AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Infections in the Drug Abuser

Infections in the injection drug user can vary from benign to life threatening. The lecturer will discuss the simple and complex soft tissue infections associated with injection drug use, as well as infective endocarditis. Emphasis will be on the rapid evaluation and emergency department management of these infections. Criteria for specialty referral and/or outpatient follow-up will be presented.

- Differentiate simple soft tissue infections from life- or limb-threatening infections.
- Review the clinical presentations of infective endocarditis.
- Discuss the importance of rapid evaluation and management of these infections.
- Identify the criteria for specialty consultation and admission versus outpatient follow-up.

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FACULTY

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Infections in the Drug Abuser

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I. Objectives:

- Differentiate simple soft tissue infections from life- or limb-threatening infections.
- Review the clinical presentations of infective endocarditis.
- Discuss the importance of rapid evaluation and management of these infections.
- Identify the criteria for specialty consultation and admission versus outpatient follow-up.

II. Introduction:

A. Spectrum of problem

There is an estimated 1.4 to 3.5 million injection drug users (IDUs) in the United States. However, this number is only an estimation as drug users represent a hidden population for various social and legal factors. Similarly, the number of infectious complications associated with injection drug use is difficult to accurately determine. Current figures are usually based on studies of limited geographic areas or other small populations. AIDS is of major concern among health officials and practitioners, and it is estimated that two-thirds of IDUs are HIV positive. A whole host of other infectious diseases are spread by injection drug use including infectious hepatitis (not just B) and other viral illnesses such as HTLV, HSV, VZV, and CMV. Sexually transmitted diseases may be contracted by sharing needles, but the entire realm of drugs-for-sex has created a setting for unchecked spread of these diseases. Tuberculosis was thought to be on the way to eradication until the AIDS epidemic came about.

B. Are injection drug users immunocompromised?

It remains unclear if injection drug use by itself leads to immunocompromise. There is evidence that alcohol supresses immune function, and animal studies indicate that other drugs such as cocaine and heroin may do the same. Sharing needles can transmit blood borne pathogens, and usage of inhalation paraphernalia by multiple users similarly spreads diseases. Other lifestyle issues associated with injection drug use may also contribute to an altered immune response. Being homeless or living in crowded or communal settings, having inadequate nutrition, and stress most likely all play a role in the immune function of IDUs.

III. Subcutaneous Abscess

A. Etiology/Appearance

Abscesses can occur on any area of the body. In the injection drug user, any site of injection may become infected – this may migrate in a patient over time as more accessible peripheral sites are used up and less accessible central sites are used. Abscesses involving the perineal region commonly contain anaerobic *Bacteroides fragilis*, whereas the most common organism in cutaneous abscesses elsewhere is aerobic *Staphylococcus aureus*. Most patients complain of pain and swelling about the abscess and very rarely show systemic toxicity. Most cutaneous abscesses are red, swollen, and tender to palpation with some degree of fluctuance (depending on location and age of the abscess). Needle aspiration can help differentiate cellulitis from abscess.

B. Treatment

In healthy, non-toxic patients, incision and drainage alone is adequate. In the afebrile patient, there is evidence that incision and drainage does not cause bacteremia. If a patient is immunocompromised and/or septic, then appropriate Gram stains and cultures followed by antibiotics are necessary.

IV. Cellulitis

A. Etiology

Cellulitis is an inflammation of the dermis and subcutaneous tissues characterized by induration, tenderness, and erythema. The causative agents in the immunologically intact host are group A β-hemolytic streptococci and less often *Staph aureus*.
Multiple pathogens must be considered in the immunocompromised host.
Recent reports indicate a change in the virulence of group A strep and all health care providers must be vigilant of the potentially fatal, rapidly progressing infection.

B. Clinical Appearance

Patients may have little or no systemic manifestations accompanying their cellulitis, may have regional adenopathy or lymphangitis, may be febrile with generalized malaise, or may present in fulminant septic shock. Multiple cellulitic areas indicate a hematogenous origin.

C. Treatment

Culture of the lesions by needle aspiration or punch biopsy may be helpful as may blood cultures. Empiric therapy to cover staph and strep is wise. The decision of oral or parenteral treatment is based on clinical findings. A penicillinase stable agent such as a first generation cephalosporin is acceptable.

V. Necrotizing Fasciitis

A. Nomenclature

1. Confusion arising from historical derivations (pathogens, site, circumstances, eponyms, etc.)
2. Preferred is "necrotizing fasciitis"
3. Multiple organisms may be present – group A strep, staph, anerobes

B. Presentation

1. Can affect any body part - most common on extremeties, abdominal wall, perianal/groin, postop wounds, IDU sites
2. Initially erythematous, swollen, exquisitely tender and painful
3. Rapid progression from red-purple to blue-gray with bullae
4. Anesthetic due to thrombosis of small vessels and superficial nerve destruction
5. Subcutaneous gas
6. Systemic toxicity with fever of 102-105^{o}F
7. Bedside exploration -look for undermining of skin and "dishwater" fluid
8. Rapid gram stain of fluid (aspirate or spontaneous)
9. Role of ultrasound, CT, MRI evolving
10. Mortality 20-47% overall

C. Management

1. Fluids
2. Monitoring
3. Antibiotics
   - Triple coverage (such as ampicillin/gentamycin/[clindamycin or metronidazole])
4. Role of hyperbaric oxygen
   – remains controversial (not to delay surgery)
5. Surgical drainage/debridement
   – early and typically multiple

VI. Gas Gangrene

C. Etiology

Gas gangrene is synonymous with clostridial myonecrosis which is an acute, rapidly progressive infection manifesting as gas production in the soft tissues, muscle necrosis, and systemic toxicity. *Clostridium perfringens* is the most common organism, but several other clostridial species have been the cause. Infection is known to follow trauma, surgery, burns, and IDU, and has been associated with malignancies.

B. Clinical Course

After inoculation, a two day incubation precedes pressure or heaviness followed rapidly by pain and tachycardia. The wound may drain dark, serosanguinous fluid followed by the development of subcutaneous emphysema. If no treatment is begun within 48 hours of the onset of systemic symptoms, mortality is 100%.

C. Treatment

Appropriate management of the ABC's and fluid resuscitation should begin immediately. A three component therapy must be instituted simultaneously based on practical availabilities:

1) Antibiotics (clindamycin, metronidazole, or chloramphenicol) have been proven beneficial.

2) Surgical debridement is also helpful, but the timing and extent of surgery is controversial – if a compartment syndrome is suspected, immediate fasciotomy is necessary.

3) Hyperbaric oxygen therapy may help stop proliferation of the clostridia, limit tissue necrosis, and better delineate healthy from non-viable tissue.

Tetanus prophylaxis should be administered.

VII. Endocarditis

A. Etiology / Pathogenesis

1. To develop infective endocarditis, there must be a combination of bacteremia and usually some valvular abnormality. However, in IDUs the majority of patients with endocarditis have no known heart disease as seen in the following table of preexisting cardiac lesions:

	Adults 15-50 years (%)	Adults: IDUs (%)
No known heart disease	10-20	50-60
Congenital heart disease	20-30	10
Mitral valve prolapse	10-30	10-20
Rheumatic heart disease	5-10	10
Previous cardiac surgery	10-20	10-20
Previous endocarditis	5	15-25

The valves involved in IDUs tend to be right sided, with the tricuspid valve being most frequently affected.

2. Typical Organisms

Common organisms that cause endocarditis in IDUs include:

Staph aureus (50-60%)
Strep
Entercocci
Gram-negative bacilli
Fungi
Miscellaneous
Culture negative

B. Clinical Presentation

1. Symptoms

The most consistent complaints are fever (in 80-85% of patients), chills, weakness/malaise, sweats, dyspnea, anorexia, weight loss, and cough. Headache, chest pain, abdominal pain, back pain, nausea/vomiting, and myalgia/arthralgia occur less commonly. Stroke is a complaint in up to 20% of patients.

2. Signs

Fever usually greater than 102^{o}F is present in about 90% of patients. Similarly, a murmur is usually heard although a new or changing murmur is infrequent. Splenomegaly may be present. Skin findings including Osler's nodes, splinter hemorrhages, petechiae, and Janeway lesions occur variably. The retinal lesion of endocarditis (Roth spot) can be seen in less than 10% of cases. Other signs may be noted due to embolic events to the spleen, brain, kidneys, bowel, or heart.

C. Diagnosis

The criteria for establishing a diagnosis of infective endocarditis include pathologic and clinical criteria. By combining major and minor criteria, cases of infective endocarditis can now be classified as definite, possible, or rejected. Among the clinical criteria, there must be either 2 major, 1 major and 3 minor, or 5 minor criteria present to be considered a definite diagnosis.

Major Criteria

1. Positive blood culture
2. Evidence of endocardial involvement
    a. Positive echocardiogram for infective endocarditis
        1) oscillating intracardiac mass
        2) abscess
        3) new partial dehiscence of prosthetic valve

or b. New valvular regurgitation

Minor Criteria

1. Predisposition
   - predisposing heart condition or intravenous drug use
2. Fever > 38.0°C (100.4°F)
3. Vascular phenomena
   - major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, Janeway lesions
4. Immunologic phenomena
   - glomerulonephritis, Osler's nodes, Roth spots, rheumatoid factor
5. Microbiologic evidence
6. Echocardiogram
   - consistent with infective endocarditis but not meeting major criterion

By using the clinical criteria indicated above, it might be possible to make a definite diagnosis of infective endocarditis in the ED.

D. Prognosis / Complications

In general, IDUs have a better prognosis (i.e. lower mortality) with infective endocarditis than the "usual" patient with endocarditis, although this does not appear to hold true in HIV cases. Acute staphylococcal endocarditis tends to have a less severe course in addicts versus non-addicts. Cardiac failure is the most important prognostic factor, and delay in diagnosis and treatment may lead to its development. Other complications include neurologic, renal, splenic, and peripheral septic emboli events.

E. Treatment

Infective endocarditis does not improve without therapy. Early and appropriate antimicrobial therapy has the best chance of affecting a cure. The agents and their dose, route, and duration are based on the infecting organism(s) either empirically or culture-proven. In most IDUs, since the likelihood of staph is high, an initial regimen containing a penicillinase-stable penicillin, a first generation cephalosprin, or vancomycin is indicated. Further therapy should proceed in consultation with an infectious disease specialist and/or cardiologist as treatment can be complex and prolonged. Management of cardiac and extra-cardiac complications should also occur in tandem with antibiotic therapy. Early cardiac surgery may be necessary if acute cardiac compromise is present.

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