Brucella

Obligate intracellular parasite of animal and human Brucella spp. are causative agent of brucellosis, malta fever or undulant fever.

4 major human pathogens and their animal reservoirs:

- B.melitensis (goats & sheep).
- B.abortus (cattle).
- B.suis (pigs).
- B.canis (dogs).

General characters:

- Gram negative rods, non-motile, non spore–forming & some are capsulated.

- Brucella are obligate aerobes, grow on blood agar and enriched media (trypticase-soy) / 2-5 days showing small, convex colonies. Virulent strains give typical smooth colonies which tend to change to rough form if become avirulent. Serum of susceptible animals contain globulin and a lipoprotein that suppress growth of a virulent type and favor the growth of virulent one.
-Only B.abortus requires 5-10% Co₂ for growth, others grow in air and all require the presence of basic pepsin dye except B.canis.

-Brucella utilize CHO with no acid or gas, reduce nitrate, catalase & oxidase positive and H₂S is produced by many strains. They are sensitive to heat and killed by milk pasteurization for 10 min at 60°C and by acidity of sour milk, also killed by exposure to 1% phenol for 15 min.

**Antigenic structure:**

-The 2 lipopolysaccharide antigens A&M are present in different proportions in the 4 spp.

-Superficial L Ag also present that resembles the Vi Ag of Salmonellae.

**Pathogenesis:**

The organisms enter the body either by ingestion of contaminated milk products or through direct contact of mucus membrane or abraded skin in an occupational setting such as farmers.

The organisms → localize in RES (L.N, liver, spleen and bone marrow) many killed by macrophages, but some survive within the cells (intracellular) protected from Abs. Granulomas may appear which can progress to focal abscesses and caseation. Osteomyelitis or cholecystitis also occasionally occur.
Brucellae which infect human have differences in pathogenicity:

*B. abortus → mild disease without suppurative complications and non caseating granuloma.

*B. canis → mild disease.

*B. suis → chronic disease with suppurative and caseating granulomas.

*B. melitensis → more acute and severe disease.

The mechanism of pathogenesis is not well defined except the role of endotoxin in active brucellosis.

**Clinical findings:**

After incubation period (1-6) weeks, non-specific insidious symptoms occur as malaise, fever, weakness, aches and sweats, fever usually undulating (rises up then fall during night) accompanied by drenching sweat. Enlarged lymph node, liver and spleen are frequently found.

Following the initial infection, a chronic stage may develop, characterized by weakness, aches, low grade fever and some psychoneurotic symptoms. The diagnosis of chronic brucellosis is difficult.
Laboratory diagnosis:

A-specimens include:

- Blood for blood culture during acute phase
- Biopsy material for culture of illness
- Serum for Antibody detection

B-Culture: on tryptcase – soy broth and sub culture every 3-5 days on solid media (under aerobic and 10% CO₂), keep blood for 4wks before being discard as negative. The colonies appear as small, transparent and non hemolytic, Gram’s stain showing the typical coccobacilli. Biochemically they are (oxidase positive & urease positive). The isolated brucellae typed by H₂S production and agglutination by specific antisera.

C-Serology

1-Agglutination test (Rose Bengal)

For detection of IgG Ab, serial tube dilution used, IgG titers>1:80 indicate active infection.

This test can give fales positive because of cross reaction with other infection and fales negative because of the presence of blocking Abs which can be overcomed by:
a. 2- Mercaptoethanol test – the addition of 2me will destroy IgM and leaves IgG for, agglutination reaction.

b. Blocking Ab (IgA) which appear during subacute stage and remain for years causing a negative test in low serum dilution, so increase dilution and using coombs antiglobulin method (patient serum + Brucella antigen + Anti human globulin)

2-ELISA & EFAT

D. Brucella skin test:

Intra dermal injection of protein extract → erythema, edema and induration develop within 24hrs ← unreliable, & rarely used.

Treatment:

Because of Intracellular location ← for best result treatment must be prolonged with combination of streptomycin & tetracycline.