Vibrio

Vibrios are curved gram negative bacilli

Motile single polar flagellum

First described by Filippo pacini

Robert Koch isolated and named comma bacillus due to curved shape

Growth stimulated by salt

Fermenters,oxidase positive and aerobic

Ubiquitious ,salt loving natural habitat sea water,sea food,sewage,rivers

Out of 35 vibrios isolated only 12 causes human infections

Most important Vibrio cholera caused pandemics and epidemics

Vibrio cholera

Classification-

Based on salt requrement-

Non halophilic-V.cholera and V.mimicus

Halophilic-V.alginolyticus and V.vulnificus

Heiberg classification-8 groups on fermentation of 3 sugars.V cholera in gr 1

Gardner and venkatraman classification-

Based on

Serogrouping

Biotyping

Serotyping

Phagetyping

1)Serogrouping-Based on somatic O ag V.cholera grouped into more than 200 serogroups

O1 serogroup-responsible for pandemics and epidemics.Agglutinated by O1 antisera

NAG vibrios-Other than O1 not agglutinated by O1 antiserum called non agglutinable vibrios or NAG vibrios.Also pathogenic

O139 serogroup-Epidemics in bangladesh and India since 1992

Non O1/O139 serogroups-causes diarrhoea and extraintestinal manifestations but no epidemic so far

Serotyping-O1 divided into 3 serotypes

Inaba,Ogawa and Hikojima

Ogawa most common followed by Inaba

During epidemics shifting between serotypes takes place

Hikojima unstable transitional stage where ag of others expressed

Biotyping

Serogroup O1 has 2 biotypes classical and Eltor

They are differentiated by biochemical reactions and susceptibility to polymyxin B and bacteriophages

Classical highly virulent and caused first six pandemics worldwide

Eltor caused 7 th epidemic and named after eltor egypt where it was identified in quarantine camp

Currently Eltor is cause of outbreaks

Phage typing-Basu and mukherjee phage typing

Susceptibility to different lytic phages

Pathogenesis-

Both V cholera O1 and O139 produces cholera toxin

Mode of transmission-

Infective dose-

Factors promoting transmission-

Crossing of protective layer of mucus-Vibrios penetrate mucus layer and reach epithelial cells due to motility ,mucinase and enzymes and haemagglutinin protease

Adhesion and colonization-adhesion to intestinal epithelium facilitates by toxin regulated pilus(TCP)

Cholera toxin-Once in small intetstine it produces cholera toxin resembles LT of E.coli but but more potent

Mechanism of action of cholera toxin-

A and B fragments

B fragment binds to GM1 ganglioside receptors on intestinal epithelium-A fragment internalized-Causes ADP ribosylation of G proteins-upregulate adenylate cyclase-intracellular accumulation of cAMP-fluid loss-dairrhoea-dehydration-acidosis and shock

Other virulence factors-

Zona occludens toxin

Siderophore

Verocell toxin

Bacterial endotoxin-Unlike other GNB doesnot contribute in pathogenesi but included as component of killed vaccines

Gene for cholera toxin-Pathogenicity islands from bacteriophage integrated into bacterial chromosome

ToxR gene-regulates expression of CT,TCP and other virulence factors

Clinical manifestations-

V.Cholera O1 or O139 causes

Asymtomatic infection(75%)

Mild diarrhoea or cholera(20%)

Life threatening diarrhoea(5%)

Watery diarrhoea

Rice water stool Fever absent ,vomiting

Muscle cramps

Epidemiology-

Epidemic,pandemic,endemic,limited ,sporadic

Homeland-ganges and brahmputra

Till 19 th century cholera restricted to homeland

First six pandemics 1817 to 1923 caused by classical biotype and involved whole world

Seventh pandemic differed fro first six by occuring outside india,caused by eltor biotype

Replacing classical biotype

Eltor associated with more carrier state,is hardier and capable of surviving longer

This accounts for its rapid spread

O139(Bengal strain)-isolatedd from chennai 1992

Not agglutinated by O1 to O138 antisera

O139 appears derivative of eltor but differs by capsulated so more invasive

Reservoir-only humans

Source-asymptomatic cases or carriers

Season-rainfall,flooding and high temperature

Other factors-Poor sanitation,povery,overcrowding,mobility

Poor immunity,O blood group.malnutrition,HIV

All age groups but mostly children

Habitat-coastal sea salt water,brackish estuaries

Lab diagnosis-

Specimen-Watery stool or rectal swab

Transport media-Vrmedia,alkaline salt transport media,cary blair media,autoclaved sea water

Direct microscopy-

Motility-

Culture-Nutrient agar,peptone water

Selective media-TCBS agar,BSA agar,TCBS agar and at last macconkey agar

Culture smear-

Biochemical testing-Indole+,citrate varible ,urease negative,MR+

Cholera red reaction-add sulphuric acid to growth in peptone water red pink ring is positive

Sugar fermentation test

String test

Decarboxylase tests

Salt tolerance tests-V.cholera tolerates 6% nacl

Biotyping-differentiated between eltor and classical

Serogrouping-

First colony tested by O1 antisera if negative than O139 antisera

Serotyping-If O1 agglutination positive then serotyping done with ogawa and inaba antisera

Ogawa agglutination-Ogawa serotype

Inaba agglutination-Inaba serotype

If agglutination by both antisera hikojima serotype

Treatment-

Fluid replacement most important for management of cholera

In mild to moderate fluid loss-ORS

In severe cases –IV fluids with ringer lactate or normal saline

Antibiotics have minor role to play as pathogenesis is due to toxin although may decrease duration and severity .

Antibiotics helps in clearance of organism from stool and prevents carrier stage

WHO recommends use of antibiotics only for severely dehydrated patients

Prevention

Provision of safe water

Improved sanitary disposal of feces

Food sanitation

Proper outbreak investigation

Notification as cholera is a notifiable disease and hence cases should be notified

Chemoprophylaxis

Tetracycline is drug of choice

Indicated for household contacts only during epidemics

Vaccines

Injectable killed vaccines-No longer used ,provide little protection and fail to induce local intestinal mucosal immune response

Oral cholera vaccine(OCV)-

2 types

1)Killed whole cell vaccine-2 preperations available

a)Whole cell vaccine-Composed of killed whole cells of V.cholera O1 both classical and eltor

b)Whole cell recombinant B subunit cholera vaccine in addition composed of recombinant cholera toxin b subunit

Protection is short lived 58% for whole cell and 85% for recombinant.Children protected better than adults

Recommended during epidemics but not interepidemic

B) Oral live attenuated vaccine-Use mutant strains that lack gene encoding for cholera toxin

CVD 103 HgR,Peru15 for O1

CVD-112 and bengal -15 for O139

Trials are going on

Non O1/O139(O2-138)V.cholera

Resemble biochemically to V.cholera O1/O139,but donot agglutinate with O1/O139 antisera

Cause gastroenteritis bloody stool but never causes epidemics

Treatment same as for cholera

Extraintestinal infections wound infection,bacteremia ,otitis media

Halophilic vibrios

Can withstand higher conc of salt

Wide spread in marine environment

Common in summer and rainy fall

1)V.parahaemolyticus

In india seen in Calcutta

First reported from Japan

Clinical manifestations

Food borne gastroenteritis due to raw sea food or oysters

Extraintestinal manifestations rare

Pathogenesis is due to polysaccharide capsule

Hemolysin,urease

Lab dignosis-

Distinct properties from vibrio cholera are

Doesnot show darting motility,capsulated and shows bipolar staining and on TCBS agar shows green colonies

Treatment-Self limiting and treatment similar like cholera

Vibrio vulnificus-

Most severe infection among vibrio species

Causes primary sepsis in patients with liver diseases and severe wound infection with erythemtous swelling or cellulitis

Only species to ferment lactose and can be cultured from blood or cutaneous lesions

Treatment-Wound debriment and general supportive care

Antibiotics tetracycline,quinolones and cephalosporins