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EDWARDSIELLA INFECTIONS E. tarda is the only member of the genus Edwardsiella that is associated with human disease. This organism is found predominantly in freshwa ter and marine environments and in the associated aquatic animal spe cies. Human acquisition occurs primarily from interaction with these reservoirs or ingestion of raw or inadequately cooked aquatic animals. E. tarda infection is rare in the United States, where acquisition occurs mainly along the Gulf of Mexico; recently reported cases are mostly from Asia. This pathogen shares clinical features with Salmonella spe cies (as an intestinal pathogen; Chap. 171), Vibrio vulnificus (as an extraintestinal pathogen; Chap. 173), and Aeromonas hydrophila (as both an intestinal and an extraintestinal pathogen; Chap. 173).

■ ■INFECTIOUS SYNDROMES Gastroenteritis is the predominant Edwardsiella-associated infectious syndrome (50–80% of reported cases). Self-limiting watery diarrhea is most common, but severe colitis also occurs. The most common extraintestinal infection is wound infection due to direct inoculation, which is often associated with brackish or freshwater injuries, snake bites, or fish-related trauma. A case of pneumonia occurred after a near-drowning incident. Cholecystitis, cholangitis, and hepatic abscess may be due to ascending infection via the biliary tree. Other infec tious syndromes result from invasion of the gastrointestinal tract and subsequent bacteremia. A primary bacteremic syndrome, sometimes complicated by meningitis, has a 40% case–fatality rate; hematogenous seeding may result in hepatic and intra- and extraperitoneal abscesses, endocarditis, mycotic aneurysm, septic arthritis, osteomyelitis, nec rotizing fasciitis, and empyema. Most hosts who develop systemic Edwardsiella infection have significant comorbidities (e.g., hepatobili ary disease, iron overload, cancer, or diabetes mellitus). PART 5 Infectious Diseases ■ ■DIAGNOSIS Although E. tarda can readily be isolated and identified, most laborato ries do not routinely screen for or identify it in stool samples. Produc tion of hydrogen sulfide is a characteristic biochemical property. TREATMENT Edwardsiella Infections E. tarda is susceptible to most antimicrobial agents appropriate for use against GNB. Gastroenteritis is generally self-limiting, but treat ment with a fluoroquinolone may hasten resolution. In the setting of severe sepsis, fluoroquinolones, third- and fourth-generation cephalosporins, carbapenems, and amikacin—either alone or in combination—are the safest choices pending susceptibility data. INFECTIONS CAUSED BY MISCELLANEOUS GENERA Other gram-negative organisms such as Hafnia, Kluyvera, Cedecea, Pantoea, Ewingella, Leclercia, Raoultella, and Photorhabdus spp. are occasionally isolated from diverse clinical specimens, including blood, sputum, urine, cerebrospinal fluid, joint fluid, bile, and wounds. Such organisms cause infection predominantly in compromised hosts or in association

with an invasive procedure or foreign body. Cephalo sporinases from Kluyvera have been implicated as the progenitors of CTX-M ESBLs. Kluyvera and Raoultella may produce carbapenemases. ■ ■FURTHER READING Antimicrobial Resistance Collaborators: Global burden of bac terial antimicrobial resistance in 2019: A systematic analysis. Lancet 399:629, 2022. [Erratum in Lancet 400:1102, 2022.] Bonten M et al: Epidemiology of Escherichia coli bacteremia: A sys tematic literature review. Clin Infect Dis 72:1211, 2021. Cheng MP et al: Beta-lactam/beta-lactamase inhibitor therapy for potential AmpC-producing organisms: A systematic review and meta-analysis. Open Forum Infect Dis 6:ofz248, 2019. David S et al: Epidemic of carbapenem-resistant Klebsiella pneumoniae in Europe is driven by nosocomial spread. Nat Microbiol 4:1919, 2019.

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Acinetobacter Infections ■ ■DEFINITION Acinetobacter species were first described in 1911 and named Micro coccus calcoaceticus. Thereafter, the genus was renamed multiple times; since 1950, it has been known as Acinetobacter. Acinetobacter species are gram-negative, oxidase-negative, nonmotile, nonferment ing coccobacilli that are easily recovered on standard culture media. Differentiation among Acinetobacter species on the basis of phenotypic characteristics alone is very difficult. Molecular-based methods such as matrix-assisted laser desorption–ionization–time-of-flight mass spec trometry (MALDI-TOF-MS) and quantitative real-time polymerase chain reaction (PCR) are usually necessary to identify Acinetobacter baumannii, the most clinically relevant species of the genus. ■ ■ETIOLOGY AND EPIDEMIOLOGY Acinetobacter species are naturally encountered in water and soil and have also been recovered from fruits and vegetables. In humans, Acinetobacter can be found on the skin and in the respiratory and gastrointestinal tracts. A. baumannii is capable of surviving environ mental desiccation for weeks; this characteristic is important from an infection-control perspective as it allows this organism to persist in the hospital environment and on equipment. Acinetobacter was historically considered a pathogen of hot and humid climates. In recent years, however, hospital outbreaks caused by A. baumannii have been reported worldwide, even in temperate climates. In the United States, the Centers for Disease Control and Prevention (CDC) estimates that 12,000 Acinetobacter infections occur every year, 7300 of which are caused by multidrug-resistant strains, with 500 attributable deaths. The increase

in the number of infections with A. baumannii is suspected to be due to the rapid spread of certain genetically distinct lineages; of the three international clonal lineages (ICLs), ICL I and ICL II are multidrug resistant. The predominance of these lineages remains unexplained, although it has been proposed that this population structure is the result of two waves of expansion.

The first wave followed a bottleneck (possibly linked to a restricted ecologic niche) that occurred in the distant past. The second wave is ongoing and is being driven by the rapid expansion of a limited num ber of multidrug-resistant clones. The COVID-19 pandemic resulted in a setback to the efforts to control the spread of multidrug-resistant organisms, with significant increases in the rates of infections with carbapenem-resistant Acinetobacter reported worldwide. Analysis of the A. baumannii pangenome (the sum of the core and dispensable genomes) has shown that its organization is charac terized by a small core genome and a large accessory or disposable genome. This organization reflects A. baumannii's high plasticity, which enables it to acquire exogenous genetic material. With few exceptions, gene functions associated with virulence are found in the core genome; this observation suggests a limited role for the acquisi tion of new virulence traits in the recent nosocomial expansion of A. baumannii clones. Genes associated with resistance to antimicrobial agents are found in both the species core genome and the accessory genome. In the accessory genome, these genes have been found in alien islands, often flanked by integrases, transposases, or insertion sequences. This pattern suggests possible acquisition by horizontal gene transfer from other Acinetobacter strains or even from different bacterial species present in the immediate environment. Acquisition of these antimicrobial resistance genes is hypothesized to have led to the recent rapid expansion of highly homogeneous clonal lineages, whose main difference from nonclonal A. baumannii appears to be their anti microbial resistance. Health Care–Associated Infections Infections caused by A. baumannii occur frequently among patients admitted to intensive care units (ICUs). Risk factors for colonization and infection with this pathogen include nursing home residence, prolonged ICU stay, central venous catheterization, tracheostomy, mechanical ventilation, enteral feedings, and treatment with third-generation cephalosporins, fluoro quinolones, and carbapenems. Acquisition of carbapenem-resistant A. baumannii is most common among patients exposed to carbapenems. Spread of A. baumannii across different regions is facilitated by the movement of patients between health care systems and throughout the continuum of health care. Within the hospital, environmental spread of A. baumannii occurs as a result of inappropriate hand hygiene among workers providing health care for infected or colonized patients and the contamination of hospital equipment, such as respiratory therapy and ventilation equipment. The air surrounding the patient may also play a role in environmental colonization with A. baumannii, especially in inpatient areas without physical barriers between patients and with an inadequate number of air exchanges. A. baumannii strains identified during hospital outbreaks are typi cally resistant to more antibiotic classes than strains from the commu nity. The prevalence of colonization with A. baumannii at the time of admission or during a stay in a long-term acute-care hospital (LTACH) or nursing home is variable and depends on regional flora. Outbreaks of A. baumannii in acute-care hospitals and LTACHs that "share" patients have been described in Ohio, Michigan, Illinois, and Indiana. Community-Acquired Infections Community-acquired infec tions caused by Acinetobacter have been described in Australia and Asia. Few cases have been reported in regions with a temperate climate, and even those few cases have taken place during warm and humid months. Risk factors for community-acquired pneumonia due to this organism include a history of alcohol abuse, diabetes mellitus, smok ing, and chronic lung disease. War Zone–Associated Infections Infections caused by Acineto

bacter in war zones include skin and soft tissue infections associated with traumatic injuries and bloodstream infections. Outbreak investi gations of A. baumannii infections among military personnel returning from Iraq and Afghanistan suggested the acquisition of A. baumannii in field hospitals rather than colonization of the skin before an injury. This view is supported by the recovery of A. baumannii isolates with similar genetic characteristics from inanimate surfaces in field hospi tals and from patients.

Disaster Medicine A. baumannii is linked to infections among victims of trauma during tsunamis, earthquakes, and terrorist attacks. The types of infections most frequently observed in these settings are soft tissue injuries, but bloodstream infections and pneumonia have also been reported. In addition, outbreaks of A. baumannii infection in ICUs caring for disaster victims have been described.

■ ■PATHOGENESIS Mechanisms of pathogenesis and virulence in Acinetobacter spe cies have not been fully elucidated. However, A. baumannii seems to have greater virulence potential than other Acinetobacter species, as evidenced by its ability to grow at 37°C and to resist uptake by macrophages. Initial A. baumannii colonization of the host and the environment is facilitated by the organism’s ability to adhere to surfaces and human cells and to create biofilms. The ability to form a biofilm is phenotypi cally associated with exopolysaccharide production and pilus forma tion. A quorum-sensing molecule encoded by the abaI autoinducer synthase gene has been implicated in A. baumannii biofilm formation on abiotic surfaces. Outer-membrane porins appear to mediate cell apoptosis. A. baumannii can survive in harsh environments within the host and on inanimate surfaces by modifying the structure of its lipid A, with a consequent decrease in susceptibility to antibiotics and antimicrobial peptides and an increase in survival upon desiccation. Acinetobacter species produce an extracellular capsule that protects the bacteria from external threats, including complement-mediated killing. Studies of mouse models showed that Acinetobacter species can increase capsule production in the presence of subinhibitory levels of antibiotic—an ability that leads to increased resistance to complementmediated killing and a hypervirulent phenotype. CHAPTER 167 Phospholipase C and phospholipase D have been identified as viru lence factors in A. baumannii. These enzymes exert cytotoxic effects on epithelial cells and facilitate their invasion. Iron-acquisition systems are also important virulence mechanisms in A. baumannii. Through secretion of siderophores (low-molecularmass ferric-binding compounds), A. baumannii is able to grow despite iron deficiencies in the surrounding environment (e.g., in the human host). Acinetobacter Infections Several protein-secretion systems have been identified in A. bau mannii. The most recently described is a type II secretion system. The substrate for this system, the LipA lipase, is required for growth on medium containing lipids as a sole carbon source. Mutants lack ing the genes for the type II secretion system or its substrate exhibit defective in vivo growth in a neutropenic murine model of bacteremia.

A. baumannii also has a type VI secretion system whose primary function seems to be to secrete antibacterial toxins that kill competing bacteria, including other strains in the same species. The type V autotransporter system has been characterized in

A. baumannii. In a murine systemic model of Acinetobacter infection, the Acinetobacter trimeric autotransporter mediates biofilm formation and maintenance; adherence to extracellular matrix components such as collagen I, II, and IV; and virulence. Outer-membrane vesicles (OMVs) play a

special role in protein secretion. Many A. baumannii strains secrete OMVs containing vari ous virulence factors, including outer-membrane protein A (OmpA), proteases, and phospholipases. The membrane proteins in OMVs are responsible for eliciting a potent innate immune response. Several studies have shown that A. baumannii OMVs could be used as an acel lular vaccine to effectively control A. baumannii infections. Nosocomial strains of Acinetobacter can deploy multiple mecha nisms of resistance, including alterations in porins and efflux pumps and expression of β-lactamases. More specifically, Acinetobacter spe cies can reduce the expression of porins, thus hindering the passage of β-lactam antibiotics into the periplasmic space. These species can overexpress bacterial efflux pumps and decrease the concentration of β-lactam antibiotics in the periplasmic space. Efflux pumps can also actively remove quinolones, tetracyclines, chloramphenicol, disinfec tants, and tigecycline. Acinetobacter species possess chromosomally encoded cephalosporinases and are capable of acquiring β-lactamases,

including serine and metallo-β-lactamases. AmpC β-lactamases are class C β-lactamases intrinsic to all A. baumannii strains. Although these enzymes are expressed at low levels and are not inducible, the addition of the insertion sequence ISAba1 next to the AmpC gene increases β-lactamase production, with resulting resistance to most cephalosporins.

Carbapenem resistance in Acinetobacter species is mostly tied to the emergence of Ambler class D oxacillinases of group 2d, some of which are intrinsic and chromosomal (e.g., OXA-51-like) while others are acquired and are found in plasmids or are chromosomally encoded (e.g., OXA-23-like, 24 [33-like, 40-like], 58-like, 143-like, and 235-like). ■ ■CLINICAL MANIFESTATIONS Pneumonia A. baumannii is a notorious cause of nosocomial pneu monia, most frequently among patients requiring prolonged mechani cal ventilation. The onset of disease tends to be later than that caused by other gram-negative bacilli; however, clinical symptoms of hospitalacquired or ventilator-associated pneumonia due to A. baumannii are similar to those of nosocomial or ventilator-associated pneumonia due to other nosocomial pathogens. Thus, the most common indica tors of infection include fever and increased sputum production. The positivity of respiratory cultures in most cases may present a challenge for the clinician since airway colonization with A. baumannii may not always indicate a diagnosis of pneumonia, but it is a known risk factor for infection itself. In addition, radiologic findings are nonspecific and can include lobar consolidations and pleural effusions, with cavitations being rarely seen. The crude mortality rates associated with nosoco mial pneumonia due to A. baumannii are reported as high as 65%. However, since these infections occur in debilitated patients, their attributable mortality has been difficult to establish. PART 5 Infectious Diseases Community-acquired pneumonia due to A. baumannii is relatively rare. Its clinical presentation is characterized by fever, severe respira tory symptoms, and multiple-organ dysfunction. Patients frequently have a cough productive of purulent sputum, shortness of breath, and chest pain. Imaging studies usually show lobar consolidation. Mortality rates associated with this process are >50%. Bloodstream Infections Bloodstream infections due to A. bau mannii are most frequent among ICU patients and usually occur in the presence of a central venous catheter or as a secondary complication of hospital-acquired or ventilator-associated pneumonia. Polymicrobial growth has been reported in 20–36% of bacteremia episodes. Fever is the most common sign of infection (developing in >95% of cases), and presentation with septic shock and disseminated intravascular coagulopathy has been described in as many as 25 to 30% of patients, respectively. A. baumannii bloodstream infections often result in higher hospitalization costs and longer ICU stays. Crude mortality rates from this infection are as high as 40%; however, rates can

be as high as 70% from infections caused by carbapenem-resistant isolates. In patients with infections caused by extremely drug-resistant strains, poor outcomes are thought to be driven by delays in the initiation of adequate antimicrobial therapy. Skin and Soft Tissue Infections Acinetobacter species have been described as part of the skin flora, yet the majority of the organisms from this genus that colonize the skin are not those associated with nosocomial infections. Discerning infection from wound coloniza tion is challenging. Gunshot wounds and the presence of orthopedic external-fixation devices are common among patients with combat trauma–associated A. baumannii skin and soft tissue infections. The report on a case series of eight U.S. military patients described the clinical presentation of their infections as evolving from an edematous peau d'orange appearance to a sandpaper appearance with overlying vesicles and then to a necrotizing process with hemorrhagic bullae. Other case series have also included necrotizing fasciitis. A. baumannii is an important pathogen in burn units worldwide. Large burns pro vide ideal conditions for A. baumannii and facilitate patient-to-patient transmission. The presence of A. baumannii in wounds contributes to healing delays and graft loss. In addition, wound colonization is a risk

factor for bloodstream infections among patients with extensive burn injuries. A. baumannii infections resulting from trauma to soft tissues in the setting of natural disasters, such as tsunamis and earthquakes, have been reported. The implication is that A. baumannii should be con sidered in the differential diagnosis of soft tissue infections following exposure to tropical and subtropical environments. Urinary Tract Infections A. baumannii is an infrequent cause of urinary tract infections. The majority of cases reported are catheterassociated infections, reflecting the ability of A. baumannii to form biofilms on these devices. A few reports have described communityacquired infections occurring in the setting of nephrolithiasis and after renal transplantation. Meningitis Central nervous system infections with A. baumannii have been reported in the context of outbreaks, traumatic injuries, neurosurgical procedures, and external ventricular drains. One case series described a petechial rash in up to 30% of patients. Acinetobacter species may look similar to Neisseria meningitidis on a Gram stain of cerebrospinal fluid; both appear as gram-negative paired cocci. Eradi cation of A. baumannii from the cerebrospinal fluid can be challenging and requires careful selection of antibiotics that adequately penetrate the site of infection. Other Miscellaneous Infections A few cases of A. baumannii ker atitis associated with the use of contact lenses have been reported. Cases of native- and prosthetic-valve endocarditis have also been described. TREATMENT Acinetobacter Infections Treatment of Acinetobacter infections is challenging due to difficul ties in differentiating colonization versus infection and because Acinetobacter can develop resistance to most available antibiotics. Therefore, the choice of empirical therapy should be based on local epidemiology and, if available, the patient's colonization status with a carbapenem-resistant isolate. Definitive therapy should be determined by antimicrobial susceptibility testing. Antimicrobial options for the management of infections caused by A. baumannii are displayed in Table 167-1. Acinetobacter species possess intrinsic β-lactamases that inacti vate first- and second-generation cephalosporins. Through acquisi tion of extended-spectrum β-lactamases, these organisms can also become resistant to third- and fourth-generation cephalosporins, along with carbapenems. Nevertheless, when the isolate is suscep tible, β-lactam agents should be used. Ampicillin-sulbactam (due to its sulbactam component) is the treatment of choice, with cefepime, meropenem, and imipenem as alternative options based on in vitro susceptibility testing. Currently, there is no antibiotic regimen that has been proven superior for the treatment of carbapenem-resistant A. baumannii. The 2024 Infectious Diseases Society of

America (IDSA) “Guidance on the Treatment of Antimicrobial Resistant Gram-Negative Infec tions” recommends sulbactam-durlobactam in combination with a carbapenem as their preferred regimen, and high-dose ampicillinsulbactam in combination with either polymyxin B, minocycline, tigecycline, or cefiderocol is an alternative. Pairing of sulbactam with durlobactam makes a novel diazabicyclooctane non-β-lactam β-lactamase inhibitor with activity against the Acinetobacterderived cephalosporinases and class D β-lactamases including car bapenemases of the OXA family. High-dose ampicillin-sulbactam is recommended in combination with either polymixn B, mino cycline, tigecycline, or cefiderocol. The use of high-dose over standard dose ampicillin-sulbactam increases binding of sulbactam to its penicillin-binding proteins (PBP) targets (PBP2 and PBP3) in order to optimize inhibition of cell wall synthesis. This recom mendation is based on two meta-analyses of small clinical trials and observational data. In a randomized clinical trial including 125 patients with carbapenem-resistant A. baumannii, patients treated

TABLE 167-1 Therapeutic Options for the Management of MultidrugResistant Acinetobacter baumannii Infections ANTIBIOTIC DOSINGa COMMENTS Sulbactam 6–9 g/d Unavailable as single drug in many countries (including the United States). Different dosing strategies proposed if administered with ampicillin. Ampicillinsulbactam 3 g q4h 9 g q8h 27 g q24h Infuse over 30 min Infuse over 4 h Infuse as continuous infusion Sulbactamdurlobactam 1 g/1 g q6h Infuse over 3 h Meropenem 2 g q8h Carbapenem-susceptible isolates only; infuse over 3 h Imipenemcilastatin 500 mg q6h Carbapenem-susceptible isolates only; infuse over 3 h Cefiderocol 2g q8h Use in combination therapy; infuse over 3 h Colistin Dosing per the international consensus guidelines on polymixins (Tsuji BT et al, Pharmacotherapy 39:10, 2019) Colistin is preferred for urinary tract infections. Polymyxin B Dosing per the international consensus guidelines on polymixins (Tsuji BT et al, Pharmacotherapy 39:10, 2019) Polymyxin B is preferred over colistin for bloodstream infections. Tigecycline 200-mg loading dose followed by 100 mg q12h Use in combination therapy Minocycline 200 mg q12h IV/PO. Use in combination therapy aAll drugs are given by the IV route unless otherwise stated. with sulbactam-durlobactam had a 28-day all-cause mortality of 19% compared to 32% in patients treated with colistin, and with lower rates of nephrotoxicity in the sulbactam-durlobactam arm. Cefiderocol is a siderophore cephalosporine with in vitro stabil ity against the Acinetobacter-derived cephalosporinase and other -Hand hygiene -Contact precautions Health care worker’s hands A. baumannii– positive patient Shared equipment Health care environment -Physical separation from A. baumannii–negative patients -Rectal surveillance -Cohorting nursing personnel -Chlorhexidine baths -Antibiotic stewardship -Daily and terminal disinfection -Limits on shared equipment -Disinfection of equipment between patients FIGURE 167-1 Strategies for the prevention of dissemination of Acinetobacter baumannii in health care facilities.

extended-spectrum β-lactamases. However, A. baumannii isolates with reduced cefiderocol susceptibility have been described, and in a randomized clinical trial that included 54 critically ill patients with carbapenem-resistant A. baumannii, the end-of-study mortal ity was 50% in the cefiderocol arm, compared to 18% in the best available therapy arm (mostly consisting of colistin).

Polymyxins are cationic detergents that have become less popu lar as a result of nephrotoxicity and neurotoxicity. Additionally, polymyxins are difficult to dose, have a narrow therapeutic win dow, and do not reach optimal tissue concentration in the lungs, which is a common site of infection for A. baumannii. Despite its disadvantages, polymyxin B and polymyxin E (colistin) have been reintroduced in clinical practice as they retain in vitro activity against carbapenem-resistant

A. baumannii. In a randomized study of patients with pneumonia due to carbapenem-resistant Acineto bacter, patients receiving colistin in combination with high-dose ampicillin-sulbactam had a higher rate of clinical improvement by day 5 compared to those receiving colistin monotherapy. The combination of colistin plus meropenem was long favored due to in vitro synergy; however, two randomized controlled trials showed this strategy had comparable outcomes to colistin monotherapy. Several tetracycline derivatives have in vitro activity against A. baumannii. Of them, tigecycline and minocycline could be con sidered as part of a combination regimen, used at high doses when minimum inhibitory concentrations are low. Although doxycycline is a widely available agent with established breakpoints against A. baumannii, it is usually less active than minocycline. Eravacycline is a newer tetracycline with promising activity against A. baumannii; however its use has been limited; it is currently being marketed to treat more resistant strains. CHAPTER 167 Bacteriophage therapy against multidrug-resistant A. baumannii has been reported with varied success rates. Furthermore, dosing and duration of therapy vary by syndrome and resistance can also arise during treatment. Acinetobacter Infections ■ ■COMPLICATIONS AND PROGNOSIS Infections caused by A. baumannii can be associated with high mor tality rates. Factors contributing to higher mortality are thought to include severity of the patient’s underlying illness and drug resistance in the infecting strain. A. baumannii– negative patient -Physical separation from A. baumannii–positive patients -Cohorting nursing personnel -Chlorhexidine baths -Antibiotic stewardship

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