# 8.6.18 Other Yersinia infections Yersiniosis 1086

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section 8 Infectious diseases 1086 8.6.18 Other Yersinia infections: Yersiniosis Michael Prentice ESSENTIALS Yersiniosis is caused by the enteropathogenic Gram-negative organ- isms Yersinia enterocolitica and Yersinia pseudotuberculosis, which are worldwide zoonotic pathogens. Disease is acquired by consumption of contaminated food or water and is commonest in childhood, and in colder climates. Presentation is with diarrhoea, fever, and abdom- inal pain, which may mimic appendicitis. Late complications include reactive arthritis, erythema nodosum, and erythema multiforme. Systemic infection is more likely with Y. pseudotuberculosis and a sub- group of Y. enterocolitica, and also in patients with diabetes or iron overload. Diagnosis is by culture of the organism or convalescent ser- ology. Most cases of enteritis are self-limiting and antimicrobials are not indicated, but septicaemia or focal infection outside the gastro- intestinal tract requires antibiotics (usually cefotaxime, ceftriaxone, or ciprofloxacin). Prevention is by standard food hygiene precautions. Introduction and historical perspective Yersinia pseudotuberculosis was first identified in 1883 and Y. entero- colitica in 1939. Water-borne outbreaks of Y. pseudotuberculosis were recognized in Japan and Korea from the 1920s onwards. Y. enteroco- litica was rarely reported before the 1960s and the first large-scale outbreak of human disease was reported in 1976. Aetiology Enteropathogenic Yersinia are Gram-negative organisms of the order Enterobacterales. Whole genome sequencing of the 18 mainly environmental species in the genus Yersinia has shown that the en- teric pathogens Y. enterocolitica and Y. pseudotuberculosis evolved independently from nonpathogens rather than from a pathogenic common ancestor. However, they are both transmitted by the oral route and share many pathogenic features. Epidemiology Both enteropathogenic yersiniae are zoonotic pathogens distrib- uted worldwide but more common in temperate and cold countries. Y. enterocolitica commonly colonizes and infects domestic animals, particularly pigs. Y. pseudotuberculosis is associated with wild mam- mals such as rodents, rabbits, deer, and birds, and human infection is more rarely diagnosed. Y. enterocolitica infection is most common in children under the age of 5 years. Virulence plasmid-negative Y. enterocolitica strains (biovar 1A or phylogroup PG1 strains) are ubiquitous in the environment and the most common Yersinia spe- cies isolated from faeces and food other than pork in most countries. In Germany 40% of blood donors have anti-Yersinia Yop antibodies thought to relate to Y. enterocolitica infection,

and it is the third most common cause of bacterial diarrhoea in Scandinavian countries and New Zealand. Seroepidemiology and culture studies suggests human disease is at least 10-fold rarer in the United Kingdom, although animals in the United Kingdom frequently carry the organism. In the United States of America, high virulence 'American' strains of Y. enterocolitica (phylogroup PG2) have been displaced by European pig-associated strains (mainly phylogroup PG3, some PG4–6) of lower virulence in recent years. Infection with serotype O:8, phylogroup PG2 strains, previously rare in Europe, was reported in Poland from 2006 onwards. Recent outbreaks of yersiniosis involving Y. enterocolitica have been mainly pork-meat related; for example, children in New Zealand consuming cocktail sausages, although large raw and pasteurized milk-related outbreaks have been reported from the United States, Japan, and Canada in the past. Recent outbreaks of Y. pseudotuberculosis have followed consumption of lettuce and raw carrot (Finland), various raw vegetables (Russia), well water (Korea and Japan), raw milk (Finland), and homogenized milk (Canada). Pathology All recognized pathogenic Yersinia contain the pYV virulence plasmid. Ingested enteropathogenic Yersinia expressing invasin proteins and adhesins, YadA and Ail, adhere to and then pass through M cells over- lying Peyer's patches. They then multiply in lymphoid tissue, remaining extracellularly located due to the activity of the pYV plasmid-speci- fied injectisome (type III secretion system). This inactivates phago- cytic cells, including neutrophils, by injecting Yop proteins into them. Y. enterocolitica classically causes terminal ileitis with or without adja- cent mesenteric adenitis (microabscesses inside lymph nodes), while Y. pseudotuberculosis causes mesenteric adenitis without terminal ileitis. Some strains of Y. enterocolitica (biovar 1B, phylogroup PG2, so-called American strains, which until recently were rarely found in Europe) and Y. pseudotuberculosis contain a high pathogenicity island and produce an additional iron-binding siderophore. These strains are more likely to produce systemic infection and bacteraemia. Correspondingly, patients with iron overload (polytransfused, haemo- chromatosis) are at risk of serious or fatal consequences if infected by any enteropathogenic Yersinia, especially when using iron che- lators. Invasive Y. enterocolitica and Y. pseudotuberculosis strains can also penetrate from the gut to the liver and spleen without traversal of Peyer's patches, possibly via phagocytic cells. Some strains of Y. pseudo- tuberculosis produce a superantigenic toxin, Y. pseudotuberculosis-de- rived mitogen (YPM). Y. enterocolitica strains produce a heat-stable enterotoxin. Y. enterocolitica strains contain several metabolic operons found in salmonella and not present in Y. pseudotuberculosis, which may contribute to the enteritis phenotype. Y. enterocolitica biovar 1A or phylogroup PG1 strains contain some virulence-associated genes, and are pathogenic in insect models, but are equally common in asymp- tomatic patients and their pathogenicity for humans is uncertain. Prevention Standard food hygiene precautions are effective including avoiding consumption of undercooked or raw meat (e.g. pork chitterlings),

1087 8.6.18 Other Yersinia infections: Yersiniosis especially by children, and pasteurization of milk. Chlorination of water supplies is important for Y. pseudotuberculosis control. Yersinia grow (slowly) at refrigerator temperature, and prolonged cold storage of contaminated food or blood products can greatly in- crease their contamination. Clinical features Following an incubation period of 1 to 11 days (usually 4–6 days), enteric Yersinia infection usually presents with diarrhoea, fever, and abdominal pain. Abdominal pain in older children and adults is often central or right-sided, simulating appendicitis (pseudoappendicitis). Diarrhoea can be minimal or absent. Y. enterocolitica diarrhoea contains blood in 25–50% of cases. Infection is usually self-limiting, but bacteraemia and systemic spread can occur with subsequent focal infection in various tissues, including mycotic aneurysm. Most patients experiencing systemic enteropathogenic Yersinia sepsis have diabetes, iron overload, or immunosuppression. Contamination of blood for transfusion with Y. enterocolitica,

presumably introduced at the time of donation and multiplying on storage, is a rare but usually fatal cause of blood transfusion reactions and systemic sepsis. Immunological complications of enteric infection are common in northern Europe where HLA-B27 is frequent. Reactive arthritis follows several weeks after diarrhoea with other complications such as erythema nodosum, erythema multiforme, vasculitis, and glom- erulonephritis. A specific Yersinia-associated variant of erythema multiforme has been reported from Germany with localization of eruption to the neck, shoulders, and arms, accompanied by ery- thema nodosum, conjunctivitis, and arthralgia. Y. pseudotuberculo- sis strains producing superantigenic toxin YPM are associated with Far Eastern scarlet-like fever (FESLF) in eastern Russia, a childhood illness with desquamating rash, arthralgia, and polyarthritis also seen in Japan (Izumi fever) and Korea. There is epidemiological overlap between populations exposed to Y. pseudotuberculosis and the incidence of Kawasaki disease, an idiopathic acute systemic vas- culitis of childhood. Differential diagnosis Differential diagnosis includes appendicitis, other causes of ter- minal ileitis, mesenteric adenitis (Crohn's disease, tuberculosis), and fever with abdominal pain. Other causes of community-acquired septicaemia should be considered for the rarer systemic infection presentation. Clinical investigation Culture of material from normally sterile sites (blood culture, lymph nodes) is carried out on standard media. Selective cefsulodin- irgasan-novobiocin agar is used for faeces and other contaminated specimens. Culture-independent diagnosis is not yet in wide use. Standard identification of cultures to species level is possible in most laboratories, but some Y. enterocolitica strains isolated from faeces lack the virulence plasmid and their pathogenicity is uncertain. Isolation of Y. enterocolitica from a sterile site (e.g. blood) suggests pathogenicity. Reference laboratories separate Y. enterocolitica into distinct biotypes and serotypes of more or less established virulence, serotype Y. pseudotuberculosis, and provide convalescent serological tests. Standard MALDI-TOF databases can speciate Y. enterocolitica and Y. pseudotuberculosis and a research database has successfully biotyped Yersinia species. Y. enterocolitica serotypes are broadly con- sistent with genomic phylogeny, but in Y. pseudotuberculosis sero- type is poorly predictive of genetic relatedness or pathogenicity for humans. A  pan-Yersinia multilocus sequence typing schema has been devised which provides speciation and typing across the entire Yersinia genus. Criteria for diagnosis Diagnosis is by culture of the organism from a sterile site, bioserotyping or genetic (e.g. multilocus sequence typing) typing of faecal isolates of Y. enterocolitica into a pathogenic group, con- valescent serology by agglutinating antibodies, enzyme-linked im- munosorbent assay (ELISA), or Western blot. Y. pseudotuberculosis is rarely isolated from faeces and serology is the usual diagnostic method. Treatment Most cases of enteritis are self-limiting and antimicrobials are not in- dicated. Septicaemia or focal infection or scarlet-like fever (FESLF) outside the gastrointestinal tract require antibiotics. Y. enterocolit- ica strains possess two different β-lactamases and, in the absence of controlled trial data, therapy with cefotaxime, ceftriaxone, or ciprofloxacin are most commonly recommended for acute sepsis. Gentamicin is sometimes given in addition to β-lactams. Y. pseudo- tuberculosis sepsis can be treated by the same agents, although this organism does not produce β-lactamase and is generally ampicillin sensitive. Acute enteritis is usually self-limiting. Septicaemic illness has a high mortality (up to 50%), probably associated with predisposing illnesses. In northern European countries with high HLA-B27 prevalence, Yersinia postinfection complications, including reactive arthritis, can result in chronic illness which responds poorly to antimicrobials. Areas of uncertainty or controversy Virulent plasmid-negative phylogroup PG1, biovar 1A Y. enteroco- litica strains might have some role in diarrhoea. Likely future developments Increased use of culture-independent diagnosis from faeces and DNA typing of isolates (e.g. multilocus sequence typing) can speed diagnosis. Because chronic oropharyngeal colonization with Y. enterocolitica is fre- quent in apparently healthy domestic animals, such as pigs, breaking the

transmission chain requires selective breeding of specific pathogen-free

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