А.Н. Этиотропная терапия вирусных гепатитов у лиц, употребляющих наркотики / А.Н. Бондаренко // Вестник Санкт-Петербургской медицинской академии им. Мечникова. – 2006. – № 4. – С. 168-175.
8. Патент деклараційний № 69221 А Україна. МПК 7 А61В 10/00; G01N 33/48. Спосіб прогнозування ускладнень від дії індукторів інтерфероногенезу у наркозалежних хворих на вірусний гепатит / Бондаренко А.М.. – № 20031211673; Заявл. 16.12.2003; Надрук. 16.08.2004. – Бюл. № 8. – 3 с.
9. Прогнозирование эффективности интерферонотерапии при различных формах патологии / [М.В. Мезенцева, А.Н. Наровлянский, Т.П. Оспельникова и др.] // Иммунология. – 2001. – № 4. – С. 41-44.
10. «Тройная» терапия хронического вирусного гепатита С у пациентов с генотипом 1b / [М.Г. Романцов, Т.В. Сологуб, С. Гуренкова и др.] // Врач. – 2006. – № 7. – С. 1-4.
11. Shiffman M.L. What future for ribavirin? / M.L. Shiffman // Liver Int. – 2009. – Vol. 29, N 1. – P. 68-73.
12. Stauber R.E. Drugs in development for hepatitis C / R.E. Stauber, H.H. Kessler // Drugs. – 2008. – Vol. 68, N 10. – P. 1347-1359.
13. Triple antiviral therapy in HCV positive patients who failed prior combination therapy / [S. Fargion, M. Borzio, A. Maraschi et al.] // World J. Gastroenterol. – 2006. – Vol. 12, N 33. – P. 5293-5300.
14. Научные разработки НИУ РАМН – практическому здравоохранению / под ред. М.И. Давыдова. – Москва, 2004. – Вып. 4. – 224 с.

ANTIVIRAL ACTIVITY OF CYCLOFERON AT THE ETIOTROPIC THERAPY OF HEPATITIS C

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SUMMARY. The article adduces the data on peculiarities of antiviral and immunomodulatory therapy of viral hepatitis. The special attention is paid to application of interferons and inductors of interferonogenesis as monotherapy and in combination with antiviral chemodrugs. The comparative analysis of efficiency of interferons and inductors of interferonogenesis at the therapy of viral hepatitis C was conducted. Tactics and criteria of optimum therapy with preparations of interferons and inductors of interferonogenesis are developed.

Key words: *interferons, inductors of interferonogenesis, treatment, viral hepatitis.*

Отримано 18.04.2012 р.

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УДК 616.995.122.21

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NECROTIZING FASCIITIS – CASE REPORT AND REVIEW OF THE LITERATURE

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Necrotizing fasciitis (NF) is a severe soft tissue infection characterized by necrosis of the skin, subcutaneous tissue and fascia. This rare condition carries high mortality rate and require prompt

diagnosis and urgent treatment includes broad-spectrum antibiotic coverage, hemodynamic support, nutritional supplements, wound care, and surgical debridement.

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We describe a case of 63-year old man who presented with the history of diffuse erythema, edema, pain on the lower leg 4 days before hospitalisation. Initially he was admitted and treated for erysipelas but later was diagnosed as necrotizing fasciitis which was successfully treated with no ill effects what so ever from this devastating condition.

Paucity of cutaneous findings early in the course of the disease makes diagnosis challenging. Prompt surgical debridement, intravenous antibiotics, fluids and electrolytes management and analgesia are mainstays of the therapy. Adjuvant treatments like clindamycin, hyperbaric oxygen therapy and intravenous immunoglobulins are discussed.

Key words: necrotizing fasciitis (NF), skin and soft tissue infections.

Necrotizing fasciitis (NF) is a rare, life-threatening, soft-tissue infection characterized by rapidly spreading inflammation and necrosis of the skin, subcutaneous fat, and fascia [1]. The earliest reference to this condition was made by Hippocrates in the 15th century BC, who spoke of it as a complication of erysipelas [1, 2]. However, the term necrotizing fasciitis was not coined until 1952. Over the years, other terms have been used to refer to NF, including phagedena gangrenosum, Meleney's gangrene, Fournier's gangrene, flesh-eating bacteria syndrome, suppurative fasciitis, hospital gangrene, and necrotizing erysipelas [3, 4, 5]. Because of the rapid progression inherent in NF, it is important to recognize and treat NF quickly to reduce mortality [1].

Case

A 63-year old man with co-morbidities (arterial hypertension, diabetes mellitus type 2, state after ischemic brain stroke) was referred to the university clinic of dermatology and venereology from emergency department with the history of high fever, diffuse erythema, edema, pain and difficulty in movement of the right lower leg four days ago. Pain got worse and he attended the emergency department from where he was referred to us with the suspicion of erysipelas.

On arrival, he was febrile (39.6 °C) and systemically stable but in considerable pain. On initial examination of the right lower leg revealed some bullae on the diffuse redness with sharply defined raised borders and increased temperature in surrounding area. Movements of the leg was reduced and associated with severe pain. Neurological and vascular examination of the limb was satisfactory.

The initial blood investigation revealed white cell count (WBC) count of $36.78 \times 10^9/L$, C-reactive protein (CRP) of 432.2 mg/L, hemoglobin (Hb) level of 14.8 g/dL, serum sodium (Na) of 135 mmol/L, glucose level of 5.4 mmol/L, serum creatinine of 131 mmol/L. X-ray of the right lower limb did not show gas in the soft tissues. We ruptured and drained the patient's bullae, a culture of the contained fluid grew only Group A streptococci.

Intravenous ciprofloxacin and cefoperazone was continued for 7 days. Even after 7 days of antibiotics patient remained symptomatic. Although he remained febrile at all times, his pain and tenderness continued to increase in distal leg. Erythematous areas turn into a dusky blue color with formation of hemorrhagic bullae and non-pitting edema extending outside the erythematous patches. Considering the failure to respond with intravenous antibiotics, changing in clinical signs and symptoms, increasing tenderness in right lower limb and patient's Laboratory Risk Indicator for necrotizing fasciitis (LRINEC) score was 6 a suspicion of necrotizing fasciitis was made. Patient was taken to theatre urgently and fasciotomy performed through antero-medial approach of the right lower leg which confirmed the diagnosis findings of necrotic fascia and muscles.

Intra-venous (IV) clindamycin was added along with ciprofloxacin and cefoperazone. IV hydration and oxygen therapy was maintained through out, with close observation of renal functions which remained stable.

Patient remained stable systemically and responded well to the treatment as evidenced by normalizing inflammatory markers. The diagnosis was confirmed on tissue histology. Patient was discharged home after 20 days of in-hospital stay. At final follow up 2 months later he had full range of motion in his right lower limb.

Discussion

Necrotizing fasciitis (NF) is a rare, life-threatening, soft-tissue infection characterized by rapidly spreading inflammation and necrosis of the skin, subcutaneous fat, and fascia [1]. Dermal and hypodermal tissue are severely infiltrated by inflammatory cells (mainly neutrophils) [6]. Epidermal and muscular structures are typically not involved. However, infection can spread to underlying muscles, resulting in myonecrosis. Hemorrhagic bullae can develop as infection progresses [7].

There are two main groups of necrotizing fasciitis depending on microbiology. Type-I NF are polymicrobial and most common bacterial species

include Gram-positive cocci (streptococci, staphylococcal species), enterococci and gram-negative enterobacteriaceae (*Escherichia coli*, *Acinetobacter* species, *Pseudomonas* species and *Klebsiella* species). *Bacteroides* species are the most common anaerobes, while *Clostridial* species are an infrequent isolate [8-12]. Type I infections are often diagnosed in immunocompromised patients and tend to occur in the perineal and trunk areas [13]. **Type-II** infections are monomicrobial and usually caused by group A *Streptococcus* (*Streptococcus pyogenes*) either alone or in association with *Staphylococcus aureus*. Streptococcal infection can be associated with toxic shock syndrome. Necrotizing fasciitis of the extremities and limbs is usually type 2 [14]. *Streptococcus* seems to be responsible for fulminating presentations. Pathogenesis of these infections has been well studied and virulent factors identified. These include factors that mediate attachment to host cells like protein F, lipoteichoic acid and M protein.

Pathophysiology

Necrotizing fasciitis is characterized by rapidly spreading infection in the subcutaneous tissues. Microbial invasion of the subcutaneous (SC) tissues occurs either through external trauma or direct spread from a perforated viscus (particularly colon, rectum, or anus). Bacteria then track SC, producing endo and exotoxins [15] that cause micro-vascular thrombosis [16], tissue ischemia, liquefactive necrosis, and often systemic illness [17] which can progress to septic shock, multisystem organ dysfunction, and death. Tissue ischemia, impedes oxidative destruction of bacteria by polymorphonuclear cells and prevents adequate delivery of antibiotics.

Etiology and risk factors

Reported etiologies of soft-tissue injury leading to necrotizing fasciitis include blunt or penetrating trauma, direct inoculation of the subcutaneous tissue from a superficial site and hematogenous spread from a distant site. Occurring in any region of the body, NF most commonly involves the abdominal wall, extremities and perineum (Fournier gangrene) [21].

With a susceptibility to cutaneous infections, diabetics are at high risk to develop NF. In the lower limbs, development of NF is enhanced by vascular and lymphatic stasis. Patients with peripheral vascular disease present a risk of NF development. Type 1 fasciitis commonly occurs in patients with immunosuppression [21].

Clinical features

Lack of cutaneous findings early in the disease make the diagnosis challenging. The most common

early signs are erythema, local warmth, skin induration and edema. These signs often make early diagnosis difficult and the condition is often diagnosed as cellulites, abscess or septic arthritis as in our case. Later, diagnosis of a necrotizing infection is based on the association of local and systemic signs and symptoms: an exquisite pain, completely disproportionate compared to clinical findings, as key symptom [20].

At this state, the local signs most commonly associated with NF are [18]:

- a rapidly progressive edema, overstepping widely the inflammatory area.
- bullae filled with serous fluid becoming hemorrhagic or purulent, sometimes malodorous.
- blue-grey cyanotic lesions (associated with thrombosis of cutaneous perforating vessels) and pale ischemic areas.
- an erythema without distinct borders (in contrast to erysipelas).

Pain out of proportion to the apparent severity of the lesion should alert the physician to the possible diagnosis of Necrotizing fasciitis. Patches of skin necrosis, tissue crepitus, fluctuance and systemic evidence of sepsis such as hyperthermia, tachycardia and hypotension are alarming signs.

Diagnosis

NF remains a diagnosis challenge, requiring laboratory, microbiology and radiologic investigations. Routine laboratory, including white blood cell count and C-reactive protein level, may distinguish NF from non-necrotic soft tissue infection. CPK concentration is also useful as marker of muscular necrosis. With an high index of suspicion (crepitus at physical examination), a clinical diagnosis of NF can be made. Wong et al. [19] have recently developed a Laboratory Risk Indicator for necrotizing fasciitis (LRINEC) (table1). The total score had a range of 0–13, and Wong and colleagues showed that, for intermediate and high-risk patients (score,>6) had a positive predictive value (PPV) of 92% and a negative predictive value (NPV) of 96% [19].

Hemoculture and aspirates of cutaneous lesions or bullae with a thin needle are often sterile, due to a prior antibiotherapy. Peroperative culture with antibiogram leads to bacterial species identification and to adequate antibiotic treatment. Biopsy is also helpful as an alternative diagnostic method. Radiologic studies are also contributive but should never delay appropriate surgical treatment of highly suspicious cases. Radiographies are more sensitive to detect gas than physical examination [22]. Other

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diagnostic adjuncts like ultrasound, CT scan and magnetic resonance imaging (MRI) are helpful in suspicious cases.

Treatment

The gold standard of treatment for NF includes intravenous antibiotics with broad-spectrum antibacterial coverage, prompt surgical debridement, and supportive care involving hemodynamic support, wound care, and nutritional support [1-5, 23-32].

Surgery

Surgery should be performed within 24 h of presentation as the mortality rate significantly increases beyond this period [11]. Early debridement of all necrotic tissues and drainage of involved fascia planes via extensive fasciotomy. Re-exploration and further debridements are often required to achieve control of the necrotizing process. Fasciotomy may be performed at the time of debridement [25, 33]. If infection progresses despite serial debridements and antibiotics, amputation may be life saving.

Antibiotics and supportive care

A combination of broad spectrum antibiotics, such as a penicillin, an aminoglycoside or third generation cephalosporin, and clindamycin or metronidazole, are typically employed to provide broad bacterial coverage. Extended coverage with vancomycin has to be added for nosocomial infections (*Pseudomonas* spp, highly resistant gram-positive bacteria) or surgical wound contamination. For type 2 fasciitis, high-dose penicillin remains the best option. Animal and human studies suggest beneficial effects of adding clindamycin in type 2 NF because clindamycin may suppress production of exotoxins and/or virulence factors and enhance phagocytosis by inhibiting M-protein synthesis [34]. This should be accompanied with supportive measures such as fluid replacement, blood pressure support, analgesia, nutritional support and intensive care involvement etc. Intravenous immunoglobulins (IVIg) [35, 36] and Hyperbaric oxygen (HBO) therapy has also been suggested as an adjunct to other treatments. There is no agreement as to the usefulness of HBO for NF [36,37].

Conclusion

NF is a rare, rapidly progressive, soft-tissue infection characterized by extensive necrosis of the skin and subcutaneous tissue. Only early identification of the necrotizing process can improve the outcome of this disease. NF should be suspected in every skin infection with fever, signs of systemic toxicity and severe pain. Immediate surgical intervention, and broad-spectrum antibiotics are critical. Surgical debridement and biopsy is the gold standard for

diagnosing and treating necrotizing fasciitis. However, other adjuvant therapies have been considered, such as hyperbaric oxygen and intravenous immunoglobulin. Despite antibiotic therapy and surgical intervention, the mortality and morbidity of NF remain high.

Table 1

Laboratory Risk Indicator for Necrotizing Fasciitis Score (19)

| Variable | Score |
|------------------------------|-------|
| C-reactive protein | |
| <150 | 0 |
| ≥150 | 4 |
| WBC (cells/mm ³) | |
| <15 | 0 |
| 15-25 | 1 |
| ≥25 | 2 |
| Hemoglobin (g/dL) | |
| >13.5 | 0 |
| 11-13.5 | 1 |
| <11 | 2 |
| Sodium (mmol/L) | |
| ≥135 | 0 |
| <135 | 2 |
| Creatinine (mcg/L) | |
| ≤141 | 0 |
| >141 | 2 |
| Glucose (mmol/L) | |
| ≤10 | 0 |
| >10 | 1 |

References

1. Kotrappa K.S. Necrotizing fasciitis / K.S. Kotrappa, R.S. Bansal, N.M. Amin // *Am. Fam. Phys.* – 1996. – Vol. 53, N 5. – P. 1691-1696.
2. Gonzalez M.H. Necrotizing fasciitis and gangrene of the upper extremity / M.H. Gonzalez // *Hand Clin.* – 1998. – Vol. 14, N 4. – P. 635-645.
3. File T.M. Diagnosing and treating the "Flesh Eating Bacteria Syndrome" / T.M. File, J.S. Tan, J.R. Dipersio // *Clev. Clin. J. Med.* – 1998. – Vol. 65, N 5. – P. 241-249.
4. Gannon T. Dermatologic emergencies / T. Gannon // *Postgrad. Med.* – 1994. – Vol. 96, N 1. – P. 67-82.
5. Necrotizing fasciitis in the lower extremity: A review and case presentation / [C.M. Lombardi, L.M. Silver, K.K. Lau et al.] // *J. Foot. Ankle. Surg.* – 2000. – Vol. 39, N 4. – P. 244-248.
6. Fulminant group A streptococcal necrotizing fasciitis: clinical and pathologic findings in 7 patients / [P.R. Dahl, C. Perniciaro, K.A. Holmkvist et al.] // *J. Am. Acad. Dermatol.* – 2002. – Vol. 47. – P. 489-492.
7. Buchanan C.S. Necrotizing fasciitis due to group A Beta-Hemolytic streptococci / C.S. Buchanan, J.R. Haserick // *Arch. Dermatol.* – 1970. – Vol. 101. – P. 664-668.
8. Elliott D. The microbiology of necrotizing soft tissue infection / D. Elliott, J.A. Kufera, R.A. Myers // *Am. J. Surg.* – 2000. – Vol. 179, N 5. – P. 361-366.

9. Necrotizing fasciitis: current concepts and review of the literature / B. Sarani, M. Strong, J. Pascual, C.W. Schwab // J. Am. Coll. Surg. – 2009. – Vol. 208, N 2. – P. 279-288.
10. Determinants of mortality for necrotizing soft-tissue infections / C.R. McHenry, J.J. Piotrowski, D. Petrinic, M.A. Malangoni // Ann. Surg. – 1995. – Vol. 221. – P. 558-563.
11. Necrotizing fasciitis: clinical presentation, microbiology and determinants of mortality / [C.H. Wong, H.C. Chang, S. Pasupathy et al.] // J. Bone Joint. Surg. Am. – 2003. – Vol. 85, N 8. – P. 1454-1460.
12. Necrotizing fasciitis: a fourteen-year retrospective study of 163 consecutive patients / [B.J. Childers, L.D. Potyondy, R. Nachreiner et al.] // Am. Surg. – 2002. – Vol. 68. – P. 109-116.
13. Necrotizing fasciitis in southeast Taiwan: clinical features, microbiology, and prognosis / [M.J. Bair, H. Chi, W.S. Wang et al.] // Int. J. Infect. Dis. – 2009. – Vol. 13, N 2. – P. 255-260.
14. Necrotizing fasciitis due to group A streptococci in western Norway: incidence and clinical features / J. Chelsom, A. Halstensen, T. Haga, E.A. Hoiby // Lancet. – 1994. – Vol. 344. – P. 114-115.
15. Hackett S.P. Streptococcal toxic shock syndrome: synthesis of tumor necrosis factor and interleukin-1 by monocytes stimulated with pyrogenic exotoxin A and streptolysin O / S.P. Hackett, D.L. Stevens // J. Infect. Dis. – 1992. – Vol. 165. – P. 879-885.
16. Stamenkovic I. Early recognition of potentially fatal necrotizing fasciitis. The use of frozen-section biopsy / I. Stamenkovic, P.D. Lew // N. Engl. J. Med. – 1984. – Vol. 310. – P. 1689-1693.
17. Cainzos M. Necrotizing soft tissue infections / M. Cainzos, F.J. Gonzalez-Rodriguez // Curr. Opin. Crit. Care. – 2007. – Vol. 13. – P. 433-439.
18. Bruin-Buisson C. Strategies de prise en charge des fasciites necrosantes / C. Bruin-Buisson // Ann. Dermatol. Venerol. – 2001. – Vol. 128. – P. 394-403.
19. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections / [C.H. Wong, L.W. Khin, K.S. Heng et al.] // Crit. Care Med. – 2004. – Vol. 32. – P. 1535-1541.
20. Smeets L. Necrotizing Fasciitis: Case Report and Review of Literature / L. Smeets, A. Bous, O. Heymans // Actachir. Belg. – 2007. – Vol. 107. – P. 29-36.
21. Necrotizing fasciitis. Importance of roentgenographic studies for soft-tissue gas / J.R. Fisher, M.J. Co Way, R.T. Takeshita, M.R. Sandoval // MR JAMA. – 1979. – Vol. 241. – P. 803-806.
22. Tsai E. Hand infections in the trauma patient / E. Tsai, J.M. Failla // Hand Clin. – 1999. – Vol. 15, N 2. – P. 373-386.
23. Sadick N.S. Current aspects of bacterial infections of the skin / N.S. Sadick // Derm. Clinics. – 1997. – Vol. 15, N 2. – P. 341-349.
24. Green R.J. Necrotizing fasciitis / R.J. Green, D.C. Dafoe, T.A. Raffin // Chest. – 1996. – Vol. 110, N 1. – P. 219-229.
25. Necrotizing fasciitis: A clinical, microbiologic, and histopathologic study of 14 patients / I.J. Umbert, R.K. Winkelmann, G.F. Oliver, M.S. Peters // JAAD. – 1989. – Vol. 20, N 5. – P. 774-781.
26. Chapnick E.K. Necrotizing soft tissue infections / E.K. Chapnick, E.I. Abter // Inf. Dis. Clin. Nor. Am. – 1996. – Vol. 10, N 4. – P. 835-855.
27. Jarrett P. The clinical spectrum of necrotizing fasciitis. A review of 15 cases / P. Jarrett, M. Rademaker, M. Duffill // Aust. N. Z. J. Med. – 1997. – Vol. 27, N 1. – P. 29-34.
28. Brenner B.E. Necrotizing fasciitis / B.E. Brenner, M. Vitullo, R.R. Simon // Ann. Emerg. Med. – 1982. – Vol. 11, N 7. – P. 384-386.
29. Childs S.G. Necrotizing fasciitis: Challenging management of a septic wound / S.G. Childs // Ortho Nurs. – 1999. – Vol. 18, N 2. – P. 11-19.
30. Callahan E.F. Cutaneous (Non HIV) infections / E.F. Callahan, K.A. Adal, K.J. Tomecki // Derm. Clin. – 2000. – Vol. 18, N 3. – P. 497-508.
31. Leitch H.A. Necrotizing fasciitis secondary to group A Streptococcus / H.A. Leitch, A. Palepu, C.M.B. Fernandes // Can. Fam. Phys. – 2000. – Vol. 46. – P. 1460-1466.
32. Meltzer D.L. Necrotizing fasciitis: A diagnostic challenge / D.L. Meltzer, M. Kabongo // Am. Fam. Phys. – 1997. – Vol. 56, N 1. – P. 145-149.
33. Mulla Z.D. Treatment options in the management of necrotizing fasciitis caused by Group A Streptococcus / Z.D. Mulla // Expert. Opin. Pharmacother. – 2004. – Vol. 5. – P. 1695-1700.
34. Darabi K. Current usage of intravenous immune globulin and the rationale behind it: the Massachusetts General Hospital data and a review of the literature / K. Darabi, O. Abdel-Wahab, W.H. Dzik // Transfusion. – 2006. – Vol. 46. – P. 741-753.
35. Schrage B. Different preparations of intravenous immunoglobulin vary in their efficacy to neutralize streptococcal superantigens: implications for treatment of streptococcal toxic shock syndrome / B. Schrage, G. Duan, L.P. Yang // Clin. Infect. Dis. – 2006. – Vol. 43. – P. 743-746.
36. Korhonen K. Hyperbaric oxygen therapy in acute necrotizing infections with a special reference to the effects on tissue gas tensions / K. Korhonen // Ann. Chir. Gynaecol. – 2000. – Vol. 214. – P. 7-36.
37. Jallali N. Hyperbaric oxygen as adjuvant therapy in the management of necrotizing fasciitis / N. Jallali, S. Withey, P.E. Butler // Am. J. Surg. – 2005. – Vol. 189. – P. 462-466.

НЕКРОТИЗУЮЧИЙ ФАСЦІЇТ – ПОВІДОМЛЕННЯ ВИПАДКУ Й ОГЛЯД ЛІТЕРАТУРИ

І.Х. Юнгарева, В.В. Ніколов, С.С. Маріна

РЕЗЮМЕ. Наведено дані про некротизуючий фасціїт (НФ) – тяжке бактерійне запалення м'яких тканин, яке характеризується некрозом шкіри, підшкірної клітковини та фасції. Цей рідкісний стан супроводжується високою смертністю і вимагає своєчасної діагностики та термінового лікування з використанням антибіотиків широкого спектру дії, гемодинамічної підтримки, хірургічної обробки некротичної ділянки (некректомія) та якісного догляду за раною. Описано випадок 63-річного чоловіка, якого спочатку було прийнято в госпіталь з діагнозом бешиха, а згодом діагностовано некротичний фасціїт з типовим перебігом.
Ключові слова: некротизуючий фасціїт (НФ), інфекції шкіри, підшкірної клітковини і м'яких тканин.

Отримано 12.02.2013 р.