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Differential Diagnosis in **PEDIATRICS**

Editor-in-Chief
K Nedunchelian

Academic Editor
P Ramachandran

Executive Editor
S Thangavelu

Managing Editor
NC Gowrishankar



Foreword
Santosh T Saons





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Indian Journal of Practical Pediatrics

Foreword

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Baldev S Prajapati, Rajal B Prajapati

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Fever of Unknown Origin

Baldev S Prajapati, Rajal B Prajapati

DEFINITION

Fever of unknown origin (FUO) is defined as fever (rectal temperature $> 38^{\circ}\text{C}$) of more than 3 weeks duration, documented by a healthcare provider on several occasions, for which the cause could not be identified after 3 weeks of an evaluation as an outpatient or after 1 week of evaluation in a hospital setting.

Reasonable working definition of FUO for clinical purpose is, fever of 38.3°C (101°F), measured on several occasions, lasting for more than a week in whom no diagnosis is apparent after initial outpatient or inpatient evaluation which includes detailed history, thorough physical examination and initial laboratory assessment.

Fever without focus is defined as fever of < 1 week duration where no diagnosis is apparent after detailed history and physical examination. FUO is differentiated from fever without focus for several reasons,

- Differential diagnosis and most frequent causes are different in each category, infection being the most common cause of fever without focus while collagen vascular diseases, malignancies and chronic infections are more common with FUO.
- Emergency testing and evaluation are needed in a case of fever without focus while patients with FUO generally do not need emergency assessment.
- Empirical therapy is not indicated for FUO whereas empirical antibiotic therapy is used in selected group of infants and young children with fever without focus.

TYPES OF FEVER OF UNKNOWN ORIGIN

- Classic FUO
- Nosocomial FUO
- HIV related FUO
- Neutropenic FUO.

CAUSES

Fever of unknown origin is usually uncommon presentation of a common disease. Infectious diseases, connective tissue disorders, autoimmune diseases and malignancies are the most common causes of FUO in children. Drug fever, factitious fevers and periodic fever syndromes are responsible for a few cases of FUO. **Flowcharts 5.5.1 and 5.5.2** show the list of common causes of FUO in children.

Epidemiologically relevant infections should be considered first in differential diagnosis and ruled out by appropriate clinical, laboratory, and radiological evaluation. Deep-seated abscesses like pelvic abscess, subdiaphragmatic, perinephric, psoas abscess, liver abscess, dental abscess,

and brain abscess should always be considered. Bacterial endocarditis should be high on suspicion.

Nosocomial, HIV-related and neutropenic FUO require specific approach for each category.

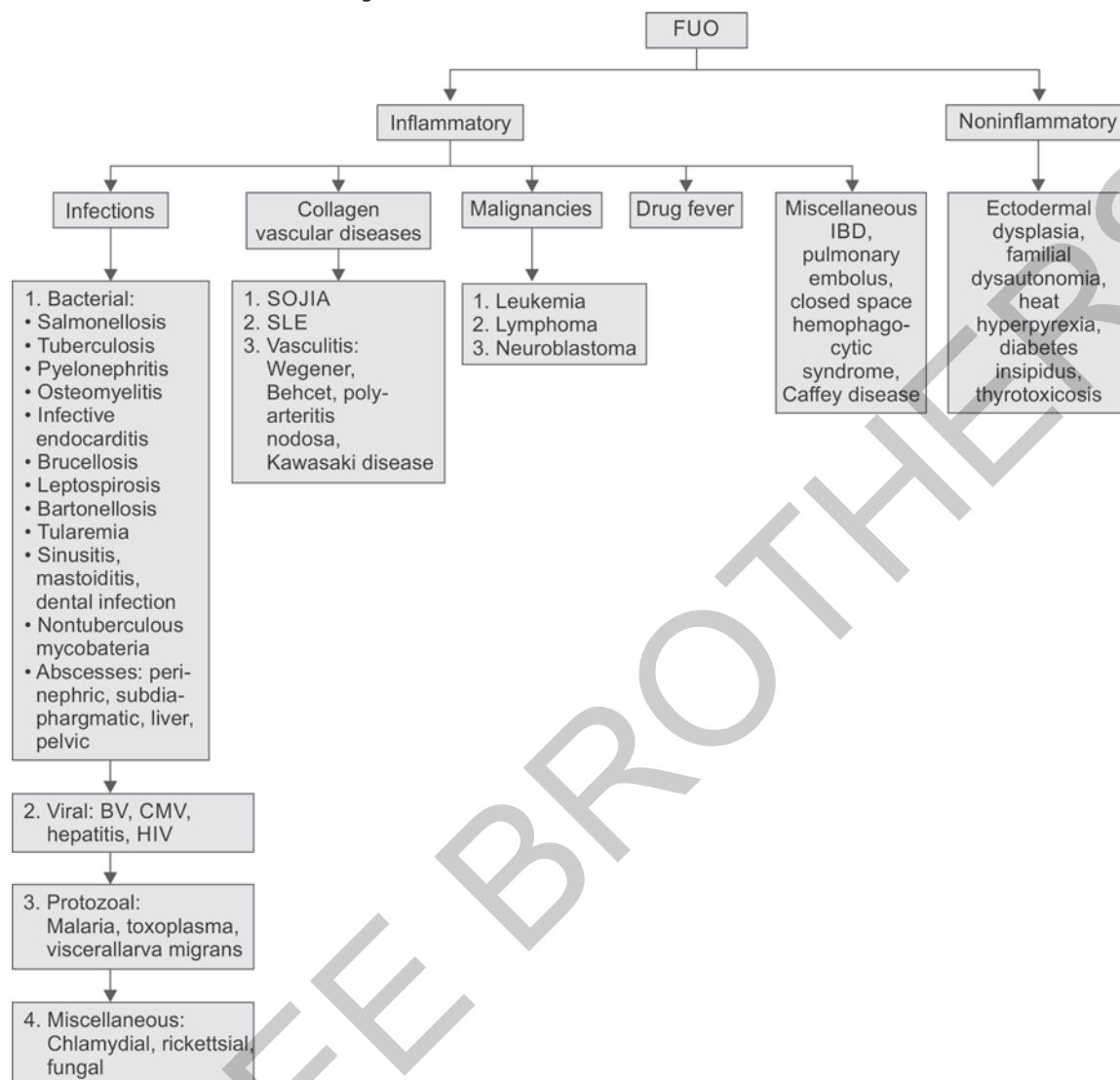
APPROACH

- Documentation of fever
- Detailed history
- Thorough physical examination
- Initial laboratory investigations
- Relevant radio images
- Repeated history and physical examination by same and different medical persons
- Repeat basic laboratory investigations
- Additional tests as indicated.

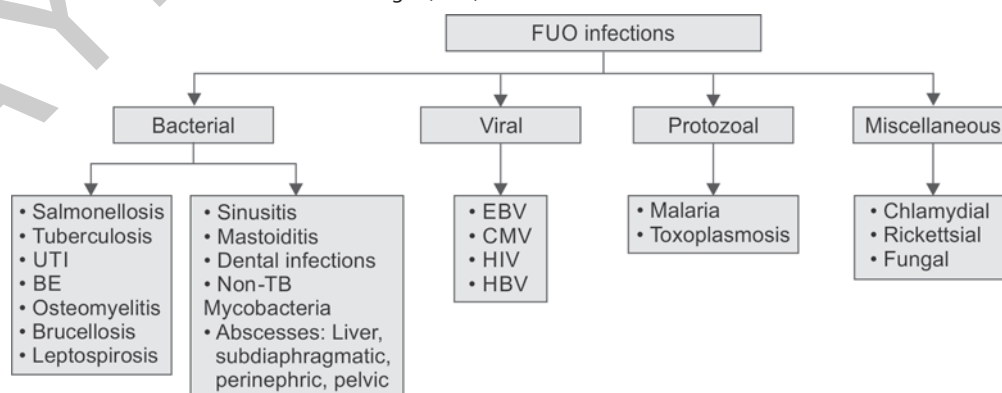
HISTORY TAKING

The history should include details of fever like onset, duration, intensity, frequency, pattern of fever, response to antipyretics, etc. The specific clues should be noted as below:

- Fever duration, pattern, and height PUO of > 6 months $\rightarrow$ Granulomatous or autoimmune disease
- Associated constitutional symptoms
- Response to NSAIDs $\rightarrow$ No response in noninflammatory fevers
- History of associated sweating:
 - Fever, heat intolerance and sweating $\rightarrow$ Thyrotoxicosis
 - Fever, heat intolerance and absence of sweating $\rightarrow$ Ectodermal dysplasia
- Associated complaints:
 - Spontaneously resolved red eyes $\rightarrow$ Kawasaki disease (KD)
 - Persistent red weeping eyes $\rightarrow$ SLE
 - Persistent nasal discharge $\rightarrow$ Sinusitis
- Contact with infected or ill person $\rightarrow$ Diagnosis of index case
- Exposure to animals/insects
- Cats $\rightarrow$ Leptospirosis, cat-scratch disease, *Toxocara cati*, tularemia
- Dogs $\rightarrow$ Rickettsial disease, leptospirosis, tularemia, *Toxocara canis*
- Ticks $\rightarrow$ Rickettsial disease, tularemia
- Unpasteurized milk or raw meat $\rightarrow$ Brucellosis, toxoplasmosis
- Pica $\rightarrow$ Visceral larva migrans, toxoplasmosis
- Medication $\rightarrow$ Drug fever
- Abdominal surgery Intra-abdominal abscesses
- History of recent vaccination or receipt of any blood component.

Flowchart 5.5.1: Causes of fever of unknown origin.

(BV: bacterial vaginosis; CMV: cytomegalovirus; FUO: fever of unknown origin; IBD: inflammatory bowel disease)

Flowchart 5.5.2: Infectious causes of fever of unknown origin (FUO).

(UTI: urinary tract infection; TB: tuberculosis; EBV: Epstein-Barr virus; CMV: cytomegalovirus; HIV: human immunodeficiency virus; HBV: hepatitis B virus)

PHYSICAL EXAMINATION

A thorough physical examination, which needs to be repeated frequently and also by different medical personnel, so as not to miss any subtle or evolving signs, remains the most important clinical tool for diagnosis in a case of FUO. While examining the patient, particular attention should be paid to evaluation of skin, eye, throat, lymph nodes, sinuses, genitalia, back, etc. Certain clues are important to keep in mind as below:

Eyes

- Bulbar conjunctivitis → Kawasaki disease
- Palpebral conjunctivitis → Infectious mononucleosis
- Phlyctenular conjunctivitis → Tuberculosis
- Ischemic retinopathy → Polyarteritis nodosa (PAN)
- Absent tears and corneal reflex → Familial dysautonomia
- Choroid tubercle → Miliary tuberculosis
- Icterus → Hepatitis, leptospirosis, malaria, typhoid
- Proptosis → Neuroblastoma, thyrotoxicosis, Wegener's granulomatosis.

Upper Respiratory Tract and Oral Cavity

- Purulent nasal secretions → Sinusitis
- Pharyngeal hyperemia with exudate → Infectious mononucleosis
- Pharyngeal hyperemia without exudate → leptospirosis, infectious mononucleosis
- Hypodontia, conical teeth → Anhidrotic ectodermal dysplasia
- Smooth tongue with excessive salivation → Familial dysautonomia
- Gingival hypertrophy → Leukemia, histiocytosis.

Musculoskeletal

- Bony tenderness → Malignancy, osteomyelitis, infantile cortical hyperostosis
- Muscle tenderness → Dermatomyositis, PAN, leptospirosis
- Arthritis → Collagen vascular disease, brucellosis, TB, HIV
- Hyperactive tendon reflexes → Thyrotoxicosis
- Hypoactive tendon reflexes → Familial dysautonomia
- Trapezius tenderness → Subdiaphragmatic abscess.

LABORATORY TESTS

Initial Laboratory Tests

Complete Blood Count, Peripheral Smear Examination

- Thrombocytosis—Nonspecific acute phase reactant, Kawasaki disease (KD)

- Eosinophilia → Visceral larva migrans
- PMN $> 10,000/\text{mm}^3$ or band cells $> 500/\text{mm}^3$ → Severe bacterial infection
- PMN $< 5,000/\text{mm}^3$ → Against indolent bacterial infection except typhoid
- Atypical lymphocytes → Viral infection (infectious mononucleosis, especially)
- Immature lymphocytes → Leukemia
- ESR, CRP
 - ESR > 100 → TB, KD, autoimmune disease or malignancy
 - ESR can be low even in presence of infection if there is consumption of fibrinogen (DIC, HLH)
 - Very high ESR → In presence of hypergamma-globulinemia
- Urine analysis: Sterile pyuria → KD, TB
- Chest X-ray (CXR)
- Mantoux test
- Serology: Widal, tests for HIV
- Urine culture
- Blood culture
- Hepatic enzymes, blood urea, S. creatinine, S. electrolytes.

Additional Tests

Additional laboratory tests should be planned as per clinical possibilities and clues from the initial laboratory parameters.

- Serology → Brucellosis, rickettsial infections
- Bone marrow examination and trephine biopsy along with culture → Malignancy, hemophagocytic syndrome, histiocytosis, typhoid
- ANA and its profile
- X-rays for mastoiditis, sinusitis, dental abscess, osteomyelitis
- USG, CT, MRI
 - Abdominal imaging by USG, CT scan, and MRI:
 - IBD
 - Caseating granulomas of Koch's
 - Noncaseating granulomas of sarcoidosis, fungal infections
 - Microabscesses in liver and spleen in brucellosis
 - CT scan/MRI of chest
 - Can detect various shadows missed by CXR like small cavities in lung and CF, bronchiectasis, congenital cystic diseases, miliary TB
 - CXR showing lobar pneumonia → sequestered lung, lung cyst, caseating LN
- CT/MRI.

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5.6

Malignant Hyperthermia

Baldev S Prajapati, Rajal B Prajapati

INTRODUCTION

In 1960, malignant hyperthermia (MH) was first described in an Australian family by Denborough and Lovell. MH manifests clinically as a hypermetabolic crisis when an MH-susceptible individual is exposed to a volatile anesthetic agent like halothane or succinylcholine. The incidence of episodes of MH in the general population is estimated as 1:100,000 administered anesthetics.

MH is a group of inherited muscle problems characterized by muscle breakdown following certain stimuli such as anesthetic agent, extremes of exercise particularly in hot conditions and fever on the use of stimulant drugs. The problems associated with this condition result from over excitable muscles that contract uncontrollably. The features include very high grade fever (105–108°F), abnormal heart rhythms, tachypnea, hypoxemia, acidosis, and acute renal failure. It is also called as *malignant hyperpyrexia*.

PATHOPHYSIOLOGY

Patients susceptible to malignant hyperthermia have genetic skeletal muscle receptor abnormalities, allowing the excessive calcium accumulation in the presence of certain anesthetic triggering agents. The calcium overload within the skeletal muscle cell leads to cellular hypermetabolism, sustained muscular contraction and its breakdown (rhabdomyolysis), anaerobic metabolism, acidosis, and their sequelae.

CLINICAL FEATURES

The clinical manifestations of MH vary from patient to patient and most patients do not develop all signs of MH.

- Hyperthermia
- Masseter muscle rigidity
- Generalized muscle rigidity
- Hypercarbia
- Arrhythmia
- Myoglobinuria.

LABORATORY FINDINGS

- Metabolic and respiratory acidosis
- Hyperkalemia
- Elevated CPK
- Myoglobinuria
- Supportive lab evidence for disseminated intravascular coagulation.

PREVENTION

- Detection of susceptible patients
 - Medical and family history
 - Patient's response to physical exertion
 - Increased incidence of MH with:
 - Duchenne muscular dystrophy
 - Burkitt's lymphoma
 - Osteogenesis imperfecta
 - Myotonia congenita
 - Myelomeningocele
 - Serum CPK level estimation
 - EMG changes are seen in 5% of susceptible patients
 - Diagnostic DNA testing
- Prophylactic dantrolene
- Alternate anesthesia
- Selection of safer drugs.

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Section 9

Gastroenterology

Section Editor: Malathi Sathiyasekaran

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Fig. 9.18.3: Hepatosplenomegaly in chronic liver disease.



Fig. 9.18.4: Hepatosplenomegaly in a child with ascites and jaundice.

Table 9.18.6: Differential diagnosis of hepatosplenomegaly (HSM) based on associated findings.

HSM with associated findings	Diagnosis
HSM with prolonged/recurrent fever	<i>Infections:</i> TB, HIV, Brucellosis, melioidosis, malaria, Epstein-Barr virus (EBV), bacterial endocarditis, histiocytosis, chronic hepatitis, connective tissue disorders, malignancy
HSM with recurrent GI bleed	<i>Portal hypertension:</i> Extrahepatic portal vein obstruction, noncirrhotic portal fibrosis, cirrhosis
HSM with jaundice	Chronic hepatitis, acute on chronic liver disease, hemolytic jaundice
HSM with anemia	Leukemias, hemolytic anemia, malaria, hemophagocytic lymphohistiocytosis (HLH), portal hypertension, storage disorder, IU infections
HSM with failure to thrive	TB, HIV, any prolonged systemic illness, inborn errors of metabolism (IEM), cystic fibrosis,
HSM with generalized lymphadenopathy	TB, HIV, leukemia, lymphoma, HLH, EBV
HSM with petechiae and purpura	Dengue fever, leptospirosis, malaria, leukemia, septicemia
HSM with neurological manifestations	Wilson's disease, glycogen storage disease (GSD), Niemann–Pick disease, Gaucher disease
HSM with ascites and pedal edema	Chronic liver disease, abdominal tuberculosis, lymphoma, metastasis
HSM with renal involvement	Wilson's disease, tyrosinemia, GSD, chronic hepatitis B, congenital hepatic fibrosis with polycystic kidney, hereditary fructose intolerance and leukemia

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9.19 Masses in Left Upper Quadrant of Abdomen

R Bhanu Vikraman Pillai

INTRODUCTION

Abdominal pathology can be identified often with the location. The left upper quadrant (LUQ) is formed by the median plane extending to the left of the patient and with the umbilical plane to the left rib cage (**Fig. 9.19.1**).

Organs found in the LUQ are the stomach, the spleen, the left portion of the liver, and the main body of the pancreas. The left kidney and the adrenal gland are also found in this quadrant. The splenic flexure of the colon and portion of the colon also is found in the LUQ.

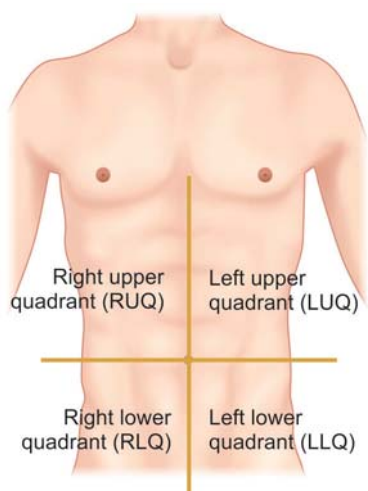


Fig. 9.19.1: Quadrants of abdomen.

The masses felt in this area originate from any of these organs as well as from the skin and subcutaneous tissue of the abdominal wall.

The causes are many and it would be appropriate to think the different structures of origin to consider the different ones (**Box 9.19.1**).

EVALUATION

Evaluating the different causes, the location is one of the most important clues as in **Box 9.19.1**.

HISTORY

Complete history including history of trauma (hematoma/pancreatic pseudocyst), fever (different causes of splenomegaly), jaundice (portal hypertension or liver disease), and pain is important. Is the mass persistent or intermittent (such as hernia), slow progression over months (e.g. storage disorders) or more rapid (e.g. infiltrative disease), and weight loss (e.g. lymphoma) needs to be asked.

PHYSICAL EXAMINATION

Detailed physical examination of the child including the general examination from head-to-toe and the local abdominal examination is important to come to a diagnosis.

Blood pressure should be taken in all cases (could be elevated in renal and adrenal pathology).

Stunted growth (storage disorders, chronic liver disease), pallor (infiltrative diseases/hemolytic anemia/portal hypertension), jaundice (chronic liver disease, hemolytic anemia), palmar erythema and spider nevi [chronic liver disease (CLD)], bruising (CLD, leukemia) as well as lymphadenopathy (leukemia/lymphoma) can certainly give a clue to the diagnosis. Hypertension may be seen in renal diseases as well as pheochromocytoma.

Abdominal examination (local) needs to be complete. Certain aspects of the examination need to be reiterated.

Abdominal examination starts with *inspection* of the abdomen with patient lying flat in the supine position. Inspect for scars, striae, dilated veins, visible peristalsis, bulges, visible masses as well as hernia. If there is a mass or bulge, examine its contour, whether it is bulging or uneven. Pay attention to the surface of the mass whether there is erythema such as an abscess.

Even though it is taught to go to palpation after the inspection, it may be more appropriate to do the *auscultation* before the palpation, since the palpation could alter the bowel sounds. Listen for bruit, bowel sounds, etc.

Palpation is probably the most important part of the abdominal examination. Examine the area of concern or pain at the end. During the palpation, watch the facial expression of the child and try to distract him/her so that one can clearly see for any tenderness. Palpate in a methodical fashion for any rigidity/tenderness throughout the abdomen, following which palpate for the liver and spleen. Liver is palpated with the border of the hand starting at the right lower quadrant, slowly up to the costal margin. Following this, the spleen is palpated starting from the right lower quadrant toward the LUQ. During these palpations, ask the patient to take breaths in and out so we can also assess the movement with respiration. The mass of concern is examined carefully for tenderness, movement with respiration, any structure of origin such as ribs, whether arising from the abdominal wall, etc. The size of the mass, ballotability (as in renal origin), pulsation as well as bruit should be examined.

Percussion of the abdomen, generally as well over the mass should be done. It will identify shifting dullness as in ascites with chronic liver disease and portal hypertension.

INVESTIGATION

Certain investigations will be necessary to come to a diagnosis.

- *Complete blood count (CBC)*: Look for anemia (CLD, hemolysis) and thrombocytopenia (hypersplenism).
- *Liver function tests (LFT)*: Elevated bilirubin (may be seen in CLD, hepatitis), elevated transaminases (CLD, hepatitis), and low albumin (CLD).
- *Creatinine*: May be abnormal in kidney disease.
- Lipase or amylase could be elevated in pancreatitis, pancreatic pseudocyst.
- Several radiological tests can be helpful.
- Plain X-ray abdomen could show calcification as well as fecal masses.
- Ultrasonography (USG) abdomen is probably very useful, given easy availability, relatively less expensive, and no ionizing radiation. This can show the description of the mass, origin, character, extent, and many times the diagnostic clue. Intussusception can be diagnosed by the USG.

Box 9.19.1: Various conditions—left upper quadrant organs.

- **Abdominal wall:**
 - Hernia
 - Hematoma
 - Lipoma
 - Fibroma
 - Rhabdomyosarcoma
- **Spleen:**
Splenomegaly is in fact one of the most common causes of a mass in the left upper quadrant (LUQ). Different causes of splenomegaly needs to be considered in the evaluation.
 - Infiltrative disorders (e.g. leukemia/lymphoma)
 - Hemolytic disorders (e.g. hereditary spherocytosis)
 - Portal hypertension (either due to chronic liver disease or extrahepatic portal vein obstruction)
 - Storage diseases (e.g. Gaucher's /Neimann–Pick disease)
 - Infections
- **Stomach:**
 - Duplication
 - Gastroparesis
 - Bezoar as well as foreign bodies
- **Intestine:**
 - Intussusception
 - Volvulus
 - Lymphoma
 - Fecal masses
 - Abscess from Crohn's disease
 - Rarely duplication
- **Pancreas:**
 - Pancreatic pseudocyst (either following trauma or after acute pancreatitis)
- **Kidney:**
Masses from the kidney is one of most frequently encountered ones especially in newborns and young children.
 - Hydronephrosis (most common)
 - Polycystic kidney disease
 - Multicystic kidney disease
 - Wilm's tumor
 - Renal vein thrombosis
- **Adrenal:**
Mass arising from the adrenal is frequently encountered, even though less common than splenic or renal origin.
 - Neuroblastoma
 - Pheochromocytoma
 - Adrenal hemorrhage
- **Peritoneum:**
 - Mesenteric cyst
 - Teratoma
- **Rib:**
Also keep in mind a mass such as osteochondroma arising from the rib cage.
- **Liver:**
In case of situs inversus.

- CT scan of the abdomen or MRI of abdomen may be necessary for further evaluation and can be extremely useful in making the diagnosis.
- Intravenous pyelogram (IVP) may be needed in selected cases of renal pathology.
- Endoscopy also may be necessary in selected cases (e.g. portal hypertension).
- Surgical explorative laparoscopy may even be necessary in certain cases.

Differential Diagnosis in PEDIATRICS

This book has been brought out under IAP President's action Plan. An exact diagnosis is essential for patient care, as further management entirely depends on it. For achieving this, all the possible differential diagnoses have to be entertained and after thorough clinical examination with appropriate investigations, the "final diagnosis" should be arrived. The need to have differential diagnoses for many common clinical situations compiled in a single book should be fulfilled by this initiative.

The differential diagnosis for the symptoms and signs are covered mainly and some important clinical conditions are also considered. Each Chapter focuses on definition, differential diagnosis for the conditions themselves and their causes, as applicable; history, clinical features and investigations leading to particular diagnosis. There are 23 Sections with 195 Chapters totally. The chapters apart from the regular text are pertinently supported by explanatory tables and illustrations in the form of figures including algorithms. Important references for *Further Reading* also have been incorporated. The chapters are contributed by the experts in their respective fields from all over India. The information has been presented lucidly and in a reader-friendly manner. The book will be a boon for the pediatricians to guide them in day-to-day practice as well as for the pediatric residents.

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