• LECTURE 3..... Measles & German measeles(rubella).

- OBJECTIVES
- what is measles / rubella?
- How does the pt with measles / rubella presented to you?
- What are the complications of measles/ rubella?
- How can u manage pt with measles / rubella?.
- How can we prevent measles / rubella)?.

MEASLES = RUBEOLA

- Measles is an acute viral infection, still an important cause of childhood morbidity and mortality in developing countries.
- It is an *RNA virus* of *paramyxoviridae* family.

TRANSMISSION:

- Measles is highly contagious disease
- about 90% of susceptible household contacts acquire the disease
- The portal of entry is through the Resp. tract or conjunctivae following a contact with large droplets or small droplet aerosols in which the virus is suspended.
- Face face contact is **not** necessary because viable virus may be suspended in air up to 1 hr after a source case leaves a room.

PATHOGENESIS OF MEASLES

- The organs affected are:-
- Skin..... small vessels vasculitis(rash)
- Mucus membrane of resp. tract and intestine....epithelial necrosis
- CNS....encephalitis and SSPE

CLINICAL FEATURES OF MEASLES:-

Measles has 3 clinical stages:-.

- 1-Incubation stages: incubation period last 8-12 days
- **2-Prodromal phase**: usually last 3-5 days with low grade fever, dry cough, coryza, conjunctivitis and photophobia, these symptoms nearly always proceed the appearance of **koplik spots**, by 2-3 days.
- Koplik spots first appear as discrete red lesions with bluish white spots in the center on the inner aspects of the cheeks at the level of the premolars.
 Koplik spots have been reported in 50-70% of measles cases but probably occur in the great majority.
- The conjunctival inflammation and photophobia may suggest measles before Koplik spots appear, (a transverse line of conjunctival inflammation, sharply demarcated along the eyelid margin, (Stimson line). may be of diagnostic assistance in the prodromal stage) As the entire conjunctiva becomes involved, the line disappears.
- **3-The rash phase**: a final stages of maculopapular rash, and sudden rising of fever reaching 40°C or higher, the rash usually start as faint macules on the upper lateral part

of the neck, behind the ears, along the hairlines, and on the posterior part of the cheek, then spread rapidly over the face ,trunk,& extremities including palms & soles.

- As the rash fade a brownish discoloration appear and then disappear within 7-10days.
- Of the major symptoms of measles, the cough lasts the longest, often up to 10 days. In more-severe cases, generalized lymphadenopathy may be present, with cervical and occipital lymph nodes especially prominent.

DIAGNOSIS OF MEASLES is almost always based on clinical and epidemiologic findings., In the absence of a recognized measles outbreak, confirmation of the clinical diagnosis is often recommended.

- Serologic confirmation may be made by identification of (Ig) M antibody in serum or by demonstration of a 4-fold rise in IgG Ab in acute and convalescent specimens collected 2-4 wk apart.
- Viral isolation from blood, urine, or respiratory secretions using human embryonic or rhesus monkey kidney cells.

TREATMENT OF MEASLES:-

- Antiviral therapy is not effective.
- supportive treatment with bed rest, antipyretic drugs, and adequate fluid intake.
- <u>Vitamin A:</u> oral vitamin A reduce morbidity and mortality in children with severe measles. Vitamin A therapy is indicated for all patients with measles. Vitamin A should be administered once daily for 2 days at doses of 200,000 IU for children 12 mo of age or older; 100,000 IU for infants 6 mo through 11 mo of age; and 50,000 IU for infants < 6 mo of age.
- In children with signs and symptoms of vitamin A deficiency, a 3rd age-appropriate dose is recommended 2 4 wk after the 2rd dose.

COMPLICATION OF MEASLES:-Morbidity and mortality from measles are greatest in patients younger than 5 yr of age (especially <1 yr of age) and older than 20 yr of age.

- Respiratory complications: Pneumonia is the most common cause of death in measles, as giant cell pneumonia, or secondary bacterial infection, or some time exacerbation of underlying mycobacterial infection.
- Otitis media, Croup, tracheitis, and bronchiolitis are common complications in infants and toddlers.
- A severe form of measles rarely seen now is hemorrhagic or "black measles "was often fatal.
- **GIT:**-Diarrhea and vomiting are common symptoms and Dehydration is a common consequence, especially in young infants and children. Appendicitis may occur from obstruction of the appendiceal lumen by lymphoid hyperplasia.
- **CNS:** Febrile seizures occur in <3% of children with measles. **Encephalitis** with greater numbers occurring in adolescents and adults Approximately 15% of patients with measles encephalitis die.
- Other rare complications including Guillain-Barré syndrome, cerebral thrombophlebitis, and retrobulbar neuritis.
- Lately **SSPE**, Subacute Sclerosing Panencephalitis, After 7-10 yr may develop.

• Prevention Of Measles In Household Contacts:

- 1. Isolation of pt until 5 days after the rash has appeared. Immunocompromised patients with measles will shed virus for the duration of the illness, so isolation should be maintained throughout the disease.
- 2. MMR Vaccination for all susceptible children >12 m age within 72 hr of contact.those who have not received 2 doses by 11-12 yr of age, a second dose should be provided...
- 3. Single IM dose of Ig should given to all:
 - A: Household and hospital contact<12 m.
 - B. Pregnant mother.
 - C. Immunocomprmised individual.

• RUBELLA=GERMAN MEASLES

- Rubella is an acute viral infection caused by single stranded *RNA virus* of rubiviridae a genus of *Togaviridae family*.
- Human are the only natural host of rubella virus, it spread either by oral droplet or transplacentally causing congenital Infection.
- Peak incidence was among children 5-14 yr, but now most cases occurs among susceptible teenagers and young adults.
- Rubella virus have been recovered from the nasopharynx 7days before rash and 7-8 days after its disappearance.

CLINICAL FEATURES OF RUBELLA

- Incubation period is 14-21days followed by prodromal phase of mild catarrhal symptoms is shorter than of measles (2/3 of cases are subclinical), followed by retro-auricular, posterior cervical and postoccipital LAP, *no other diseases cause tender enlargement of these nodes to the extent that rubella does*, it evident 24 hr before the rash and may remain for 1 wk or more.
- in 20% of pt an enanthem appear as a rose colored spots on the soft palate just before the appearance of the rash which start on the face with rapid spread and rapid evolution on the trunk as discrete maculo-papules that may be confluent, on the 2nd day the rash may assume pinpoint appearance especially over the trunk with mild itching.
- The rash usually clear by the 3rd day associated with low grade fever anorexia, mild conjunctivitis but no photophobia.
- Older girls and women may have **polyarthritis** with arthralgia, swelling, tenderness, and effusion of any joint but most frequently the small joints of the hands.

DIAGNOSIS OF RUBELLA:-

- 1-Clinical features
 - o lab. Finding:- WBCs count is noraml or slightly reduced;
 - o thrombocytopenia is rare, with or without purpura
- 2-Confirmation by serology, or viral culture.

TREATMENT:

There is no specific antiviral therapy; treatment is entirely supportive. Antipyretics are indicated for fever.

- Iv Ig or corticosteroids can be considered for severe, nonremitting thrombocytopenia. **COMPLICATION OF RUBELLA:**
- 1. Encephalitis similar to that seen with measles occurs in about 1 in 6,000 cases. The severity is highly variable, and overall mortality rate of 20%.
- 2. Thrombocytopenic Purpura
- 3. Congenital Rubella syndrome.
- 4. Progressive rubella panencephalitis (PRP) It has an onset and course similar to SSPE.
- 5. Other CNS complications rarely reported include Guillain-Barré syndrome and peripheral neuritis. Myocarditis is a rare complication.
- Congenital rubella: infection during pregnancy result in virus croosing the placenta.
- The virus replicate extensively in fetal tissues causing cells death growth disruption, and impair differentiation. The damage more sever in 1st trimester.
- Congenital rubella virtually affect all organs systems. the risk of congenital disease is greatest with primary infection during the 1st trimester (about 90 % of infants whose mothers acquire the infection before the 11th wk of pregnancy, diminishing to about 10-20 % by the end of the 1st trimester) The most common manifestation of congenital Rubella is IUGR,
 - > other common finding include:
 - o Cataract: either unilateral or bilateral +/- microphthalmia.
 - o Structural cardiac defect:
 - o PDA or PS, myocarditis.
 - o Sensorineural hearing loss +/- meningoencephalitis.
- congenital rubella can be confirmed by: detecting specific Ig M Ab in neonatal serum, or by culturing rubella virus from the nasopharynx or urine.

PREVENTION:

- PT isolation..
- MMR vaccine

References:

- Nelson Textbook of Pediatrics, 20th edition.
- Nelson essentials Textbook of Pediatrics . 7th edition.
- Illustrated Textbook Of Pediatrics.

Lecture 3 Mumps .pertussis

- OBJECTIVES:-
- Have some ideas about:-
- Causative agents of mumps/ pertussis.
- Clinical features and epidemiology of mumps/ pertussis .
- Complications of mumps/ pertussis .
- Management of mumps/ pertussis .
- Prevention of mumps/ pertussis .

Mumps

Mumps is an acute viral infection caused by RNA virus of paramyxoviridae family. it is endemic in most unvaccinated population.

EPIDEMIOLOGY OF MUMPS

Mumps is spread from person to person by respiratory droplets, contaminated fomites and possibly by urine.

Age: Before vaccination 5-9 yrs old children, now young adults producing outbreak in colleges

- Epidemics occurs at all seasons but are slightly more frequent in late winter and spring.
- The period of maximum infectiousness is 1–2 days before to 5 days after parotid swelling *Clinical features of Mumps:*
- Incubation periods range from 5days, peak at 16-18 days, 30-40% of infection are subclinical.
- The typical patient presents with a prodrome lasting 1-2 days and consisting of fever, headache, vomiting, and achiness. Parotitis then appears and may be unilateral initially but becomes bilateral in approximately 70% of cases
- Mumps parotitis: The pt usually have pain and swelling of the parotid gland 1st fill the space between the posterior border of mandible and mastoid then extend downward and forward and may be preceded or accompanied by ear pain on the ipsilateral side and may proceed rapidly reaching a maximum within few hrs, although it usually peak in 1-3 days and slowly subside within 3-7 days but occasionally lasts longer.
- As swelling progresses, the angle of the jaw is obscured and the ear lobe may be lifted upward and outward. The swollen area is tender and painful, the pain being elicited by tasting sour liquids such as lemon juice or vinegar the parotid swelling is usually accompanied by low grade fever. Fever and other systemic symptoms resolve in 3-5 days.
- A morbilliform rash is rarely seen.
- Submandibular salivary glands may also be involved

DIAGNOSIS OF MUMPS:-

- When mumps was highly prevalent, the diagnosis is clinical.
- The lab finding includes:
 - o Elevation of S. amylase(normal within 2 wk.)
 - Leucopenia with relative lymphocytosis.

- **Confirmation**:- detection of viral antigen by direct immunofluorescence, or identification of nucleic acid by reverse transcriptase PCR.
- **viral culture** from upper resp sec. ,CSF or urine during the acute illness *COMPLICATION OF MUMPS:*-
- Meningoencephalitis is most frequent in childhood especially in male.(in 10-30% of cases)
- Orchitis& epidedymitis: in pre-pubescent boys is extremely rare, but after puberty, orchitis occurs in 30-40% of males. Infertility is rare even with bilateral orchitis.
- **Oophoritis** is uncommon in post pubertal females but may cause severe pain and may be confused with appendicitis when located on the right side.
- Others: pancreatitis, myocarditis, arthritis, thyroiditis, oophoritis, and sensory neural hearing loss.
- **TREATMENT OF MUMPS:-** There is no specific antiviral therapy.
- The treatment is entirely supportive, antipyretic for fever, bed rest as guided by the pt needs, the diet should be adjusted to the pt abilities to chew.
- *orchitis* should be treated by local support and bed rest.
- Arthritis can be treated by 2 wk course of NSAID or steroid.

PREVENTION OF MUMPS

- Pt isolation
- Immunization with the live mumps vaccine is the primary mode of prevention It is given as part of the MMR 2-dose vaccine schedule, at 12-15 mo of age for the 1st dose and 4-6 yr of age for the 2nd dose.
- vaccine effectiveness after 2 doses is 88%

• Pertussis `` whooping cough``

- Pertussis is acute respiratory tract infection caused by Gram negative, toxin producing coccobacilli, *Bordetella pertussis*, Which is the sole cause of epidemic pertusis.
- **Bordetella parapertussis** is an occasional cause (<5%) of sporadic cases. **EPIDEMIOLOGY OF PERTUSSIS**
- Pertussis is an endemic disease, with superimpose epidemic cycles every 3-4 yr, it is extremely contagious airborne disease, attack rate approach 100% in susceptible individual.
- Neither natural disease nor vaccination provide complete or lifelong immunity, protection begins to wane 3-5 yr after vaccination.
- In non vaccinated areas it affect 1-5 yr age, while it occur mostly in infants, adolescent,& adults in vaccinated areas.

CLINICAL MANIFESTATION OF PERTUSSIS:

• classically pertussis is a prolonged disease of three main stages:
1-Catarrhal stage (1-2 weeks):-

• after incubation period of 3-12 days the pt have congestion, rhinorrhea, low grade fever, sneezing and lacrimation.

2-Paroxysmal stage (2-6 week)

- The pt have dry intermittent irritative cough that evolve into paroxysms `hall mark of pertussis` start as machine-gun burst of uninterrupted coughs with protruded tongue, bulging watering eyes, chin and chest held forward, the face become purple until the cough cease with a loud whoop (forceful inspiratory gasp),
- post tussive emesis is common in all ages. and exhaustion is universal.
- At the peak of the paroxysmal stage, patients may have more than 1 episode hourly.
 - 3- Convalescent stage (≥2 weeks):-
- With reduction in number, severity, and duration of episodes of coughing.
- Paradoxically in infants the cough and whoop may be louder and more classic in this stage.
- Infants younger than 3 mo of agedo not display the classic stages.
- Adolescents and previously immunized children have foreshortening of all stages of pertussis.
- Adults have no distinct stages.
- Findings on physical examination generally are uninformative. Signs of lower respiratory tract disease are not expected unless complicating secondary bacterial pneumonia is present. Conjunctival hemorrhages and petechiae on the upper body are common
- Diffrential diagnosis:
- Protracted coughing (which in some cases is paroxysmal) can be caused by Mycoplasma, parainfluenza viruses, influenza viruses, enteroviruses, respiratory syncytial viruses, or adenoviruses
- Diagnosis of pertussis:-
- 1-Clinical features: cough > 14 days, with at least 1 associated symptom of paroxysms, whoop, or post tussive vomiting has a sensitivity of 81% and a specificity of 58% for confirmation of pertussis.
- 2-Lab finding:
- Leukocytosis:15000-100000 cell/mm3, with absolute lymphocytosis.
- Thrombocytosis.
- 3-CXR: perihilar infiltrates or edema with butterfly appearance and variable atelectasis.
- Parenchymal consolidation suggests secondary bacterial infection. Pneumothorax, pneumomediastinum, and air in soft tissues can be seen .
- 4- Confirmation:-
- Culturing of **deep** nasopharyngeal aspiration.
- D F T: Direct fluorescent antibody test.
- PCR to test nasopharyngeal specimens has sensitivity similar to culture TREATMENT OF PERTUSSIS

1-Non specific treatment:

- Hospital admission:-Infants younger than 3 mo of age with suspected pertussis usually are admitted to hospital, as are many between 3 and 6 mo of age unless witnessed paroxysms are not severe, as well as are patients of any age if significant complications occur.
- Care of feeding, prevent dehydration, removal from aggravating environmental smoke, excessive stimulation, or a dry or polluting heat source

2-Antimicrobial therapy:- use either of:-

- *Erythromycin:* 40-50 mg/kg/day in 4 doses for 14 days.
- Clarithromycin 15 mg/kg day Bd PO for 7days.
- Azithromycin: 10 mg/kg in a single dose on day 1, then 5 mg/kg/day on days 2-5
- TMP-SMZ: for pt > 2m :TMP 8 mg/kg/day, SMZ 40 mg/kg/day in 2 divided doses for 14 days
- **ADULTS:-Azithromycin:**500 mg in day1 then 250 mg/day on days 2-5

Erythromycin: 2 g/day in 4 doses for 14 days.

TMP-SMZ, 1 g/day in 2 divided doses for 7 days TMP 320 mg/day, SMZ 1,600 mg/day in 2 divided doses for 14 day

COMLPICATION OF PERTUSSIS

- 1- Apnea: may lead to resp. failure.
- 2- Secondary bacterial infection such as otitis media and pneumonia (S. aureus , S. pneumoniae)
- 3- Convulsion (hypoxia, hypoglycemia, hyponatremia due to increase ADH, alkalosis, The only neuropathology documented in humans is parenchymal hemorrhage and ischemic necrosis.)
- 4- Bronchiectasis and collapsed lobes has been reported rarely after pertussis.
- 5- Increased intrathoracic and intra-abdominal pressure during coughing can result in conjunctival and scleral hemorrhages, petechiae on the upper body, epistaxis, hemorrhage in the CNS and retina, pneumothorax and subcutaneous emphysema, umbilical and inguinal hernias.

PREVENTION OF PERTUSSIS:-

- <u>1-pt isolation</u>: pt should placed in respiratory isolation with use of masks by all health care personnel entering the room until 5days after initiation of macrolid therapy.
- <u>2-Immunization</u>: 3 doses of DTaP should be administered during the 1st year of life, at ages 2, 4, and 6 mo of age. In addition to 2 booster doses.
- <u>Chemoprophylaxis for contacts:</u> A macrolide agent should be given promptly to all household contacts and other close contacts, regardless of age, history of immunization, and symptoms.

References:

- Nelson Textbook of Pediatrics , 20 edition .
- Nelson essentials Textbook of Pediatrics , 6th edition.
- Illustrated textbook of pediatrics.