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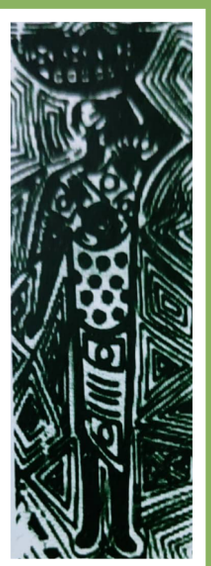

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Synopsis: Burden and Current Concepts in Respiratory Syncytial Virus (RSV) Infections in Children

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Introduction

Respiratory Syncytial Virus (RSV) is a significant cause of morbidity and mortality among infants and young children worldwide. Childhood deaths from RSV exceed 100,000 annually, with the highest risks seen in premature newborns, infants aged less than six months, and children with pre-existing conditions.[1] Recent studies also implicate RSV as an important pathogen in adults, particularly older people and those with severe underlying conditions.[1] Lower-and middle-income countries (LMIC) bear the most burden, as 95% of acute lower respiratory tract infection (LRTI) episodes and 97% of the mortality from RSV in children below five years of age occur in these countries. [1,2]

The virus was first named chimpanzee coryza virus when it was discovered by researchers studying the effects of poliovirus in chimpanzees in 1955.[3] In 1956, Robert Chanock, a paediatrician and virologist, confirmed its role in the causation of LRTI among children and its cytopathic effects on respiratory cells, and coined it 'respiratory syncytial virus'.[3] Awareness about the RSV infections remains poor, especially in the LMICs.[4] RSV causes mostly upper respiratory tract infections that are self-limiting. However, young children and susceptible individuals may develop LRTI, such as bronchiolitis or pneumonia, that may progress to respiratory failure and death. Additionally, RSV has been linked to long-term respiratory consequences such as recurrent wheezing, asthma and impaired lung function in later life.[5] Non-respiratory manifestations have also been reported.[6] These all result in a significant burden to the healthcare system and increased healthcare costs.

Significant strides have been made in the development of preventive strategies in the last decade. Newly licensed preventive strategies include maternal vaccinations that aim to provide antibodies to infants for their protection in the first six months of life, as well as the use of long-acting monoclonal antibodies. [1] Very recently, Gavi, The Vaccine Alliance, approved support for RSV maternal vaccines in Gavi-eligible countries. [7] For the successful introduction of these agents, it is vital that healthcare workers be well informed on the burden of RSV and the recent updates in RSV infection management and prevention strategies.

Epidemiology and Transmission

RSV is the most common cause of acute LRTI, including bronchiolitis and pneumonia, in infants and young children worldwide. It is estimated that by two years of age, over 90% of children would have been infected, at least once by the virus. [2] RSV is spread from person to person via respiratory droplets and has an incubation period of 2 to 8 days (mean of 4 to 6 days).

Young infants (<6 months), premature infants, children with congenital heart diseases, bronchopulmonary dysplasia, Down's syndrome, neuromuscular disease, malnutrition and the immune-compromised are at the highest risk of developing disease.[1,2] Host factors and vial factors Host factors which contribute to disease severity include young age, prematurity, congenital heart/lung disease, Down's syndrome, neuromuscular diseases.[1,8] Other factors that contribute to infection include exposure to tobacco smoke, indoor air pollution, daycare attendance, multiple siblings, lack of breastfeeding and hospitalisation. [1,8] RSV is a significant nosocomial hazard on paediatric wards, especially during outbreaks.

About 33 million LRTIs are associated with RSV annually, of which 10% result in hospitalisations.[1] More than 95% of RSV-associated acute lower respiratory infection episodes occurred in LMICs in

2019. Approximately 101,400 RSV-associated deaths occurred in children 0-60 months of age, and 97% of these occurred across all age bands in LMICs in the same year. [1,2] RSV-attributable deaths were about 2% among children aged 0-60 months. Thus, 1 in 50 deaths in children aged 0 to 60 months was attributable to RSV. Furthermore, it is estimated that for every child that dies in a hospital from RSV, three to four die in the community. [2] It is estimated that the RSV positivity rate among children aged 0-60 months with severe LRTI infection is 23-26% in LMICs and 29% in high-income countries (HIC), respectively.[2] In a recent prospective, multi-country study involving 10 LMICs (Bolivia, Cameroon, The Gambia, Ghana, Haiti, Mozambique, Nepal, Nigeria, Sudan, and Tanzania), 29% of all children aged less than two years admitted to paediatric intensive care units (PICU) or high dependency units with severe acute LRTI were positive for RSV (Figure 1). [9] There have been few studies on RSV prevalence in children in Nigeria, but the findings have been consistently similar. Among two communities in the southwest of Nigeria, Robertson *et al.*, [10] reported RSV-associated LRTI prevalence of 35% which is comparable to findings in other parts of Nigeria, such as Oladele *et al.* [11] and Garba *et al*.[12]

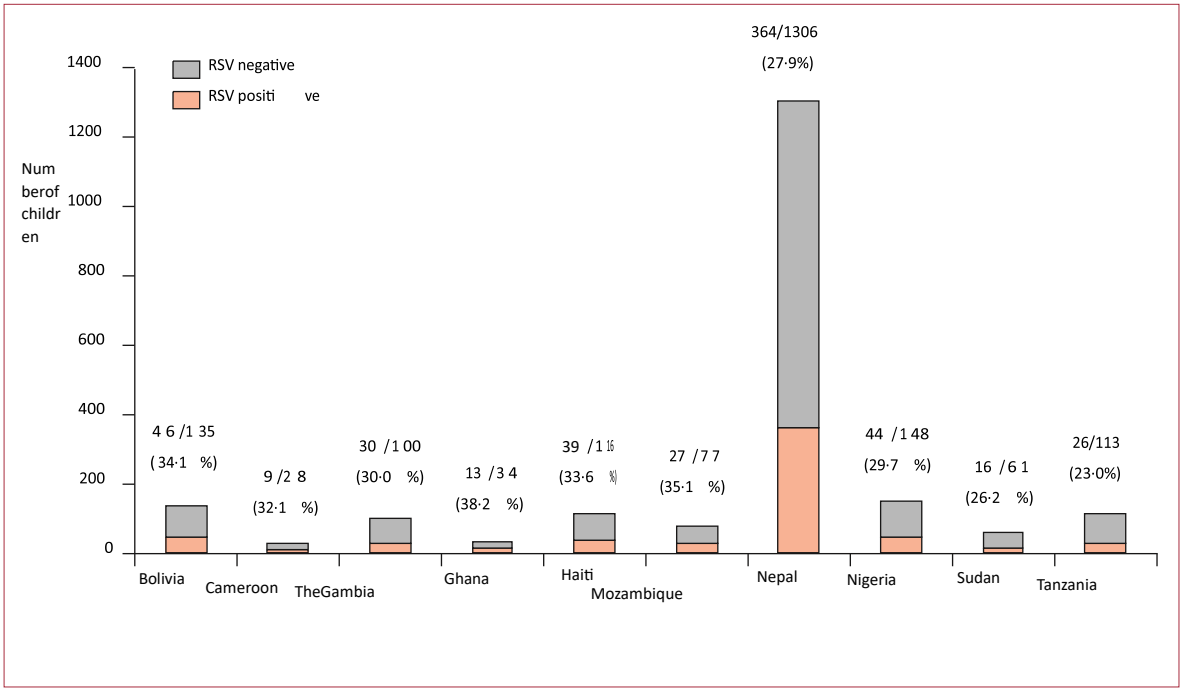

Figure 1: RSV positivity among children younger than 2 years admitted to the paediatric intensive care unit with extended severe acute respiratory infection RSV=respiratory syncytial virus. (Abdelrahman DN, et al. Respiratory syncytial virus infection among children younger than 2 years admitted to a paediatric intensive care unit with extended severe acute respiratory infection in ten Gavi-eligible countries: the RSV GOLD—ICU Network study. Lancet Glob Health. 2024;12(10):e1611-e1619.)

The seasonality of RSV is variable depending on geographical location.[13] In countries in the equatorial regions of the world, infections occur in up to 10 months of the year, with more cases observed during the rainy season. [12, 13] RSV season typically begins around March to June in the southern hemisphere, and wanes by August to June. In the northern hemisphere, RSV activity starts between September and February, and declines between February and May. [13]

Pathophysiology

RSV is a single-stranded RNA virus which belongs to the Genus *Pneumovirus*, Subfamily *Pneumovirinae*, Family *Paramyxoviridae*.[14] The envelope contains three viral transmembrane surface glycoproteins: the large glycoprotein G, the fusion protein F, and the small hydrophobic SH protein (Figure 2). The non-glycosylated matrix M protein is present on the inner face of the envelope. RSV has only one serotype but two subtypes, subtypes A and B, which can be identified based on the protein G sequence. Both subtypes circulate simultaneously during an epidemic season, but usually one of the

two predominates each year. [14] Once inside the host cell, RSV replicates within the cytoplasm, producing viral RNA and proteins that assemble into new virions.

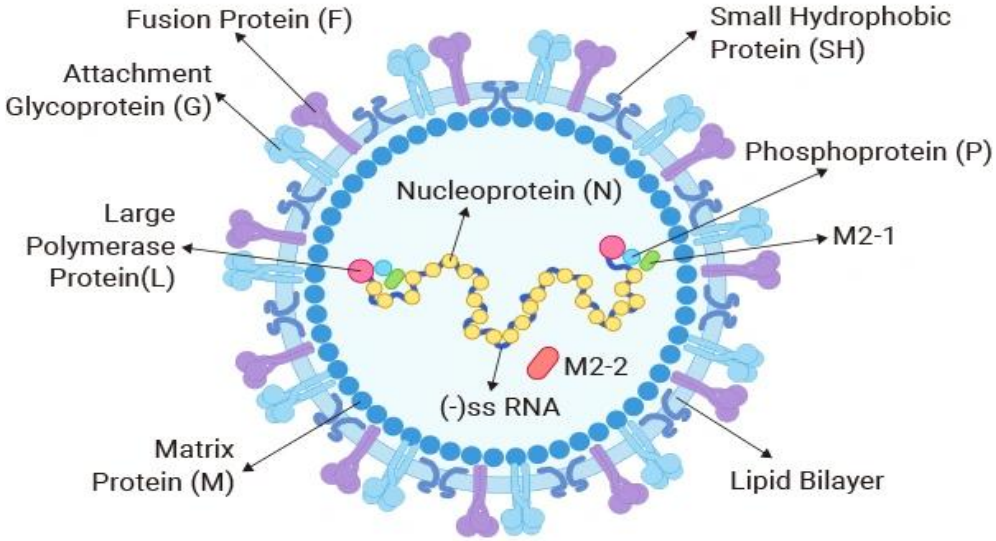

Figure 2: RSV viral structure (credit to http://www.cusabio.com)

The formation of syncytia enables direct cell-to-cell spread of the virus. This facilitates rapid dissemination of the virus within the respiratory epithelium, exacerbating tissue damage. The replication cycle of this virus is error-prone, allowing for a rapid generation of mutations, thus resulting in changes in the virulence of RSV and contributing to challenges in the development of antiviral agents or vaccines.[14,15]

Following inoculation into the nasopharyngeal or conjunctival mucosa, the virus rapidly spreads into the respiratory tract, with a predilection for the apical ciliated epithelial cells. The immune response is mediated by infected epithelial cells of the respiratory system and alveolar macrophages. Host genetic factors can lead to excessive immune reaction outside the lungs. Similar to other *Paramyxoviridae,* RSV can also affect non-epithelial cells. This mechanism could partially explain some of the non-pulmonary manifestations of RSV infection, where direct invasion of the relevant organs has been documented.[6]

The host immune response to RSV involves both innate and adaptive components. Once the host detects the virus, interferons and proinflammatory cytokines such as IL-4, IL-6, and IL-8 are produced. When activated in excess, this response recruits immune cells to the site of infection, resulting in tissue damage. Airway passage inflammation, mucus production, epithelial cell destruction, airway obstruction, and impaired gas exchange are attributed to excessive immune activation. In a young child, the release of proinflammatory molecules, dysfunction of neural pathways and compromised epithelial integrity could affect the development of the airways and lead to bronchial hyper-reactivity and asthma, irrespective of atopic status. [5] Studies have shown that children with RSV infection may be more vulnerable to secondary bacterial infection, which may occur in 1-3% of cases of severe RSV disease. [9, 15] This is probably mediated via induction and gene expression of bacterial receptors that enhance the binding of bacteria to the lower respiratory tract. These include intercellular adhesion molecule-1 (ICAM-1), platelet activating factor-receptor (PAF-r) and carcinoembryonic antigen-associated cellular adhesion molecule 1 (CEACAM1). [15]

Spectrum of Illness

The majority of cases manifest as self-limiting mild upper respiratory tract infection (rhinitis, coryza, otitis media or croup). An upper respiratory tract involvement will present with rhinorrhoea, nasal congestion, cough, sneezing, and sometimes fever, myalgia and otalgia. Among infants and children

who are at risk, infection may also progress to the lower respiratory tract. Up to 40% of infants may develop bronchiolitis.[16] Typically, young infants present with a low-grade fever, poor feeding, tachypnoea, chest retractions, wheezes, and even apnoea. Complications include respiratory failure, acute respiratory distress syndrome, pulmonary consolidation, and atelectasis.[14, 15] Non-respiratory manifestations of RSV have been reported in the literature (Table I). Re-infection with RSV may occur, even during the same RSV season, but is usually milder compared to primary infection, except in young infants and the immunocompromised, where severe disease may occur.[14]

Table I: Non-respiratory manifestations of RSV

System	Manifestation
Neurological	Convulsions Lethargy Feeding difficulties Changes in muscle tone Strabismus Cerebrospinal fluid abnormalities Encephalopathy
Cardiovascular	Myocarditis Myocardial dysfunction Rhythm abnormalities Hypotension
Endocrine	Syndrome of inappropriate ADH secretion Hypercortisolism
Gastro-intestinal	Acute hepatitis Hepatic failure

(Adapted from Gkentzi D, Dimitriou G, Karatza A. Non-pulmonary manifestations of respiratory syncytial virus infection. J Thorac Dis 2018;10(Suppl 33):S3815-S3818. doi: 10.21037/jtd.2018.10.38.)

Diagnosis

RSV diagnosis is made by a thorough clinical evaluation, which includes assessing the child's symptoms, medical history, and physical examination. Important things to seek in the evaluation include underlying risk factors such as congenital heart disease, lung conditions, exposure to cigarette smoke and other environmental pollutants, prematurity, nutritional status and immune deficiency disorders.[16] Laboratory tests for RSV are often reserved for severe cases with underlying conditions where the health care provider wants a confirmation to diagnose complications, to rule out other conditions with similar symptoms, to guide treatment decisions and supportive care or to inform infection control measures to prevent transmission.[16] The diagnostic tests include Rapid Antigen Detection Tests (RADTs), Polymerase Chain Reaction (PCR) and Direct Fluorescent Antibody (DFA) testing on RSV antigens or genetic material in nasopharyngeal samples. Chest X-rays may be used to evaluate lung involvement and complications. A lymphocytic picture in a full blood count and C-reactive protein or procalcitonin can also help support the diagnosis of a viral infection. Differential diagnosis of RSV infection includes other respiratory viruses, bacterial pneumonia, asthma, severe malaria and sepsis.

Management

The treatment approach may vary depending on the patient's age, the severity of the disease, and underlying health conditions. The indications for hospitalisation include a history of inability to tolerate oral hydration, signs and symptoms of respiratory distress, apnoea, failure to maintain an oxygen saturation of 90% or greater, or progression of symptoms over the first 72 hours.[16] Mild symptoms are self-limiting. The treatment of severe RSV infection focuses on relieving symptoms, supporting respiratory function, and preventing complications. The principles of management of severe disease include adequate hydration and humidification of inhaled air to aid in loosening of mucus in the respiratory passages. [16] Dehydrated children or those with poor feeding should receive intravenous fluids. It may be necessary to withhold oral feeding and feed through a nasogastric tube in children with significant tachypnoea to prevent aspiration. Supplemental oxygen should be provided to children with saturation of less than 90 per cent via high flow nasal cannula (HFNC) to maintain

adequate oxygen saturation. Mucus and secretions from the nose and throat should be removed by gentle suctioning. Continuous Positive Airway Pressure (CPAP), which provides a constant flow of air pressure, may be necessary to help keep airways open, and mechanical ventilation in cases of respiratory failure.

Antibiotics should be given where there is a high suspicion of bacterial co-infection or if the child is severely ill, as specified by the WHO guidelines.[17] Antipyretics may be administered to decrease the fever in some patients.

The use of nebulised 3% hypertonic saline solution, bronchodilators, and inhalational/injected steroids has not been found to have any benefits and is no longer recommended.[16] Ribavirin is the only post-infectious antiviral agent which had been approved for severe RSV. Still, its cost, potential side effects, and limited efficacy have limited its use,[18] and it is also not recommended. For patients for whom a decision is taken to manage as an outpatient, they should be followed up within 48 hours. Caregivers should be taught to identify and monitor for 'danger signs' and informed to return for re-evaluation on identification of any. Danger signs include central cyanosis, difficulty breastfeeding/drinking, vomiting everything, convulsions, lethargy/unconsciousness, or head nodding. [17]

Prevention of RSV Disease

Preventive measures include the practice of frequent hand-washing with soap and water or alcohol-based rubs, cessation of smoking by caregivers, prevention of exposure to bio-fumes and exclusive breastfeeding for infants in the first six months of life. Efforts should be made to prevent contact between noticeably infected individuals and high-risk groups. In such cases, nursing mothers should wear face masks while caring for their infants. Infection control measures such as isolation may minimise transmission among infants requiring hospitalisation.

A monoclonal antibody, Palivizumab, has been successfully used for decades in HIC to prevent severe RSV disease in high-risk infants. Still, the need for monthly administration during RSV season and the high cost have limited its use. [15] In recent times, maternal vaccines and long-lasting monoclonal antibodies have been licensed for use in pregnant women and infants. [1, 18]

Current concepts and future directions

The pharmaceutical landscape for RSV is gradually transforming. As knowledge of the structure of RSV proteins and pathogenic mechanisms expands, several vaccines and post-infectious agents are currently in various phases of development. In the vaccine space, this includes vaccines designed to mimic natural infection and subunit vaccines, which focus on specific viral proteins to induce a protective immune response. Others are mRNA vaccines designed to instruct messenger RNA to produce specific RSV proteins that can stimulate an immune response.[15] Currently, the WHO approves the use of a vaccine containing the prefusion confirmation F protein (RSVpreF) for pregnant women.[1] RSVpreF is given during late pregnancy to help protect newborns through transplacental antibody transfer. Gavi decided to support its use in eligible countries in July 2025. [8] There are also long-acting monoclonal antibodies that are administered to infants for temporary protection against RSV infection. Nirsevimab and Clesrovimab are two such agents that have been licensed for use in infants. [1,18] These are given to infants at birth or shortly after birth, and the protection conferred from these agents lasts up to five months.

Post-infectious antiviral agents also appear very promising, as these agents target different stages of the RSV life cycle. Some of the mechanisms of action include inhibition of viral attachment (e.g. Resveratrol), viral membrane fusion with host cells (e.g. Ziresovir, Sisunatovir), and inhibition of protein transcription and translation (PC 786, remdesivir).[19] These agents, if successfully approved, have the potential to reduce the burden of RSV-associated LRTI significantly.

Public Health Policy Implications

The World Health Organisation (WHO) recommends RSV vaccination for all pregnant women and administration of long-acting monoclonal antibodies to infants.[1] The recent decision by the Gavi board to support maternal RSV vaccination will help to close the gap in protection and reduce inequities in child health, especially in LMICs.

Public health campaigns to raise awareness of RSV infection and prevention strategies among healthcare providers, pregnant women, and families could help reduce the global burden of RSV. Healthcare provider recommendations will play a crucial role in ensuring uptake by the target

populations. Studies on cost-effectiveness may help determine each country's preparedness to incorporate these interventions into its national vaccination programme. Disease surveillance will require strengthening to monitor trends of RSV infections and vaccine effectiveness, while long-term safety of RSV vaccines and monoclonal antibodies should be evaluated by continued research.

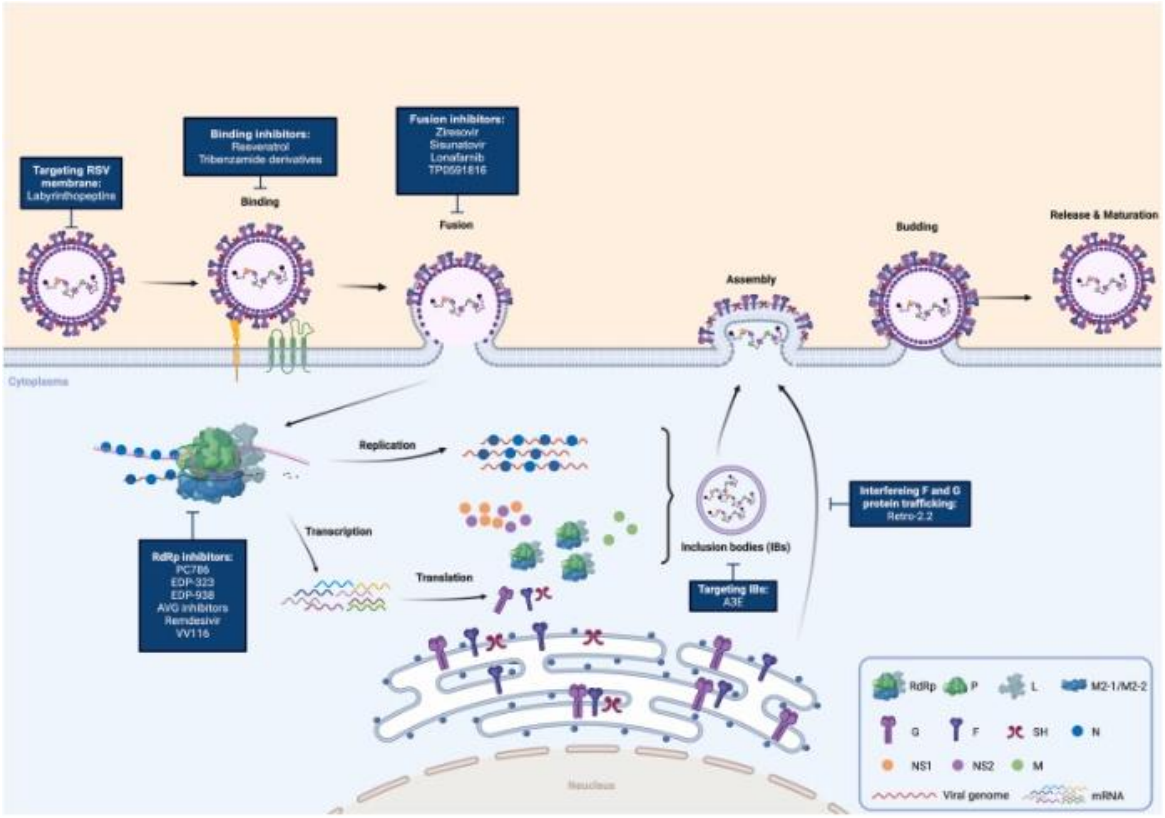

Figure 3: The life cycle of RSV and the indication of drugs targets different steps of RSV life cycle. (Adapted from Fig. 1 in Hu M., Bogoyevitch M.A., Jans D.A. Impact of respiratory syncytial virus infection on host functions: implications for antiviral strategies. *Physiol. Rev.* 2020;100:1527–1594. doi: 10.1152/physrev.00030.2019.)

Conclusion

RSV is an important contributor to childhood morbidity and mortality as well as a significant strain on healthcare systems. After decades of repeated attempts, effective pharmaceutical strategies for its prevention are now available. Equitable distribution of these strategies will significantly accelerate the progress to attaining SDG Target 3.2. Creating awareness among health care providers, pregnant women, and families is most vital to achieve vaccine acceptability, demand and uptake among expecting mothers.

Summary of key points

- A high index of suspicion and prompt diagnosis are crucial for effective management and prevention of RSV infection.
- The treatment of RSV infection focuses on relieving symptoms, supporting respiratory function, and preventing complications.
- The use of bronchodilators, corticosteroids and antiviral agents has not shown any benefits, and is therefore not recommended.
- RSV vaccines administered to women during late pregnancy and long-acting monoclonal antibodies administered to infants, which protect against severe RSV disease in infants, have recently been recommended by the WHO.
- Awareness of RSV burden and prevention strategies among health care providers, pregnant women and families is crucial for acceptance, demand and uptake of prevention strategies.

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EXCERPTS FROM THE 2025 PAN WEBINARS

Overview of Childhood Hypertension

Peter Igoche
Limi Children's Hospital, Abuja

Introduction

- Blood pressure = Cardiac output x Peripheral Resistance
- Blood pressure = Stroke volume x Heart rate x Peripheral Resistance
- Blood pressure gets elevated if either or more of the followings increases
  - Stroke volume
  - EDV – ESV
  - Dependent on contractility, preload and afterload
  - Heart rate
  - Peripheral resistance

Table II: American Academy of Paediatrics updated definition for Paediatric blood pressure categories 2017

Patterns	Age range	
	<13 years	**≥13 years**
Normal BP	Systolic and diastolic BP <90th centile	Systolic BP <120 and diastolic BP <80mmHg
Elevated BP	Systolic and diastolic BP ≥90th centile to <95th centile, OR 120/80mmHg to 95th centile (whichever is lower)	Systolic BP 120 to 129 and diastolic BP <80mmHg
Stage 1 HTN	Systolic and diastolic BP ≥95th centile to <95th centile + 12mmHg, OR 130/80mmHg to 139/89mmHg (whichever is lower)	130/80 to 139/89mmHg
Stage 2 HTN	Systolic and diastolic BP ≥95th centile + 12mmHg, OR ≥140/90mmHg (whichever is lower)	≥140/90mmHg

BP CONCEPT: TWO SIDES OF A COIN

ACCOMMODATION EFFECT

Occurrence of lower BP levels with repeated measurements in clinical settings because of inherent BP variability as well as an adjustment to the experience of having BP measured.

WHITE COAT PHENOMENON

- When BP readings in clinical settings are higher than readings outside clinical settings. Anxiety related.
- Not as benign as once thought- considered "elevated BP"
- Requires follow-up! eg school nurse takes daily BP for 3-4 weeks. OR
- Average of 2 or 3 BP readings taken morning and night for 1 week OR
- 24-hour apart BP measurement

METHODS OF BP MEASUREMENT

- Upper arm auscultation- bases for reference
- Wrist BP measurement
- Concept of peripheral amplification of systolic BP; Radial BP>arm BP in children
- Ambulatory BP Measurement
- More accurate for HTN diagnosis than clinic-measured BP
- For confirmation of HTN in children >5yrs.
- LVH correlates more strongly with ABPM than casual BP measurement

BP MEASUREMENT

-Auscultate with the bell of stethoscope on brachial artery in antecubital fossa.
-Lower end of the cuff should be 2-3cm above the antecubital fossa.
-Inflate cuff to 20-30mmHg above point radial pulse disappears.
-Deflate at rate of 2-3mmHg per second
-SBP = 1st Korotkoff sound (1st appearance of clear tapping sounds)
-DBP = 5th Korotkoff sound (Sounds disappear)

BP measurement in childhood
-The bladder length at least 80% to 100% of the arm circumference.
-The bladder width at least 40-50% of arm circumference.

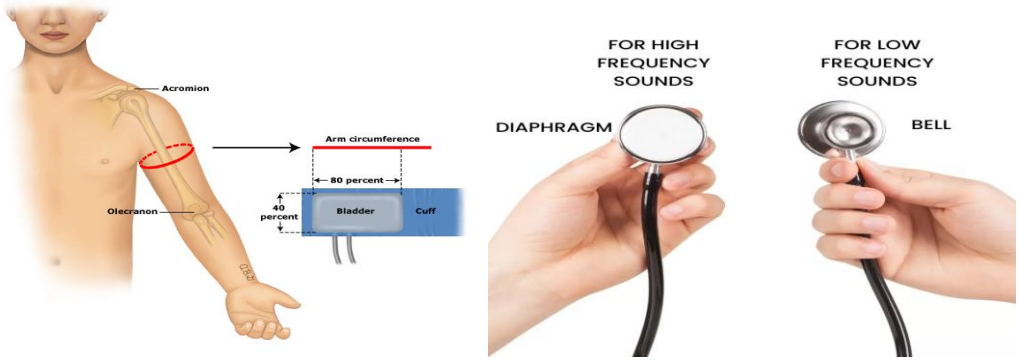


Figure 4: BP Measurement in children

Methods of BP measirement
-BP measurement with automated devices
-No reliable normative BP standards for this approach
-Accuracy of some such devices is uncertain!
-At best, for follow-up after HTN has been diagnosed.

BP MEASUREMENT FREQUENCY BEGINS AT 3 YEARS OF AGE
For otherwise healthy children, measure annually thereafter.
For obese children (BMI ≥95 centile), renal disease, DM, coarctation of aorta, those on medications known to increase BP, measure at every health encounter
At less than 3years, measure BP at well-child care visits if they are at increased risk for developing hypertension (HTN). For example:

- Prematurity GA<32 weeks or SGA,
- VLBW,
- Umbilical arterial line insertion history
- Congenital heart defects (repaired or not)
- Recurrent UTI,
- Haematuria,
- Proteinuria,
- Known renal disease or family history
- Systemic illnesses e.g. SCD, tuberous sclerosis
- Evidence of raised intracranial pressure

CLASSIFICATION OF HYPERTENSION BASED ON AETIOLOGY
Essential (Primary)

- Specific cause not identifiable

Secondary

- **Specific cause can be identified**
- Renal- glomerulonephritis, pyelonephritis, obstructive uropathy, renovascular, RVT
- Cardiovascular- Coarctation of Aorta, PDA, aortic insufficiency
- Endocrine- hyperthyroidism, catecholamine (pheochromocytoma, neuroblastoma)
- Neurogenic- Raised ICP, GBS, poliomyelitis
- Drugs- NSAIDS, Corticosteroids, sympathomimetic drugs (nose drops, cough meds, theophylline)

Goals of evaluation

- Distinguish between primary and secondary hypertension
- Identify any treatable conditions that may be causing or contributing to hypertension

- Identify comorbidities or risk factors for early CVD
- Obesity, dyslipidemia, DM
- Identify patients for whom antihypertensive drug therapy is warranted
- Secondary or symptomatic hypertension without an associated risk factor amenable to treatment e.g. obesity
- Any stage of HTN with comorbid CKD or DM
- Those failing a trial of lifestyle change

Table III: Distinguishing features between primary (Essential) and secondary Paediatric Hypertension

Clinical features	Primary HTN	Secondary HTN
Age		
Prepubertal		Secondary HTN is more likely in children especially those less than 6 years of age
Post-pubertal	Older children and adolescents are more likely to have primary HTN	
Diastolic HTN		Diastolic HTN more likely to be associated with secondary HTN
Nocturnal HTN		Nocturnal HTN is more likely to be associated with secondary HTN
Overweight/obesity	Children/adolescents with overweight/obesity are more likely to have primary HTN	
Family history of HTN	Children with positive family history of primary HTN are more likely to have primary HTN	Family history may be positive in some cases of secondary HTN due to a monogenic cause (e.g. autosomal dominant polycystic kidney disease)
Symptoms of underlying disorders	Children with primary HTN are typically asymptomatic	Children with secondary HTN often have other symptoms related to the underlying cause (e.g. headache, sweating and tachycardia due to catecholamine excess in patients with pheochromocytoma)

Diagnostic workup

Presenting complaint

Mild hypertension usually asymptomatic

Highlighting importance of accurate measurement of BP

Acute, severe HTN- may be symptomatic in presentation

Acute glomerulonephritis- headache, dizziness, nausea/vomiting, irritability

±neurological manifestations (eg stroke), congestive heart failure or renal dysfunctions.

Diagnostic workup- History

Review of system

Cardiovascular system

Of CoA or surgery for it

Renal

History of obstructive uropathies, UTI, renal surgery

Medications history

Corticosteroids, antiasthmatic drugs, cold medications, aminoglycosides, cocaine use

Social history

Smoking or consumption of excessive amount of coffee or tea

Family history

Essential HTN, atherosclerotic heart disease and stroke

Diagnostic workup- Physical examination

General Examination; BMI

Obesity is a common cause of essential HTN, and HTN from adrenocortical disorders

Children with secondary HTN from renal disease are rarely obese

Pulse analysis

Bounding peripheral pulse- in PDA or aortic regurgitation

Weak or absent femoral pulses + BP differential between the arms and legs (CoA)

In normal children, SBP in LL is > 5-10mmHg higher than in UL

Diagnostic workup – physical examination

For end-organ damage
Retinal vascular changes due to HTN
Cardiac heave or laterally displaced point of maximal impulse, which may indicate left ventricular hypertrophy

Retinal fundus photographs of hypertensive retinopathy
(A) Mild hypertensive retinopathy is indicated by the presence of generalized arteriolar narrowing, arteriovenous (AV) nicking, and opacification of the arteriolar wall ("copper wiring").
(B) Mild hypertensive retinopathy with focal arteriolar narrowing.

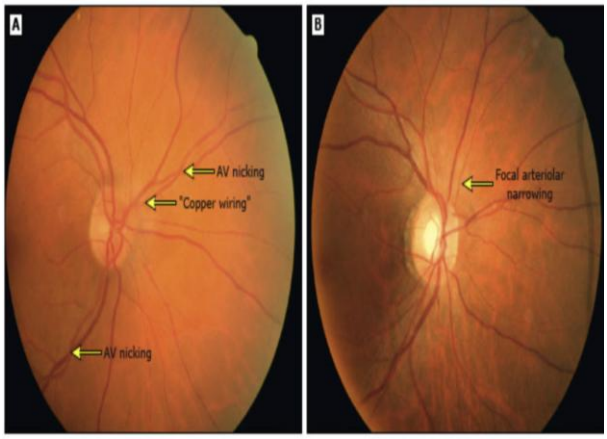
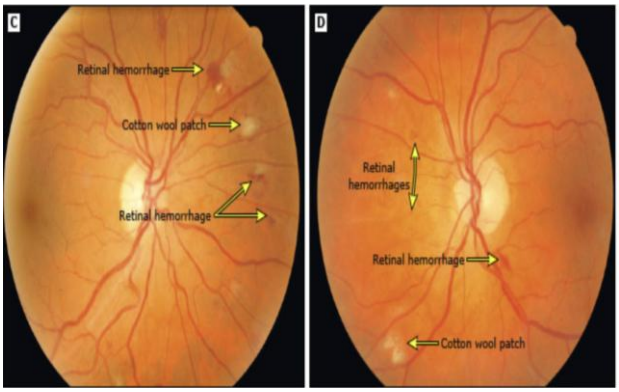


Figure 5: Retinal fundus photographs of Mild hypertensive retinopathy

(C, D) Moderate hypertensive retinopathy with multiple retinal hemorrhages and cotton wool patches.



(E, F) Severe hypertensive retinopathy with swelling of the optic disk, retinal hemorrhages, hard exudates, and cotton wool patches.

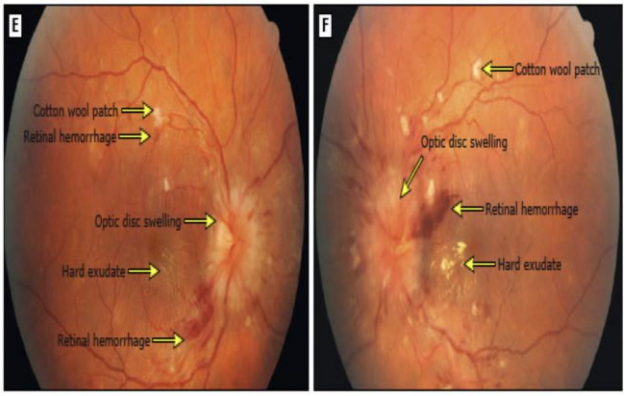


Table IV: Diagnostic workup – General investigations

Patient Population	Screening Tests
All patients	Urinalysis
	Chemistry panel (electrolytes, BUN, creatinine)
	Lipid profile, fasting or non-fasting (total cholesterol, HDL)
	Renal ultrasonography in those <6 yr of age or those with abnormal urinalysis or renal function
Obese (BMI >95th percentile) children and adolescents	Hemoglobin A1c
	AST and ALT (screen for fatty liver)
	Fasting lipid panel (dyslipidemia)
Optional tests to be obtained on the basis of history, physical examination, and initial studies	Fasting glucose for those at high risk for diabetes mellitus
	TSH
	Drug screen
	Sleep study (if found snoring, daytime sleepiness, or reported history of apnea)
	CBC (those with growth delay or abnormal renal function)

Diagnostic workup – specialised investigations
Echocardiography
CoA -

- LVH secondary to HTN follow-up
- LVH = LV mass > 51g/$m^{2.7}$ (boys & girls), OR LV mass > 115g/BSA for boys & > 95g/BSA for girls
- LV relative wall thickness of 0.42cm= concentric LV hypertrophy
- LV relative wall thickness >1.4cm = abnormal
- Decreased LV ejection fraction if <53%
- Target organ assessment; repeat echo 6-12 monthly intervals

Image for renovascular disease – detection of renal artery stenosis

- Doppler renal ultrasonography in normal weight children >8 yrs
- Computered Tomography Angiography
- Magnetic Resonance Angiography

Specialized chemistries

- Peripheral Plasma Renin Activity
- ↑ (high-renin HTN) ⇒ renal parenchymal or renovascular disease
- ↑ (low-renin HTN) ⇒ excess mineralocorticoid effects eg hyperaldosteronism
- Aldosterone levels in serum and urine- to exclude hyperaldosteronism if patient is hypokalemic
- Primary aldosteronism secondary to benign adrenal adenoma or bilateral idiopathic adrenal hyperplasia → ↑plasma aldosterone
- Secondary aldosteronism (overactivity of RAAS) secondary to juxtaglomerular cell tumor and renal artery stenosis → ↑plasma aldosterone
- 24-hour urine collection for catecholamine/metabolite (metanephrine, normetanephrine & VMA) assay; elevated in tumors eg pheochromocytoma, neuroblastoma
- 24-hour urine collection for free cortisol (↑ in Cushing syndrome) & 17-Ketosteriod (↑ in congenital adrenal hyperplasia)

Management of essential hypertension- *Nonpharmacological intervention*
Started as an initial treatment
Counselling on:

- Weight reduction if overweight or obese
- Healthy diet
- Regular aerobic exercise
- Avoidance of smoking and oral contraceptives.

Table V: Dash diet recommendations

Food	Servings per Day
Fruits and vegetables	4–5
Low-fat milk products	≥2
Whole grains	6
Fish, poultry, and lean red meats	≤2
Legumes and nuts	1
Oils and fats	2–3
Added sugar and sweets (including sweetened beverages)	≤1
Dietary sodium	<2300 mg per d

Management of essential hypertension- pharmacological intervention
-Used when nonpharmacological approaches are ineffective
Indication for drug therapy
-Severe symptomatic HTN- use IV antihypertensives initially
-Persistent HTN despite a trial of lifestyle modification
-Symptomatic HTN
-Stage 2 HTN without clearly modifiable factor (like obesity)
-Any stage of HTN associated with renovascular and renoparenchymal disease or DM
-Hypertensive target-organ damage: LVH, ↑LV mass
-Family history of early complications of HTN
-Child who has dyslipidaemia

Drugs of choice

Initiating drugs

-ACE inhibitors, ARB, long-acting calcium channel blockers (CCBs), or thiazide diuretic

(NB: African American children do not show as robust response to ACE inhibitors, so CCBs appear better for them)

-In children with HTN and CKD, proteinuria and Diabetes mellitus

-ACE inhibitor or ARB as initial drugs as these are reno-protective

-Overweight children at risk of developing Diabetes mellitus

-ACE inhibitor, ARB, and CCBs. Avoid diuretics and beta-blockers as they raise blood glucose levels

Table VI: Lifestyle modifications to prevent and manage hypertension

Modification	Recommendation	Approximate SBP Reduction (Range)†
Weight reduction	Maintain normal body weight (body mass index 18.5–24.9 kg/m²).	5–20 mmHg/10kg[92,93]
Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and lowfat dairy products with a reduced content of saturated and total fat.	8–14 mmHg[94,95]
Dietary sodium reduction	Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride).	2–8 mmHg[94-96]
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week).	4–9 mmHg[97-98]
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks (e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey) per day in most men, and to no more than 1 drink per day in women and lighter weight persons.	2–4 mmHg[99]

Goals of treatment

For uncomplicated primary HTN without hypertensive end-organ damage

Reduce BP to <95th centile

For children with CKD, DM or hypertensive end-organ damage

Reduce BP to <90th centile

Step-down or cessation of therapy

In selected patients (overweight children with successful weight loss) with uncomplicated primary HTN.

Discontinuation of therapy

A trial of gradually discontinuing pharmacotherapy is appropriate for:

Patients with mild initial HTN who are well controlled on a single drug and who have made progress with lifestyle intervention, such as weight loss and sodium restriction.

These patients will require ongoing lifestyle intervention and BP monitoring after drug therapy is discontinued.

Patients with secondary HTN if the cause has been identified and corrected.

Those with prolonged secondary HTN (eg, those with coarctation of the aorta or renal artery stenosis) may have persistent HTN even after successful repair and may require continued antihypertensive medication.

Follow-up Evaluation

A trial of gradually discontinuing pharmacotherapy is appropriate for:

-Patients with mild initial HTN who are well controlled on a single drug and who have made progress with lifestyle intervention, such as weight loss and sodium restriction.

-These patients will require ongoing lifestyle intervention and BP monitoring after drug therapy is discontinued.

-Patients with secondary HTN if the cause has been identified and corrected.

-Those with prolonged secondary HTN (eg, those with coarctation of the aorta or renal artery stenosis) may have persistent HTN even after successful repair and may require continued antihypertensive medication.

Management of secondary hypertension

Based on aetiology

NB- About 90% of secondary HTN are caused by CKD, renovascular diseases, and CoA.

Remaining 10% by other diseases

Correctable secondary HTN in children CoA, Renovascular HTN

Renovascular hypertension

-Secondary to any lesion that impairs blood flow to a part or all of one or both kidneys

-Accounts for 3-10% of children with HTN

-Imaging-

- Doppler ultrasonography
- CTA
- MRA
- Renal arteriography; gold standard

Renovascular hypertension – treatment

- Drugs while awaiting definitive treatment
- ACE inhibitors most effective
- But monitor renal function as ACEI can cause a GFR drop
- CCBs good for HTN patients with less impairment of the ischemic kidney
- Surgical/endovascular revascularization by bypass graft

Take home messages

-Proper measurement of BP in children is a necessary skill for medical practitioners who care for children

-Height measurement is a necessary anthropometry

-There are "at risk paediatric populations" who require very early BP surveillance

-Always rule out renovascular and CoA-related HTN in children

-Always have the BP charts handy…

-Treatment goals differ in childhood HTN

Questions on Childhood Hypertension

1. Which of the following is an incorrect definition of blood pressure categories in children?

a) Normal BP (systolic and diastolic) is <90th centile in those aged 1 to <13 yrs

b) Pre-hypertension currently replaces elevated BP defined as BP (systolic and diastolic) ³90th centile to <95th centile

c) Stage 1 hypertension in ³ 13 yrs olds; 130/80 to 139/89

d) BP ³140/90mmHg is accepted as stage 2 hypertension in ages 1 to13 yrs olds

2. Which of these is correct concerning BP measurement in children?

a) Auscultate with the diaphragm of the stethoscope

b) Lower end of cuff should be >5cm above antecubital fossa

c) Cuff is inflated to 20-30mmHg above point radial pulse disappears

d) Deflate cuff at the rate of 5mmHg

3. Antihypertensive drug therapy is warranted in the following except

a) Those failing a trial of lifestyle change

b) Any stage of hypertension with comorbid conditions such as CKD or DM

c) Secondary hypertension without an associated risk factor amenable to treatment

d) Initial management measure in all cases of essential hypertension regardless of stage

4. Which of the following is not regarded as a general investigation for paediatric hypertension?

a) Urine microscopy, culture and sensitivity

b) Urinalysis

c) Serum electrolytes, urea and creatinine

d) Renal ultrasound scan in those <6 yrs of age or those with abnormal urinalysis

5. Which of these is a correct treatment goal for paediatric hypertension?

a) Reduce BP to <95th centile for hypertensive children with CKD, DM or hypertensive

b) Reduce BP to <95th centile for uncomplicated primary hypertension without hypertensive end-organ damage

c) Consider step-down or cessation of therapy in all cases of hypertension that has recorded normal readings on antihypertensive drug therapy

d) BP reduced to less than 120/80mmHg is good for children generally

Keys to the questions

Question	Correct options
1.	B
2.	C
3.	D
4.	A
5.	B

Twins or Brothers? Tackling the Foes of Measles and Rubella

Okpala Somkene

Facts on measles

-Measles is a highly infectious disease caused by a germ (virus)

-Caused by Measles Virus (Rubeola, RNA Virus, Paramyxoviridae Family)

-Spreads easily from person to person through: Coughing, Breathing, Sneezing

-Global Epidemiology: From 2022 to 2023, estimated measles cases increased by 20% worldwide, from 8,645,000 to 10,341,000; the number of countries experiencing large or disruptive outbreaks increased from 36 to 57.

-Estimated measles deaths decreased 8%, from 116,800 in 2022 to 107,500 in 2023, primarily because an increased number of cases occurred in countries with lower risk for death.

-Complications: Measles causes severe disease, with complications such as blindness, deafness, cancrum oris, brain inflammation, severe diarrhoea, and death.

-Prevention: Vaccination is the safest and cheapest way of preventing against measles

Epidemiology of Measles in Nigeria

Measles remains a burden in Nigeria: epi-week 01 – 49, 2023 and 2024.

In 2023, there were 20,065 suspected measles cases: 81 deaths out of 11,704 confirmed measles cases were recorded, this represents a 0.7% case fatality rate.

While in 2024, there were 19,801 suspected measles cases: 77 deaths out of 9,908 confirmed cases were recorded, this represents a 0.8% case fatality rate.

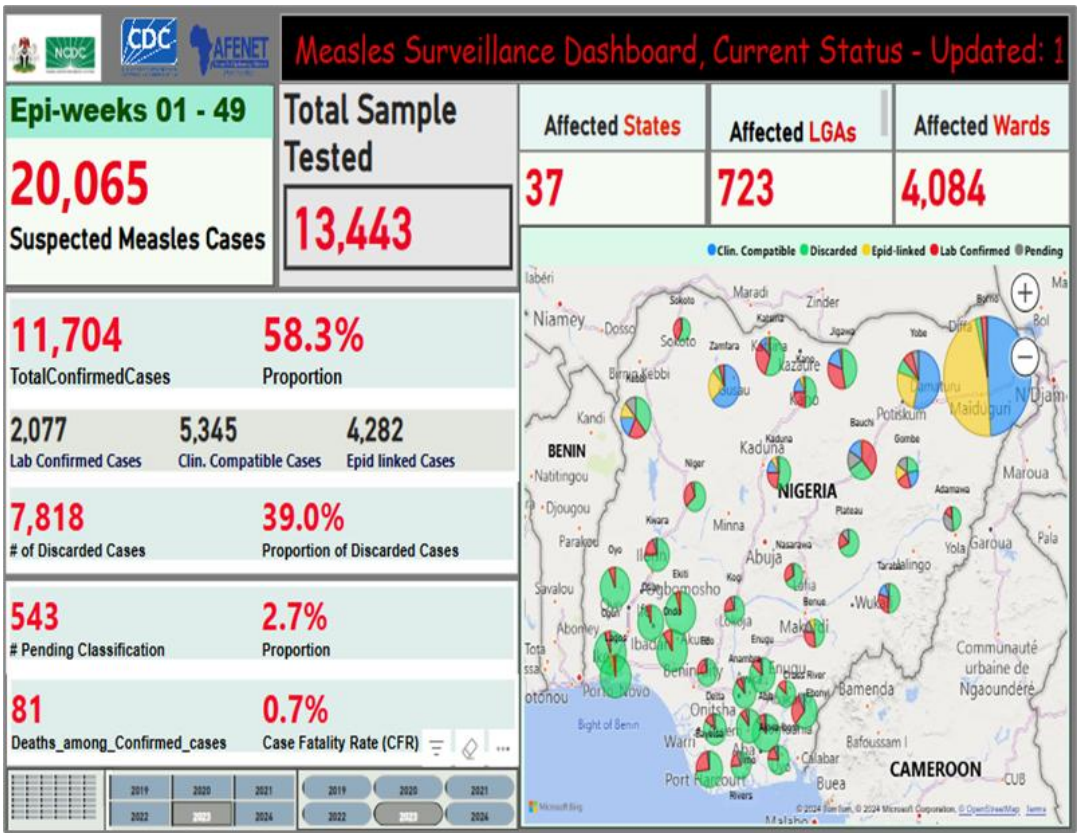


Figure 6: Epi week 01 – 49 2023

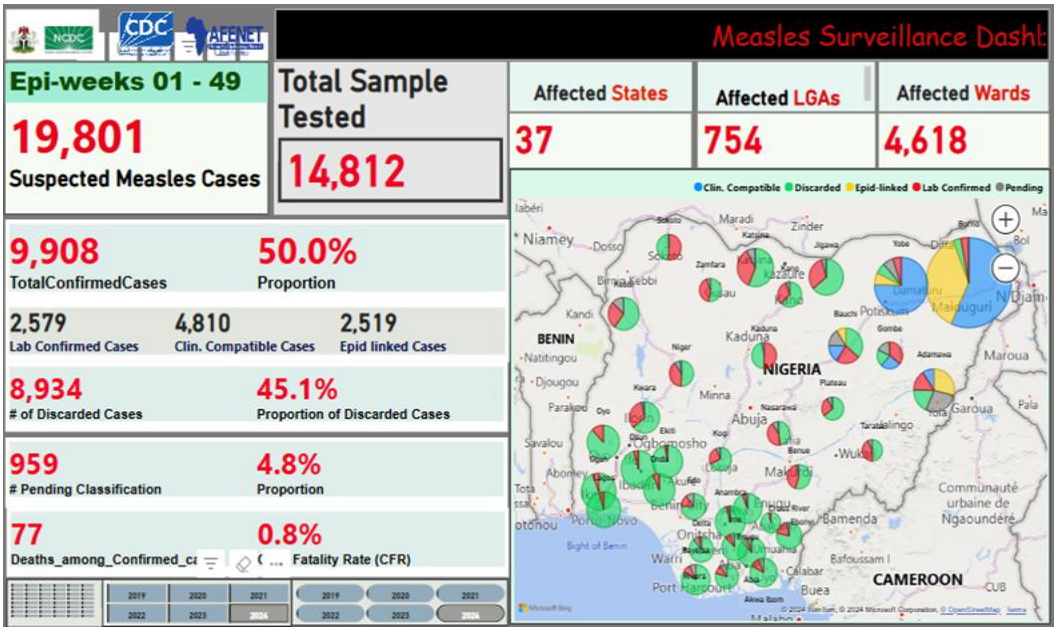


Figure 7: Epi week 01 – 49 2024

Measles Outbreak Summary: Epi-week 49, 2024

As at Epi-week 49 of 2024, a total of 306 LGAs across 36 States and the FCT have recorded a measles outbreak in 2024.

Osun had the highest number of LGAs (18) that have experienced measles outbreak this year. Followed by Oyo and Ogun States (15) while Adamawa and Bauchi recorded 14 LGAs each.

Furthermore, 298 LGAs across 37 States have ended their measles outbreak as at end of epi-week 49.

Osun (18), Oyo (15), Ogun (14), Adamawa (14), Ekiti (13) and Borno (13) are among States with the highest number of LGAs that have ended their outbreak by end of epi-week 49.

By the end of Epi-week 49, only 6 LGAs (Damban, Asa, Edu, Abeokuta North, Sardauna and Tarmuwa) across 5 States (Bauchi, Kwara, Ogun, Taraba and Yobe) still have ongoing measles outbreak.

Two LGAs (Lafia and Shendam) in Nasarawa and Plateau States respectively recorded new measles outbreak in Epi-week 49.

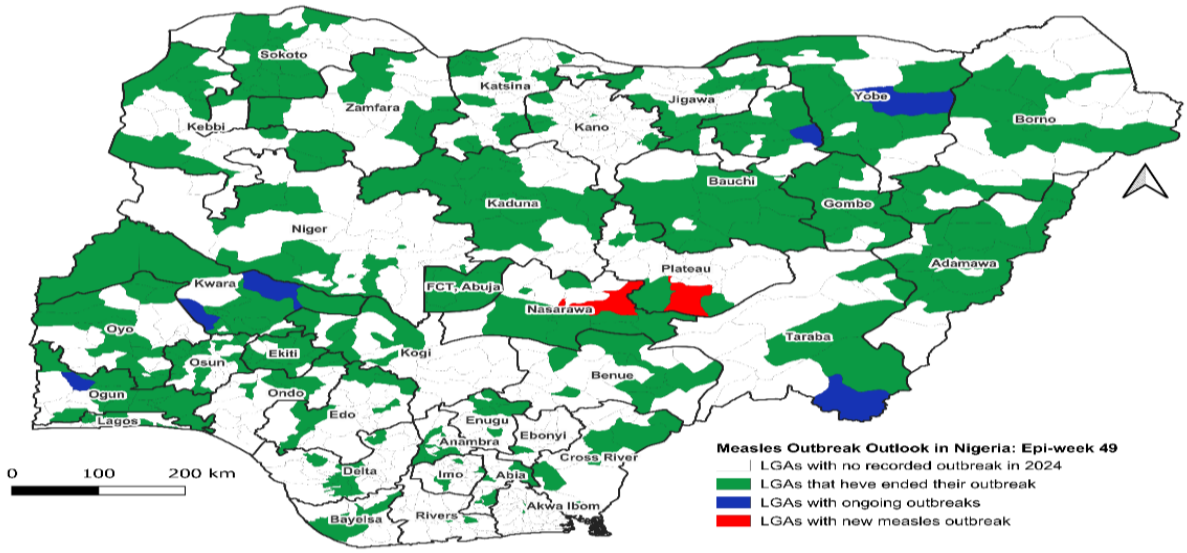


Figure 8: Map of Nigeria showing measles outbreak status by states/LGAs as at Epi-week 49, 2024

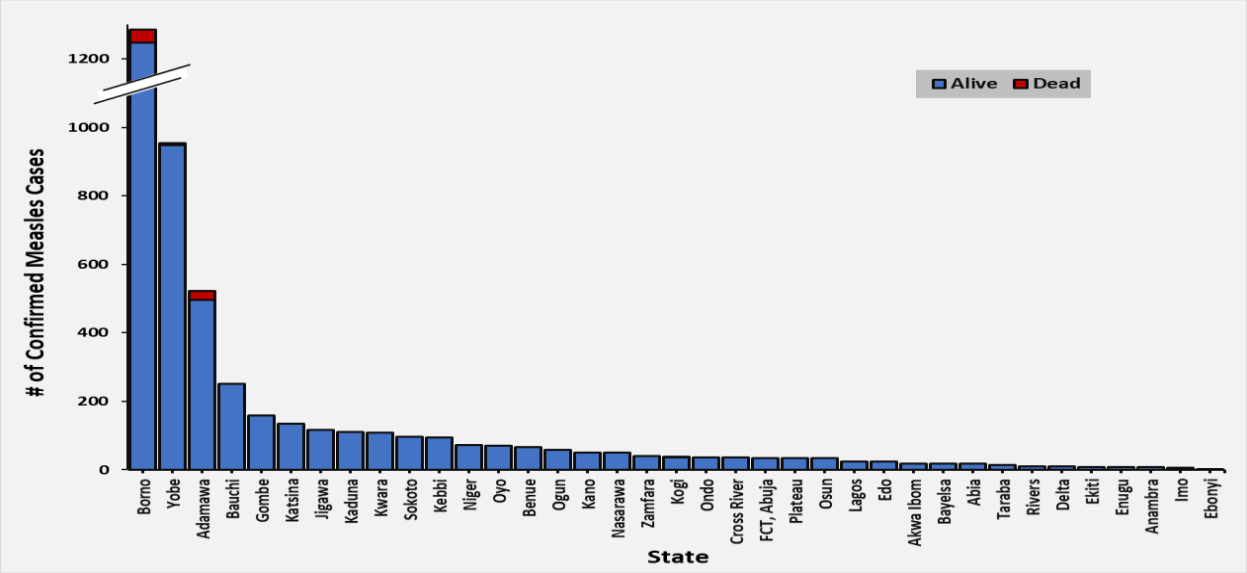


Figure 9: Distribution of Confirmed Measles cases and Outcomes by States: Epi-week 01 – 49, 2024

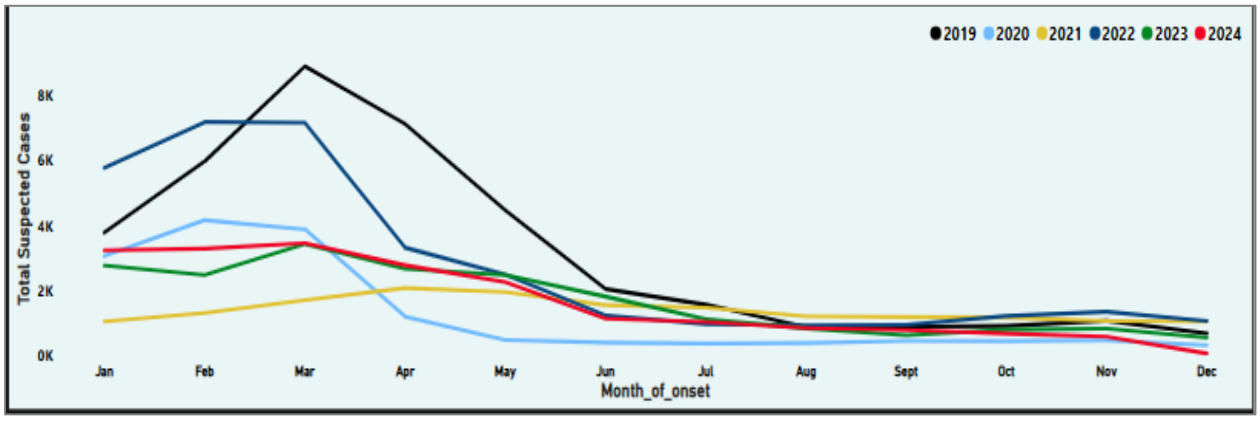


Figure 10: National Line Graph of Suspected Measles cases, 2019 – 2024

The Peak period for transmission is February-March, it is more effective to vaccinate before the measles incidence starts rising in October – November.

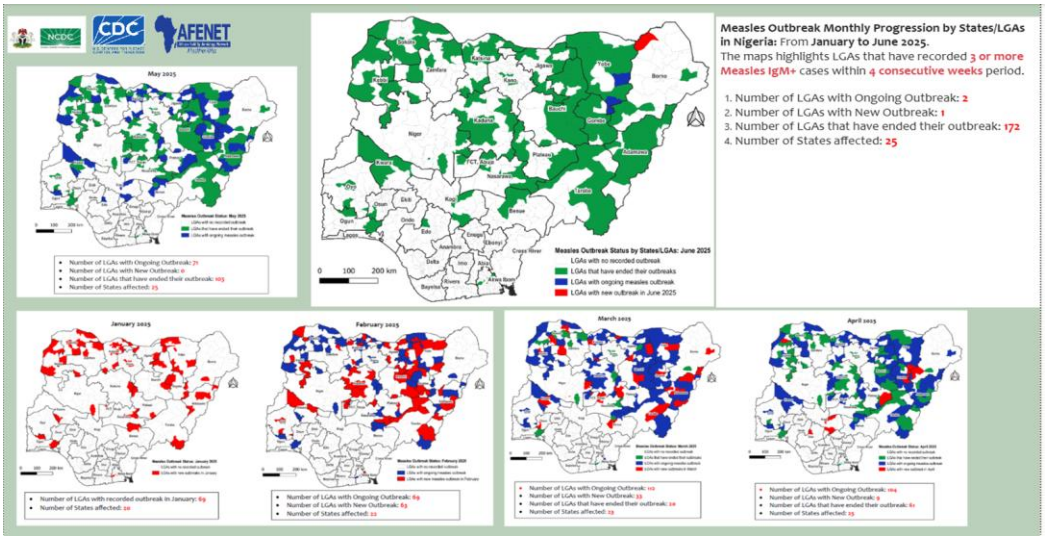


Figure 11: Monthly Measles Outbreak Status by states/LGAs: January – June 2025

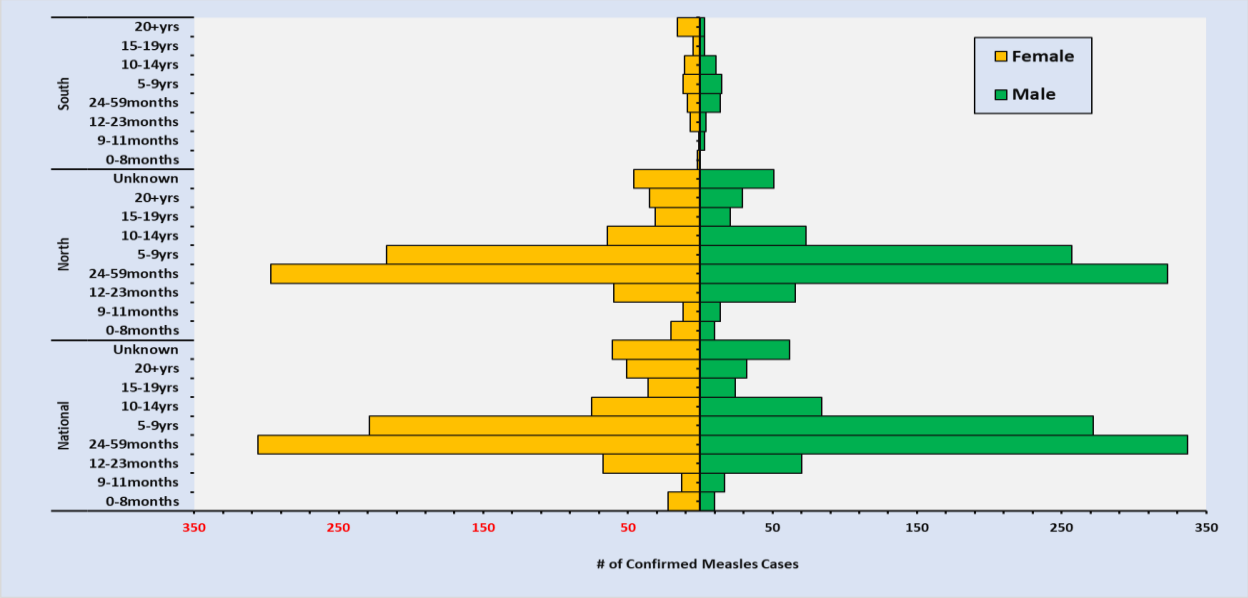


Figure 12: Age and Gender Distribution of Confirmed Measles Cases in Nigeria: epi-week 01–25, 2025

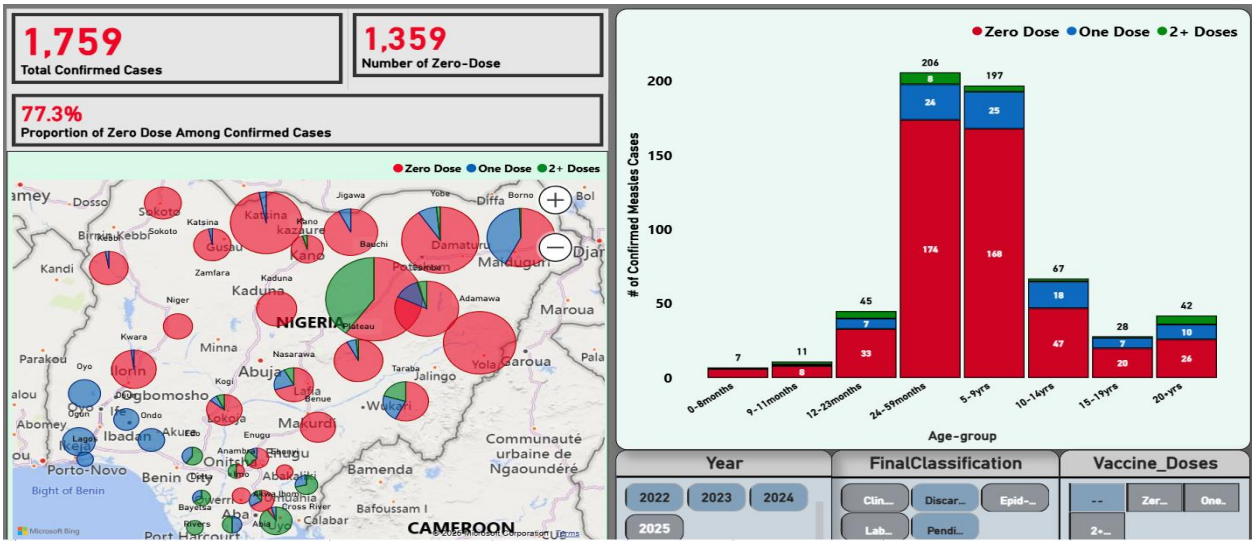


Figure 13: Distribution of Measles Vaccination Status among Confirmed Cases by Age-group: Epi-week 01-25, 2025

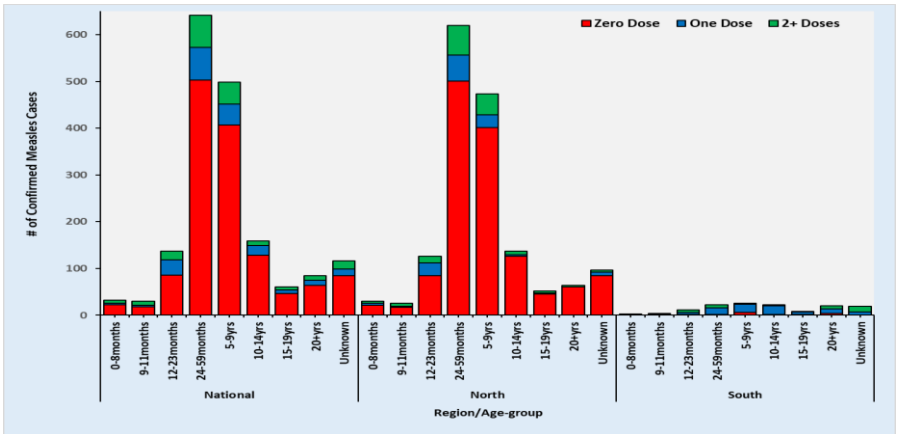


Figure 14: Vaccination status of confirmed measles cases by Age-group and Regions in Nigeria: Epi-week 01-25, 2025

Clinical course of measles

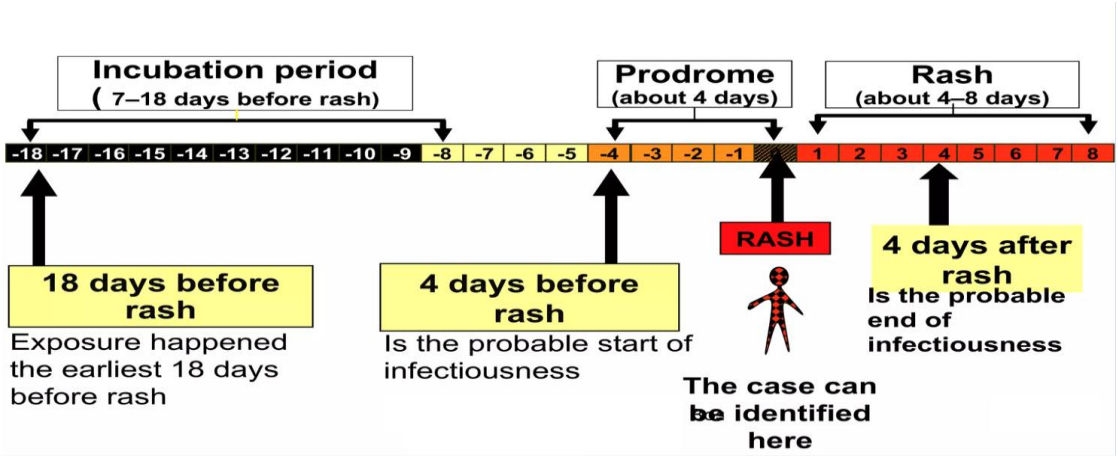


Figure 15: Clinical course of measles

Immune response in acute infection

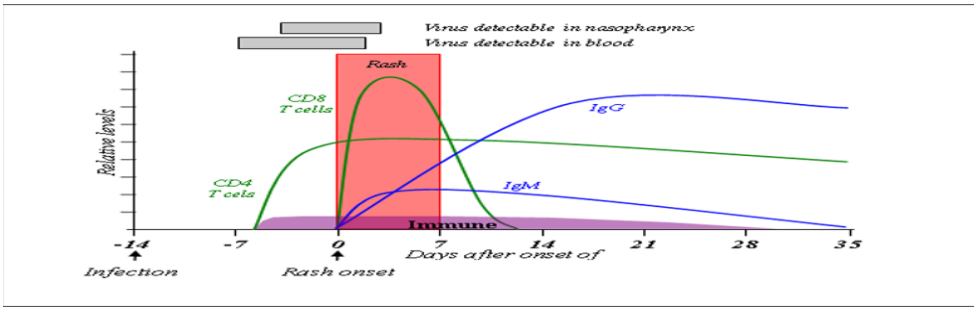


Figure 16: Immune response in acute Measles infection

FACTS ON RUBELLA

Rubella, also known as German measles, is a contagious viral infection caused by the rubella virus. It primarily affects children and young adults, but it can occur at any age. Rubella is generally mild in healthy individuals but can have severe consequences if contracted by a pregnant woman during her first trimester.

Aetiology

Low-grade fever, Mild rash (pink or light red spots starting on the face and spreading to the body), Swollen lymph nodes, especially behind the ears and at the back of the neck, Joint pain (more common in adults, especially women), Cold-like symptoms (e.g., runny nose, sore throat).

Global epidemiology

97% reduction in reported rubella cases, from 670,894 cases in 102 countries in 2000 to 17,865 cases in 78 countries in 2022. As of January 2024, 175 out of 194 countries had introduced rubella vaccines into their national immunization programs, with global coverage estimated at 69%. CRS rates remain highest in regions with lower vaccination coverage. In 2024, out of the 11,300 confirmed measles cases reported by surveillance, 7% were rubella cases, predominantly from southern Nigeria.

Transmission

Spread through respiratory droplets when an infected person coughs or sneezes. Close contact with an infected individual.

Complications

If a pregnant woman contracts rubella, especially during the first trimester, it can cause congenital rubella syndrome (CRS). CRS can lead to: Birth defects like blindness, deafness, holes in the heart and neural impairment.

Prevention

The most effective way to prevent rubella is through vaccination with the Measles-Rubella (MR) vaccine. It is typically administered to children between 9 months to 15 years.

Facts about Congenital Rubella Syndrome (CRS)

- CRS occurs when a pregnant woman is infected with rubella, and the virus is transmitted to the developing fetus through the placenta
- Leads to severe birth defects or fetal death.
- Affects multiple systems, causing deafness, heart defects, cataracts, intellectual disabilities, and low birth weight.
- It is a major preventable cause of lifelong disabilities in areas with low vaccine coverage.
- The best prevention is vaccination with rubella-containing vaccines (e.g., MR or MMR). Routine immunization and mass campaigns, especially targeting women of childbearing age, are crucial to protecting pregnancies. High vaccine coverage can eliminate rubella and CRS, reducing health, social, and economic burdens.

Overview of Rubella in Nigeria

- Under-Reported Cases: Rubella burden in Nigeria is under documented due to limited surveillance. However, seroprevalence studies indicate high levels of susceptibility in women of childbearing age
- Congenital Rubella Syndrome (CRS):
- In 2024, the global burden of Congenital Rubella Syndrome (CRS) continues to be a significant public health concern, with approximately 100,000 babies born with CRS each year
- The high susceptibility to Rubella among women of childbearing age contributes to CRS cases in the country

Incidence of Rubella IgM-positive cases by states (2019 -2023)

Rubella incidence in majority (19) of States is greater than or equal to 25 cases/ million population Incidence is higher in South compared to Northern States.

Reference

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4. Durowade KA, Omotosho I, Osagbemi GK. Rubella Transmissibility and Reproduction Number (Ro): A Critical Appraisal of the Prospects for its Control in Nigeria. *Niger Postgrad Med J* 2020;27(3):156-62. https://doi.org/10.4103/npmj.npmj_84_20.
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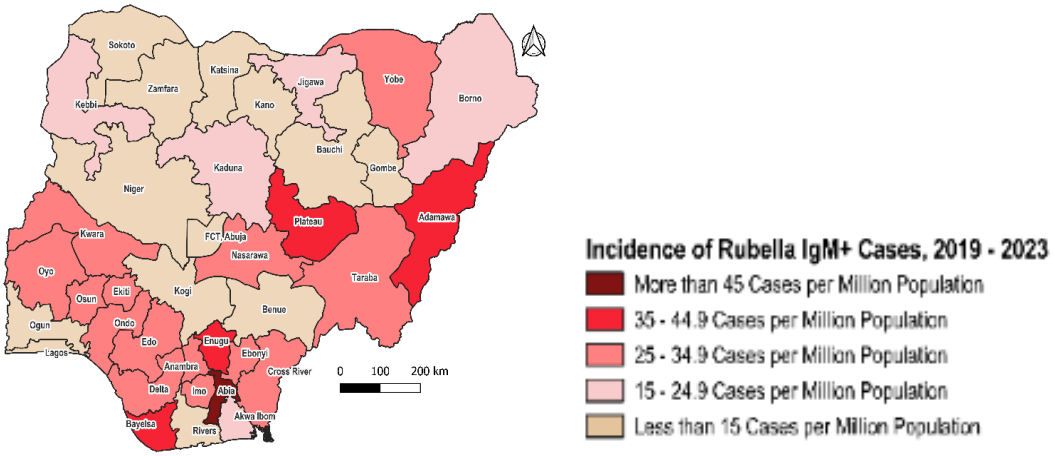


Figure 17: Incidence of Rubella IgM cases in Nigeria 2019 -2023

In 2024, there were 8,928 suspected cases of Rubella, with 627 Rubella IgM+ cases with a 7.0% positivity rate while 2 cases resulted to CRS.

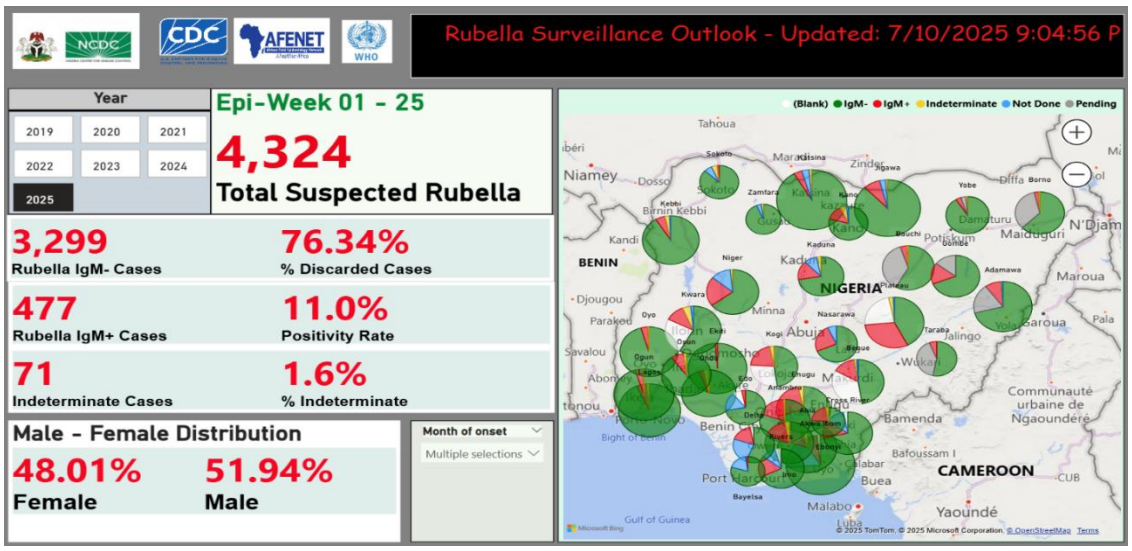


Figure 18: Rubella cases Outlook in Nigeria: epi-week 01 – 49, 2024

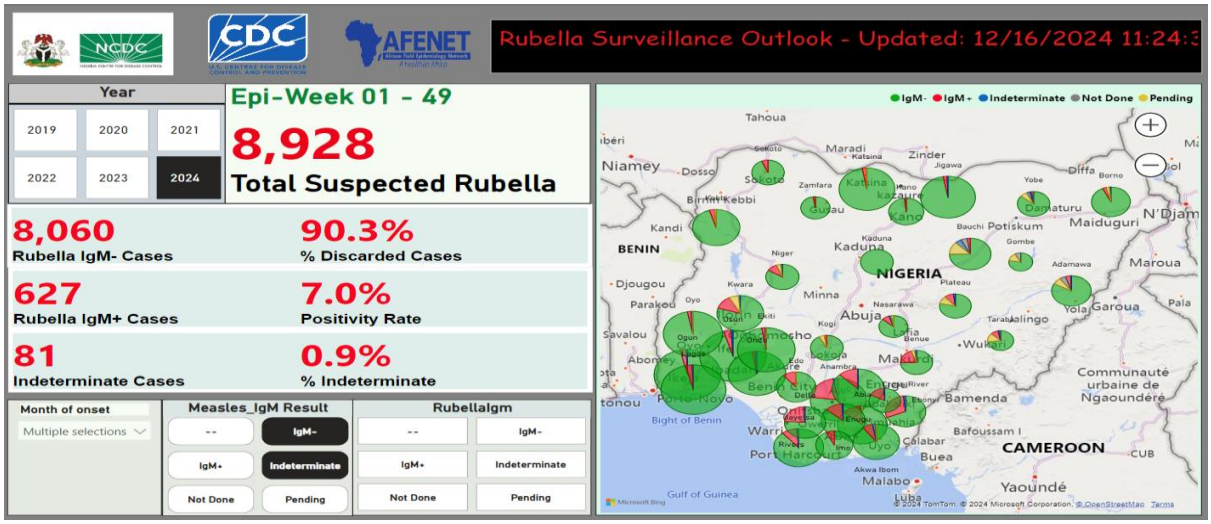


Figure 19: Epi-Curve of Rubella Cases by National and Regions: Epi-week 01 - 45 of 2024

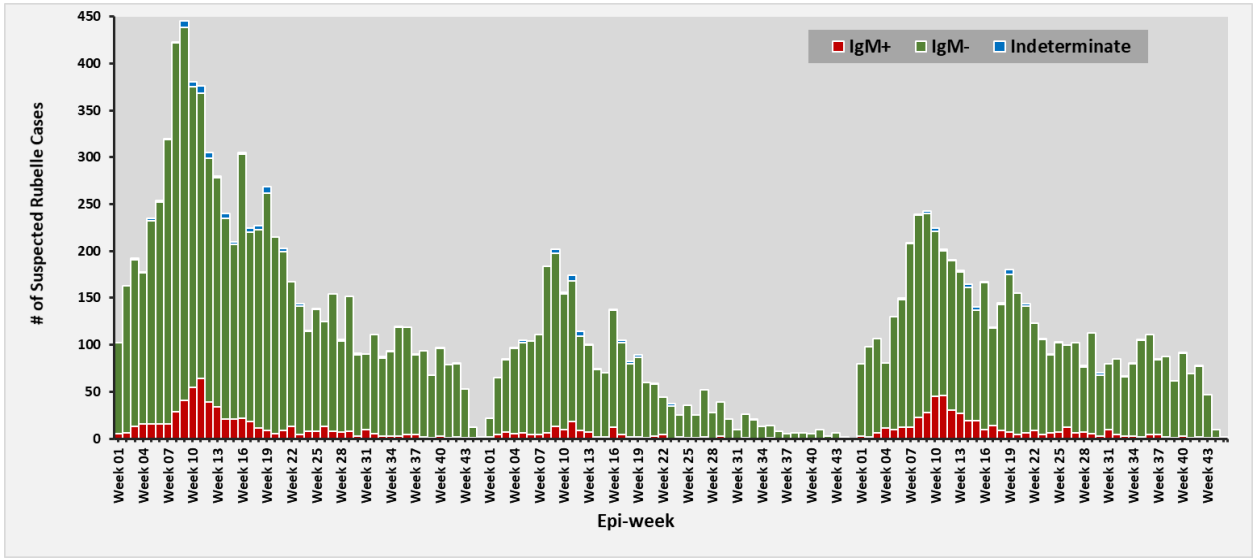


Figure 20: Rubella cases Outlook in Nigeria: epi-week 01 – 25, 2025

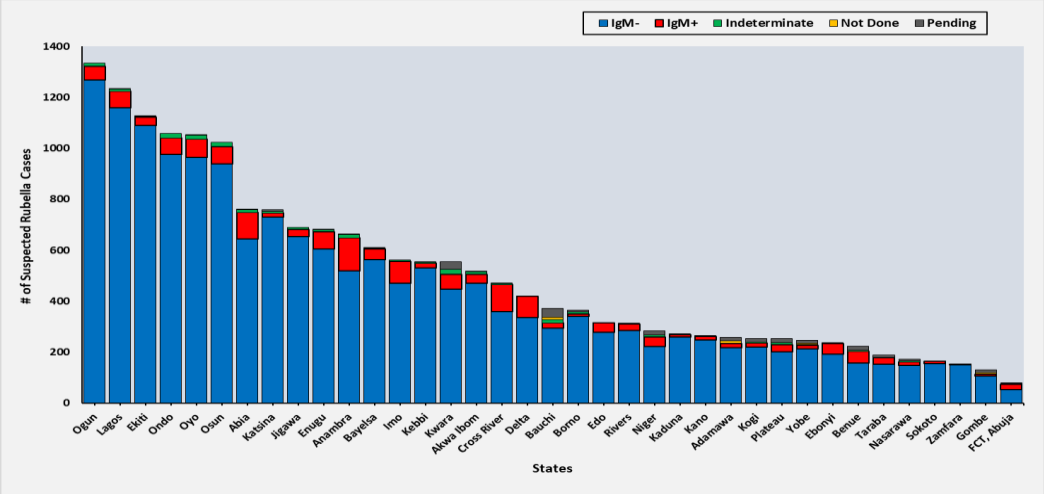


Figure 21: Distribution of Rubella Cases by States in Nigeria: epi-week 01 – 12, 2025

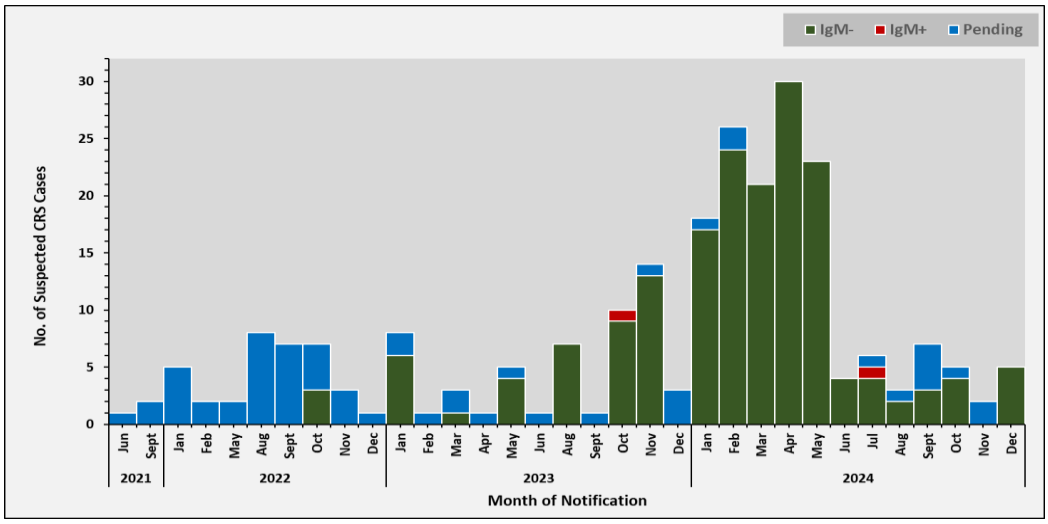


Figure 22: CRS Trend of Notification: 2021 – 2024

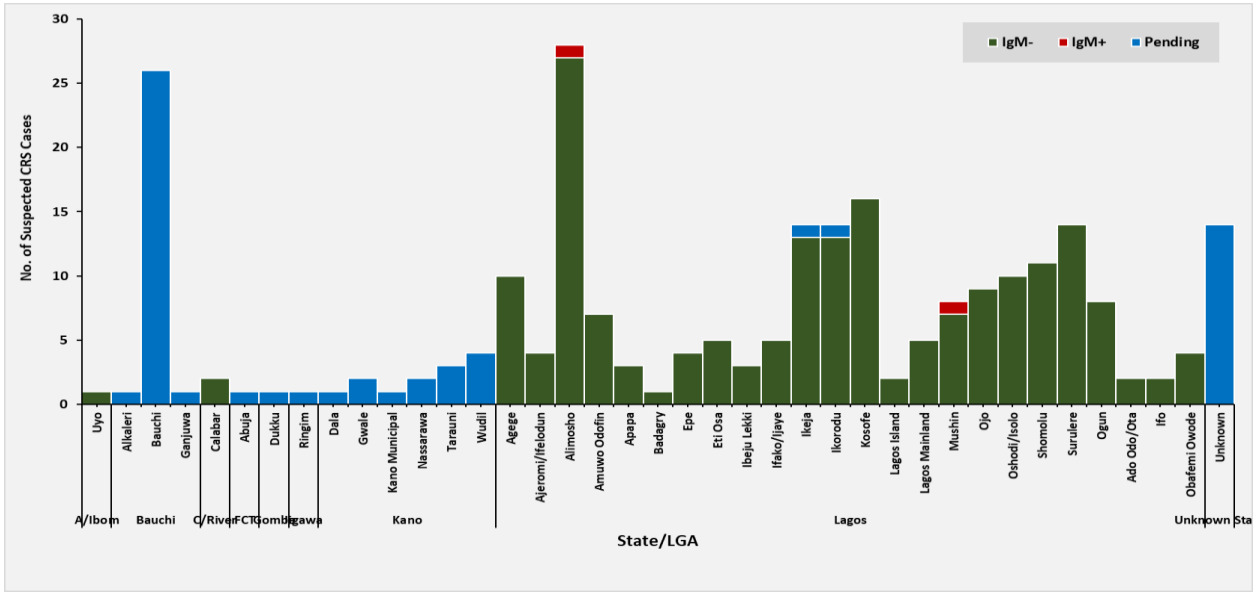


Figure 23: Distribution of Suspected CRS Cases by LGAs in Nigeria

Table VII: CRS Reporting was from Sentinel Sites only (Summary)

S/N	CRS Sentinel Sites	State	Zone	No. of Suspected Cases uploaded	No. of Cases Tested for Rubella IgM	No. of Rubella IgM+ Cases	Remarks
1	National Hospital Abuja	FCT	North-Central	1	1	0	Results not uploaded
2	Lagos University Teaching Hospital	Lagos	South-West	178	178	2	Completed
3	University of Calabar Teaching Hospital	Cross River	South-South	3	3	0	Results not uploaded
4	Lagos State University Teaching Hospital	Lagos	South-West	3	3	0	Results not uploaded
5	Aminu Kano Teaching Hospital	Kano	North-West	27	27	0	Results not uploaded
6	University of Nigeria Teaching Hospital	Enugu	South-East	0	0	0	Results not uploaded
7	Jos University Teaching Hospital	Plateau	North-Central	0	0	0	Results not uploaded
8	Abubakar Tafawa Balewa Teaching Hospital	Bauchi	North-East	30	30	0	Results not uploaded
Total				**242**	**242**	**2**	

Table VIII: Socio-Demographic Distribution of Suspected CRS Cases in Nigeria: 2021 – 2024

Socio-demographics	Testing Results			Total (Proportion)
	IgM-	IgM+	Pending	
Sex				
Female	74	1	23	98 (40.7%)
Male	106	1	36	143 (59.3%)
Age-groups				
0 - 28 days	7	0	9	16 (6.6%)
1 - 11 months	158	2	37	197 (81.4%)
12 - 23 months	12	0	8	20 (8.3%)
2yrs and above	3	0	6	9 (3.7%)
State of Residence				
Akwa-Ibom	1	0	0	1 (0.4%)
Bauchi	0	0	28	28 (11.6%)
Cross River	2	0	0	2 (0.8%)
FCT	0	0	1	1 (0.4%)
Gombe	0	0	1	1 (0.4%)
Jigawa	0	0	1	1 (0.4%)
Kano	0	0	13	13 (5.4%)
Lagos	169	2	2	173 (71.5%)
Ogun	8	0	0	8 (3.3%)
Unknown	0	0	14	14 (5.8%)

Symptoms and signs of Rubella

Rubella cases present with FEVER which is mild, RASH that is discrete and appears like a heat rash.

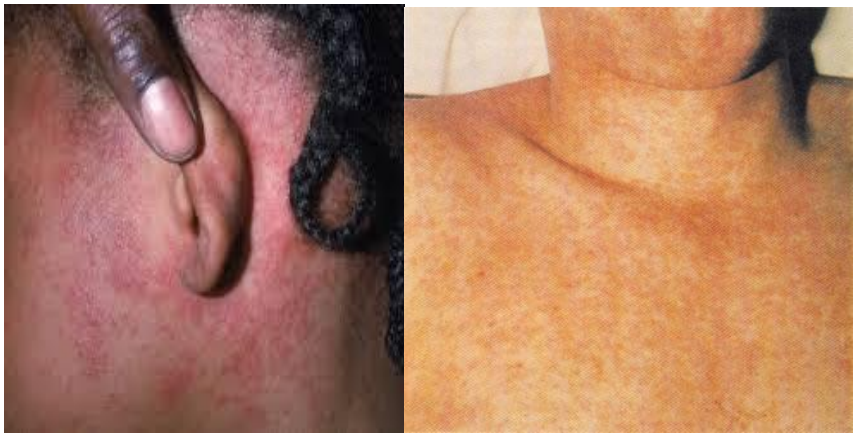


Figure 24: Rash in Rubella

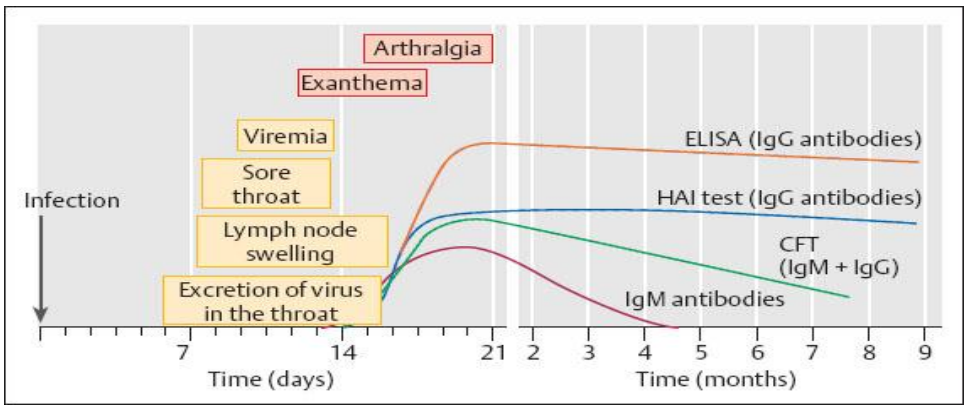


Figure 25: Rubella clinical course and serology

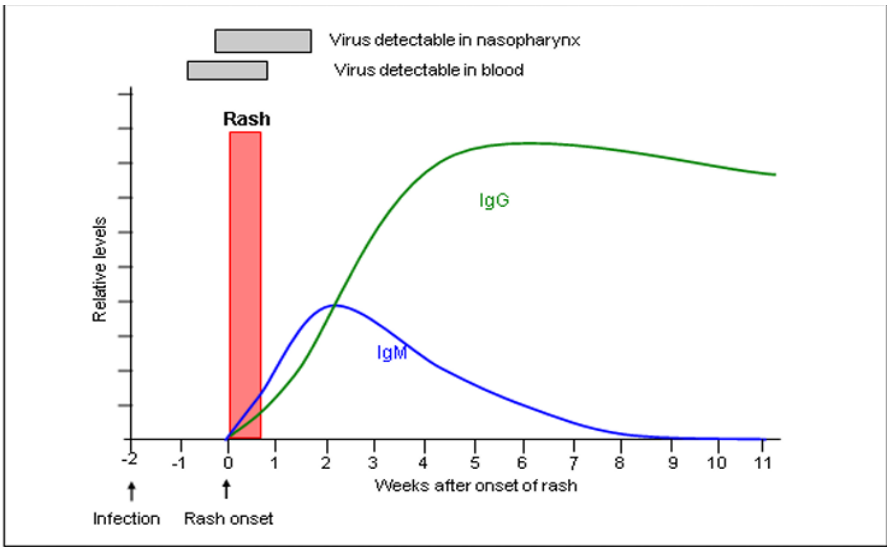


Figure 26: Immune response in postnatal rubella infection

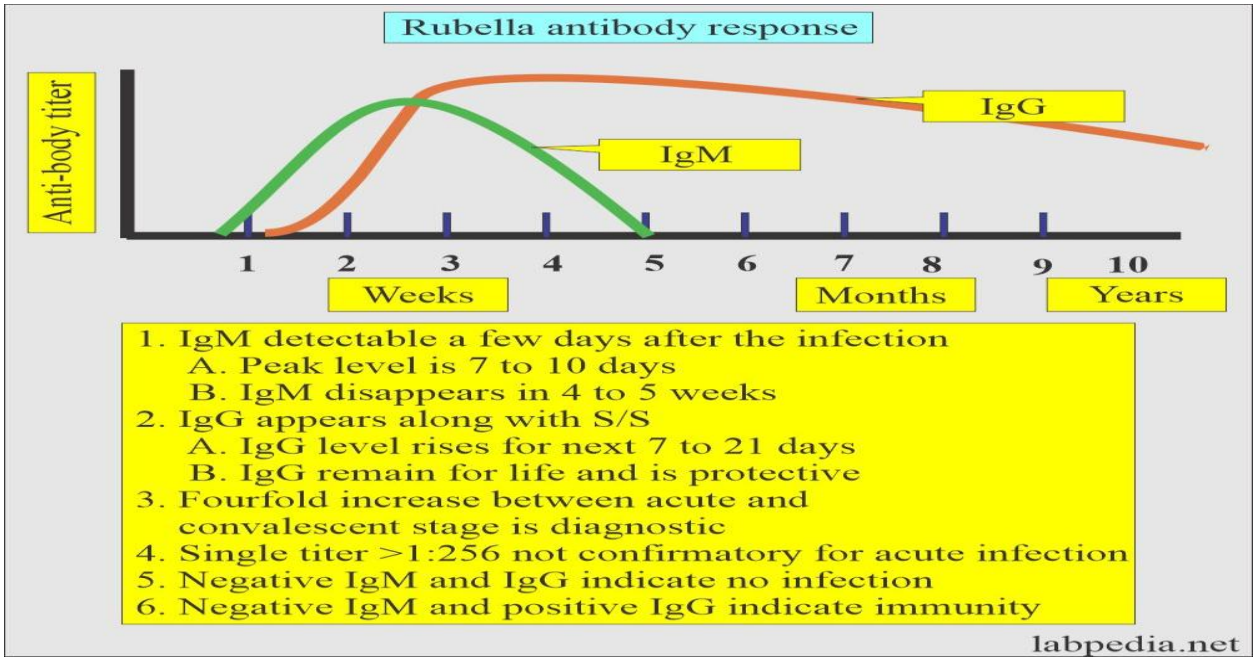
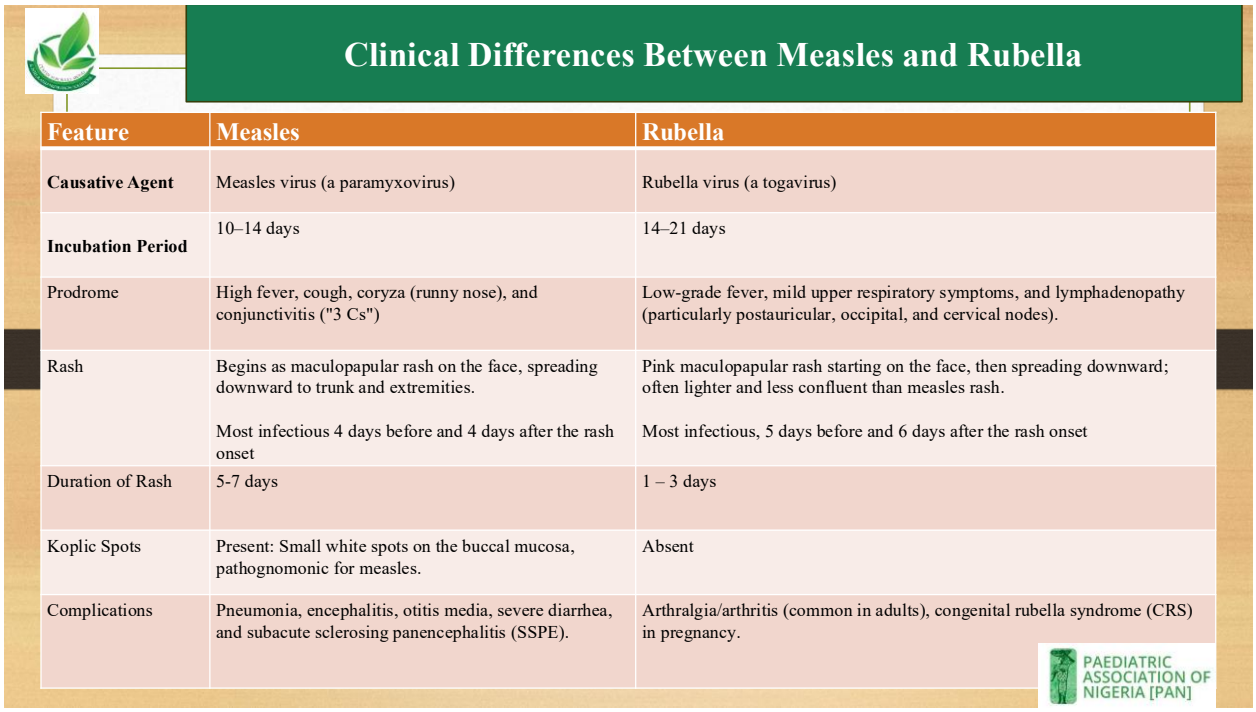


Figure 27: Rubella antibody response

Table IX: Differences between Measles and Rubella



Clinical Differences Between Measles and Rubella

Feature	Measles	Rubella
Causative Agent	Measles virus (a paramyxovirus)	Rubella virus (a togavirus)
Incubation Period	10–14 days	14–21 days
Prodrome	High fever, cough, coryza (runny nose), and conjunctivitis ("3 Cs")	Low-grade fever, mild upper respiratory symptoms, and lymphadenopathy (particularly postauricular, occipital, and cervical nodes).
Rash	Begins as maculopapular rash on the face, spreading downward to trunk and extremities. Most infectious 4 days before and 4 days after the rash onset	Pink maculopapular rash starting on the face, then spreading downward; often lighter and less confluent than measles rash. Most infectious, 5 days before and 6 days after the rash onset
Duration of Rash	5-7 days	1 – 3 days
Koplic Spots	Present: Small white spots on the buccal mucosa, pathognomonic for measles.	Absent
Complications	Pneumonia, encephalitis, otitis media, severe diarrhea, and subacute sclerosing panencephalitis (SSPE).	Arthralgia/arthritis (common in adults), congenital rubella syndrome (CRS) in pregnancy.

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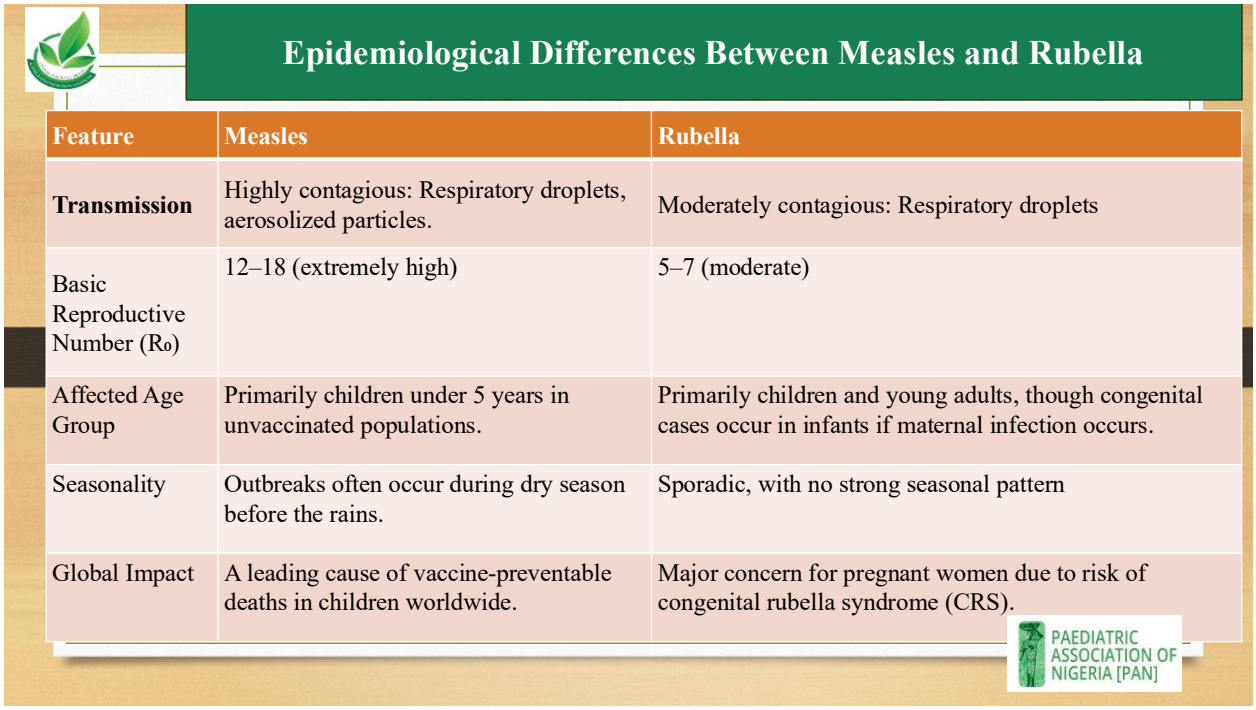
Table X: Epidemiological differences between Measles and Rubella



Epidemiological Differences Between Measles and Rubella

Feature	Measles	Rubella
Transmission	Highly contagious: Respiratory droplets, aerosolized particles.	Moderately contagious: Respiratory droplets
Basic Reproductive Number (R_0)	12–18 (extremely high)	5–7 (moderate)
Affected Age Group	Primarily children under 5 years in unvaccinated populations.	Primarily children and young adults, though congenital cases occur in infants if maternal infection occurs.
Seasonality	Outbreaks often occur during dry season before the rains.	Sporadic, with no strong seasonal pattern
Global Impact	A leading cause of vaccine-preventable deaths in children worldwide.	Major concern for pregnant women due to risk of congenital rubella syndrome (CRS).

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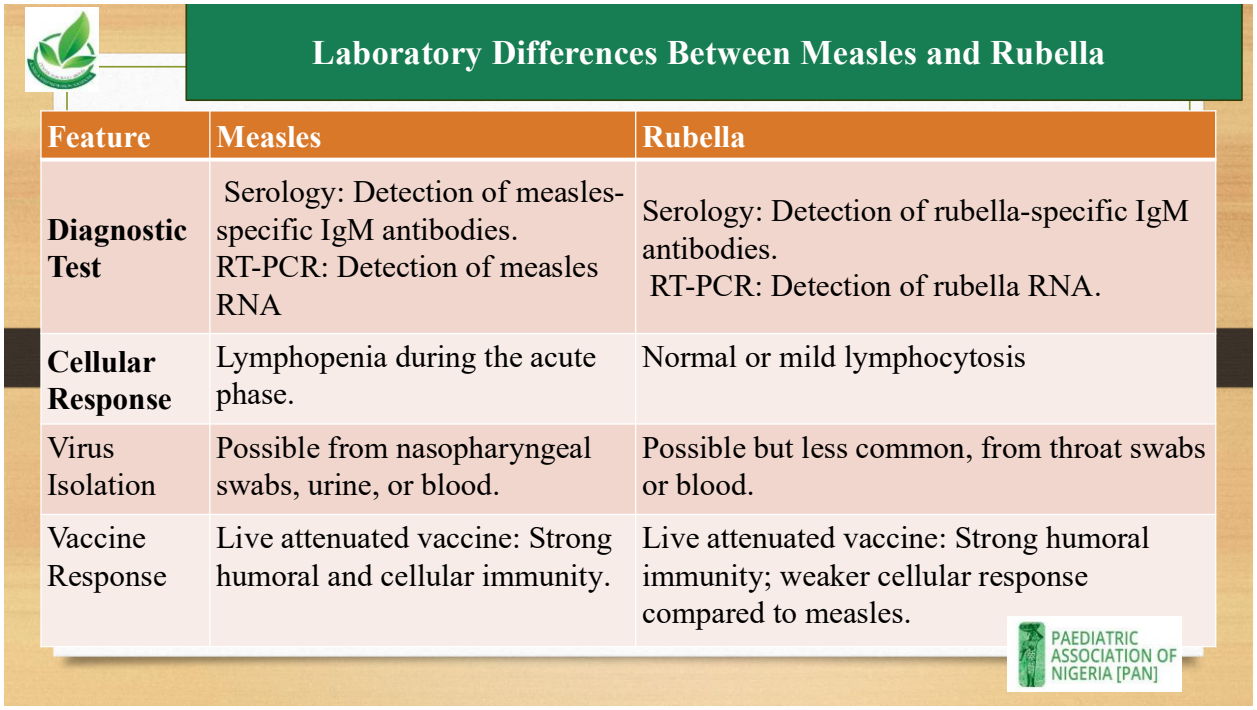
Table XI: Laboratory difference between Measles and Rubella



Laboratory Differences Between Measles and Rubella

Feature	Measles	Rubella
Diagnostic Test	Serology: Detection of measles-specific IgM antibodies. RT-PCR: Detection of measles RNA	Serology: Detection of rubella-specific IgM antibodies. RT-PCR: Detection of rubella RNA.
Cellular Response	Lymphopenia during the acute phase.	Normal or mild lymphocytosis
Virus Isolation	Possible from nasopharyngeal swabs, urine, or blood.	Possible but less common, from throat swabs or blood.
Vaccine Response	Live attenuated vaccine: Strong humoral and cellular immunity.	Live attenuated vaccine: Strong humoral immunity; weaker cellular response compared to measles.

PAEDIATRIC ASSOCIATION OF NIGERIA [PAN]

CLINICAL QUIZ

A 6-year-old boy presented with blood-stained vomitus, dark stools, and palor. The symptoms started following the administration of Camosunate®, paracetamol and Ibuprofen for a febrile illness. He was weak, but there was no difficulty with breathing. He had the same symptoms three years earlier, but they were milder and there was no palor or weakness. He was delivered as a preterm, VLBW baby and had neonatal jaundice for which an exchange blood transfusion was done.

Endoscopic studies were done as part of his evaluation, and treatment was instituted, including PPI therapy and blood transfusion.

The endoscopic findings are depicted in the picture shown below.

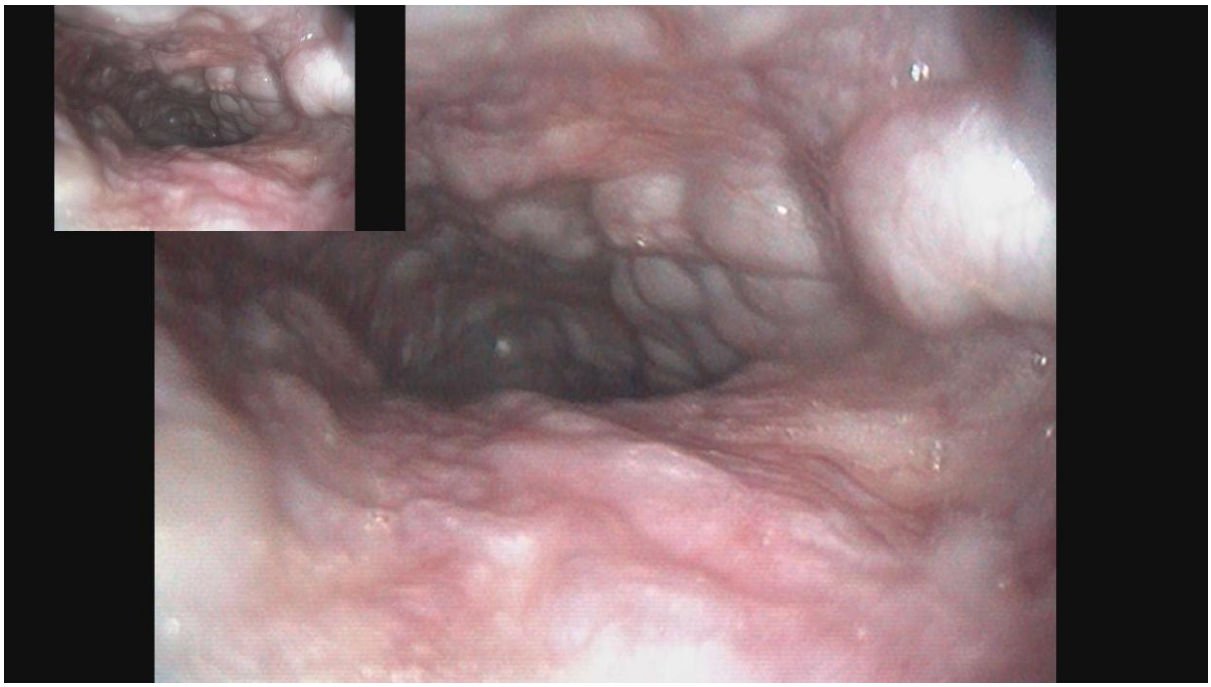


Figure 28: Endoscopic findings

Questions

1. Which of the following is the most likely diagnosis?

a) Peptic ulcer disease
b) Gastritis
c) Gastrointestinal polyps
d) Oesophageal varices

2. Which of the following is a danger signal in the condition depicted?

a) Grade 1 disease
b) Red wale markings
c) Multiple superficial erosions
d) White mucosal markings

3. Which of the following is an important risk factor for the condition depicted in the patient?

a) Being a Low birth weight baby
b) Febrile illness
c) Previous Exchange blood transfusion
d) Age

4. In the emergency management of a child with acute upper GI bleeding, the first priority is:

a) Start intravenous proton pump inhibitors
b) Establish airway and secure breathing & circulation
c) Arrange urgent endoscopy
d) Send blood for clotting profile and LFT

5. Which of the following drugs is used for secondary prophylaxis of variceal bleeding?

a) Omeprazole
b) Prednisolone
c) Propranolol
d) Furosemide

6. Which of the following is a useful procedure for the management of variceal bleeding?
   a) Percutaneous liver biopsy
   b) Hepatic artery embolisation
   c) Endoscopic retrograde cholangiopancreatography
   d) Transjugular Intrahepatic Portosystemic Shunt

Question	***Answer***
1	D
2	B
3	C
4	B
5	C
6	D

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