THE OEDEMATOUS CHILD

Gong Wei Kin, Yap Hui Kim

Oedema is a common presenting problem in paediatrics. It is defined as accumulation of excess interstitial fluid and could be localised or generalised. Oedema results from either excess salt and water retention or from increased transfer of fluid across the capillary membranes. As such, it could be a presentation of mild conditions such as insect bite reaction to more serious conditions such as glomerulonephritis, hepatic and cardiac disease. Understanding the pathophysiology of oedema is important in the clinical approach and management of this condition in children.

Contents
Causes of oedema
Clinical approach
Investigations
Management
CAUSES OF OEDEMA

Oedema results when there is:

1) Increased hydrostatic pressure
   - acute nephritic syndrome, acute tubular necrosis, cardiac failure
2) Decreased plasma oncotic pressure (hypoproteinaemic states)
   - nephrotic syndrome, chronic liver failure, protein losing enteropathy, protein caloric malnutrition
3) Increased capillary leakage
   - insect bite, trauma, allergy, sepsis, angio-oedema
4) Impaired lymphatic flow
   - lymphatic obstruction (tumour), congenital lymphoedema
5) Impaired venous flow
   - hepatic venous outflow obstruction, superior/inferior vena cava obstruction

Mechanism of oedema formation in renal disease

Oedema formation between nephritic and nephrotic syndrome is markedly different. Understanding oedema formation in these two conditions is important to differentiate the two, as their management is entirely different.

As such, a child with nephritic oedema will have symptoms and signs of intravascular fluid overload such as orthopnoea, cardiomegaly, raised jugular venous pressure, pulmonary congestion and hepatomegaly.
CLINICAL APPROACH TO AN OEDEMATOUS CHILD

1. Confirm oedema.

2. Assess distribution of oedema: Localised versus generalised. In generalised oedema, look for dependent areas such as pretibial, sacral, scrotal, vulval oedema other than peri-orbital oedema and ascites.

3. Detailed history and physical examination to assess severity, associated complications and underlying cause of the oedema (algorithm 1).

A) Localised oedema

i. History of trauma, insect bite or infection
ii. Peripheral lymphoedema in newborn – to exclude Turner’s syndrome
iii. Acute oedema of the face and neck – to exclude superior vena cava obstruction syndrome

B) Generalised oedema

i. Renal disease (most common cause in children)
   - gross haematuria, oliguria, hypertension, cardiomegaly, pulmonary oedema to suggest acute glomerulonephritis. Frothy urine suggests nephrotic syndrome. Absence of circulatory congestion differentiates nephrotic syndrome from nephritic syndrome.
   - signs and symptoms of chronic renal insufficiency such as anaemia, growth retardation and uraemic symptoms such as nausea and vomiting
   - exclude secondary causes such as post-infectious glomerulonephritis, systemic lupus erythematosus, Henoch Schonlein purpura nephritis.

ii. Liver disease
   - stigmata of chronic liver disease such as jaundice, palmar erythema, clubbing, pruritic rash
   - hepatosplenomegaly with gross ascites in the absence of jaundice to exclude portal vein thrombosis
   - previous operation scar such as Kasai porto-enterostomy

iii. Allergic reactions
   - oedema usually mild, commonly periorbital
   - history of allergen exposure such as medications, animal dander, food preservatives and colouring
   - associated rashes such as urticaria
   - assess for Stevens-Johnson reaction
   - if recurrent oedema, consider C1 esterase deficiency
iv. Cardiac disease
   - symptoms of congestive cardiac failure such as decreased effort tolerance, orthopnoea, paroxysmal nocturnal dyspnoea and signs such as cardiomegaly, gallop rhythm, lung crepitations and turgid liver
   - assess for underlying cause such as structural heart disease, cardiomyopathy and myocarditis

*Note: oedema in cardiac disease often denotes a late sign in small children*

v. Protein losing enteropathy
   - history of chronic diarrhoea, steatorrhoea and recurrent abdominal pain
   - detailed dietary history for possible milk allergy and gluten hypersensitivity
   - consider coeliac disease and inflammatory bowel disease in northern Indians and Caucasians
   - assess for complications of anaemia, malnutrition and vitamin deficiency states

4. Investigations

Basic investigations could be conducted at the general practice set-up to delineate the major causes of oedema and assess its severity. They include:

a) **Urine dipstick and microscopy**
   - proteinuria, haematuria and casts are indicative of renal disease

b) **Renal function test**
   - raised serum urea and creatinine are indicative of renal disease

c) **Full blood count**
   - normochromic normocytic anaemia suggest chronic disease
   - hypochromic microcytic anaemia suggest iron deficiency from occult gastrointestinal bleeding i.e. cows milk allergy
   - megaloblastic anaemia suggest vitamin B12 and folate deficiency from small bowel disease

d) **Liver function test**
   - hypoalbuminaemia in the absence of circulatory overload suggests hypoproteinaemic states
   - hyperbilirubinaemia and transaminitis suggest liver disease
e) Chest X-ray and electrocardiogram
- cardiomegaly with prominent perihilar vascular markings/upper lobe diversion and left ventricular hypertrophy confirms intravascular fluid overload

Further investigations are indicated upon specialist referral depending on the most likely cause.

Algorithm 1: Clinical approach and assessment of an oedematous child

MANAGEMENT

Subsequent management of oedema is dependent on the primary cause and its severity.

What you can do

A) Localised oedema

1. Insect bite reaction  Anti-histamine, anti-inflammatory (topical steroid)
2. Local infection  Incision and drainage, wound dressing, antibiotics
B) *Generalised oedema*

General measures

a) Dietary management
   - sodium restriction to 2 grams/m²/day
   - fluid restriction to 2/3 maintenance depending on severity of oedema

<table>
<thead>
<tr>
<th>AGE</th>
<th>Daily requirements (ml/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>60</td>
</tr>
<tr>
<td>Day 2</td>
<td>90</td>
</tr>
<tr>
<td>Day 3</td>
<td>120</td>
</tr>
<tr>
<td>Day 4</td>
<td>150</td>
</tr>
<tr>
<td>1 year</td>
<td>100</td>
</tr>
<tr>
<td>5 years</td>
<td>75</td>
</tr>
<tr>
<td>10 years</td>
<td>50</td>
</tr>
</tbody>
</table>

b) Diuretic therapy

   i. Loop diuretics
      - Frusemide (Lasix)
        1-5 mg/kg/dose 6-8 hourly (IV/IM/PO)
      - Bumetanide (Burinex)
        0.02-0.1 mg/kg/dose 12-24 hourly (PO)
        0.1-0.2 mg/kg/dose 8-12 hourly (IV/IM)

Note: higher doses are required in acute glomerulonephritis or chronic renal impairment

Side effects- hyponatremia, hypokalemia, metabolic alkalosis

   ii. Hydrochlorothiazide
       - 1 mg/kg/dose 12-24 hourly (PO)

   iii. Spironolactone
       - used in combination with loop diuretics/ thiazides for potassium sparing effects
       - 0-10 kg: 6.25 mg/dose 12 hourly (PO)
       - 11-20 kg: 12.5 mg/dose 12 hourly (PO)
       - 21-40 kg: 25 mg/dose 12 hourly (PO)
       - > 40 kg: 25 mg/dose 8 hourly (PO)
### SPECIFIC MANAGEMENT UPON SPECIALIST REFERREMENT

<table>
<thead>
<tr>
<th>Cause of oedema</th>
<th>Further investigations</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nephrotic syndrome</td>
<td>Fasting lipids, screen for secondary causes e.g. SLE, Hepatitis B</td>
<td>Prednisolone (ISKDC) Nephrotic diet IV albumin with diuretics for severe oedema Steroid sparing agents in steroid dependent/resistant cases</td>
</tr>
<tr>
<td>Acute glomerulonephritis</td>
<td>Serum complements, screen for secondary causes e.g. Post-streptococcus infection, SLE, Ig A nephropathy etc</td>
<td>Diuretics (first line) Anti-hypertensives Dialysis in severe cases Treat underlying cause</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>Screen for underlying cause, assess complications e.g. hyperammonemia, coagulopathy</td>
<td>IV albumin with diuretics for severe oedema Liver failure management</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Cardiac enzymes, 2D-echocardiography</td>
<td>Diuretics Salt and fluid restriction Digoxin Treat arrhythmias</td>
</tr>
<tr>
<td>Cow’s milk allergy</td>
<td>FAST test for cow’s milk Ig E, Patch test, jejunal biopsy</td>
<td>Avoid cow’s milk Consider soy based formula (20% cross sensitivity) Treat iron deficiency</td>
</tr>
<tr>
<td>Gluten hypersensitivity</td>
<td>Anti-gliadin IgG/IgA, anti-endomysial IgA, jejunal biopsy</td>
<td>Gluten free diet Treat iron deficiency</td>
</tr>
</tbody>
</table>

### CONCLUSION

Oedema can be due to
- increased hydrostatic pressure
- decreased plasma oncotic pressure
- increased capillary leakage
- impaired lymphatic flow
- impaired venous flow

This could be a manifestation of a mild or a serious medical conditions such as renal, hepatic or cardiac diseases. Early recognition of oedema and prompt diagnosis of the underlying cause is important. Initial assessment and stabilisation of patient should be instituted prior to specialist referral.