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# An unusual case of suspected head injury

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Case Report

# An unusual case of suspected head injury

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## Abstract

A healthy two-year-old female child presented to the Emergency Department (ED) with lethargy and recurrent episodes of non-bilious and non-projectile emesis after sustaining a fall. On examination, she was found to be drowsy, but could be easily aroused with Glasgow Coma Scale (GCS) of 15. Her abdominal examination revealed a left quadrant mass. Initial laboratory test showed low haemoglobin. In the setting of trauma, the low haemoglobin and abdominal mass, there was a concern for splenic injury and haemorrhage. A CT scan of the abdomen and pelvis revealed a large mass arising from the left kidney suggestive of Wilms tumour. Wilms tumour is the most common primary renal malignancy in children and constitutes about 5% of all paediatric cancers. It is typically described as an asymptomatic abdominal mass noted by a caregiver and it may have vague and atypical presentations. Paediatric ER physicians are often the first point of medical contact for children with malignancy and play an important role in early diagnosis of paediatric cancers.

**Case**

A previously healthy, immunized two-year-old female child presented to the ED at a tertiary care children's hospital with the chief complaint of vomiting after sustaining a possible head injury. She was in the recreation center with her parents. While she was playing, she fell and hit her face on the hardwood floor of the gymnasium. She did not fall on any toys or objects. There was no loss of consciousness. Immediately after the fall, she picked herself up and was crying, pointing to her abdomen indicating pain. She then became tired and fell asleep on her dad's shoulder for about 15 minutes. She had an episode of non-bilious, non-projectile emesis after she woke up. When they got her home, she took a nap for 4 hours that is more than what is typical for her. She then had a few more episodes of non-bilious, non-projectile emesis. Parents decided to bring her to the ED for her repeated episodes of emesis and unusual lethargy.

She had no recent history of fever, cough, congestion, diarrhea or constipation. Her appetite had been good. Her mom felt that her abdomen was more distended than usual but it seemed to have resolved on its own.

On examination in the ED, her vitals were significant for a tachycardia of 150 (crying at the time of measurement), normal temperature of 99.2F, respiratory rate of 28 and saturation of 98% in room air. Physical examination revealed a sleepy but easily arousable child with appropriate interaction with the parents. Head was normocephalic and atraumatic. Pupils were reactive. Ears showed no haemotympanum. Neck with a few shotty lymph nodes palpated in the left anterior cervical chain with no C spine tenderness. Respirations were clear bilaterally without any wheezing or crackles. Heart examination was normal with regular rhythm, normal S1 and S2 and no murmur. Her abdominal examination revealed a large mass, occupying

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the entire left upper quadrant of the abdomen, extending up to the umbilicus. It was measured to be three inches below the costal margin. It was firm in consistency and tender on palpation. The edge of the liver was palpable below the right costal margin. There were no rashes, petechiae or purpura noted on the skin. Neurological examination was normal with a GCS of 15. She was tired but would awaken appropriately and interact with her parents.

Initial lab tests were obtained. Complete blood count was significant for elevated white count of 19.7 with neutrophilia (86.3% with immature granulocytes 0.4%) and haemoglobin was low at 8.4 with a normal RBC count. Electrolytes were within normal limits. Renal function and liver enzymes were normal. Coagulation studies showed minimally elevated international normalized ratio (INR) at 1.18, while Partial thromboplastin time (PTT) was normal. (Table 1 – Table 6)

**Table 1: Complete blood count (CBC) with differential**

| Result | Value | Range |
|---|---|---|
| WBC | 19.7 (H) | 5.5 – 10.5 10e9/L |
| RBC count | 4.08 | 3.7 – 5.3 10e12/L |
| Haemoglobin | 8.4 (L) | 10.5 – 14.0 g/dL |
| Haematocrit | 28.3 (L) | 31.5 – 43.0 % |
| MCV | 69 (L) | 70 – 100 fL |
| MCH | 20.6 (L) | 26.5 – 33 pg |
| MCHC | 29.7 (L) | 31.5 – 36.5 g/dL |
| RDW | 18.8 (H) | 10.0 – 15.0 % |
| Platelet count | 423 | 150 – 450 10e9/L |
| Diff method | Automated | |
| % Neutrophils | 86.3 | |
| % Lymphocytes | 10.9 | |
| % Monocytes | 2.2 | |
| % Eosinophils | 0.0 | |
| % Basophils | 0.2 | |
| % Immature granulocytes | 0.4 | |
| Absolute Nuetrophil | 17.0 | 0.8 – 7.7 qpe9/L |
| Absolute Lymphocytes | 2.2 (L) | 2.3 – 13.3 10e9/L |
| Absolute Monocytes | 0.4 | 0.0 – 1.1 10e9/L |
| Absolute Eosinophils | 0.0 | 0.0 – 0.7 10e9/L |
| Absolute Basophils | 0.0 | 0.0 – 0.2 10e9/L |
| Absolute Immature Granulocytes | 0.1 | 0.0 – 0.8 10e9/L |

**Table 2: C- reactive protein**

| Result | Value | Range |
|---|---|---|
| CRP | 3.4 | 0.0 – 8.0 mg/L |

**Table 3: Complete metabolic panel**

| Result | Value | Range |
|---|---|---|
| Sodium | 137 | 133 – 143 mmol/L |
| Potassium | 3.9 | 3.4 – 5.3 mmol/L |
| Chloride | 103 | 96 – 100 mmol/L |
| Carbon Dioxide | 23 | 20 – 32 mmol/L |
| Anion Gap | 11 | 3 – 14 mmol/L |
| Glucose | 145 (H) | 70 – 99 mg/dL |
| Urea Nitrogen | 16 | 9 – 22 mg/dL |
| Creatinine | 0.23 | 0.15 – 0.53 mg/dL |
| GFR | Not calculated | |
| Calcium | 9.1 | 9.1 – 10.3 mg/dL |
| Bilirubin Total | 0.2 | 0.2 – 1.3 mg/dL |
| Albumin | 3.7 | 3.4 – 5 g/dL |
| Protein Total | 6.9 | 5.5 – 7.0 mg/dL |
| Alkaline Phosphatase | 161 | 110 – 320 U/L |
| ALT | 18 | 0 – 50 U/L |
| AST | 41 | 0 – 60 U/L |

**Table 4: Other Significant Lab Studies**

| Result | Value | Range |
|---|---|---|
| Lipase | 42 | 0 – 194 U/L |
| LDH | 984 (H) | 0 – 337 U/L |
| UA | 4.9 (H) | 1.4 – 4.1 mg/dL |
| DAT Broad Spectrum | Neg | |

**Table 4: Reticulocyte Count**

| Result | Value | Range |
|---|---|---|
| % Retic | 2.0 | 0.5 – 2.0 % |
| Absolute Retic | 71.9 | 25 – 95 10e9/L |
| Retic Method | Automated | |

The low haemoglobin along with the abdominal mass raised suspicion for a ruptured spleen and a CT scan of the abdomen was obtained to assess splenic injury. The CT scan revealed a large left renal mass,

**Table 5: Coagulation studies**

| Result | Value | Range |
|---|---|---|
| PTT | 29 | 22 – 37 sec |
| INR | 1.18 (H)` | 0.86 – 1.14 |

**Table 6: Blood Group: Type and screen**

| Result | Value | Range |
|---|---|---|
| ABO | A | |
| Rh (D) | Neg | |
| Antibody screen | Neg | |

with the left renal vein and inferior vena cava (IVC) draping the margin of the tumour. There was free fluid in the retro-peritoneum on the left concerning for haemorrhage secondary to capsular haemorrhage of the left renal mass. (Table 7, Figure 1)

**Table 7: Imaging Study Reports**

| | |
|---|---|
| **Chest X-Ray** | Low lung volumes, no acute pulmonary disease<br>Mild to moderate gastric distention with paucity of bowel gas in the left hemiabdomen. Consider dedicated abdominal ultrasound for more information. |
| **Ultrasound Abdomen** | Large mass within left kidney measuring up to 9.3 cm with left sided hydro nephrosis. Findings are most concerning for Wilms tumour. In the left renal vein and IVC are patent.<br><br>Small amount of free fluid adjacent to the left kidney and in the pelvis. |
| **CT Chest Abdomen Pelvis with contrast** | Large heterogenous left renal mass highly suspicious for Wilms tumour. Left renal vein and Superior Mesenteric Vein (SMV) drape along the margin of the tumour.<br><br>There is marked left sided hydronephrosis and hydroureter with hyperdense material in the distal third of the left ureter, which presumably represents haemorrhage.<br><br>There is no evidence of tumour thrombus within the left renal vein or IVC.<br><br>Extensive complex free fluid throughout the retroperitoneum on the left concerning for haemorrhage secondary to capsular rupture of the left renal mass.<br><br>Two non-calcified pulmonary nodules in the lower lobes. Contrast enhanced left para-aortic lymph nodes at the level of the left kidney. These findings are suspicious for metastatic disease. |

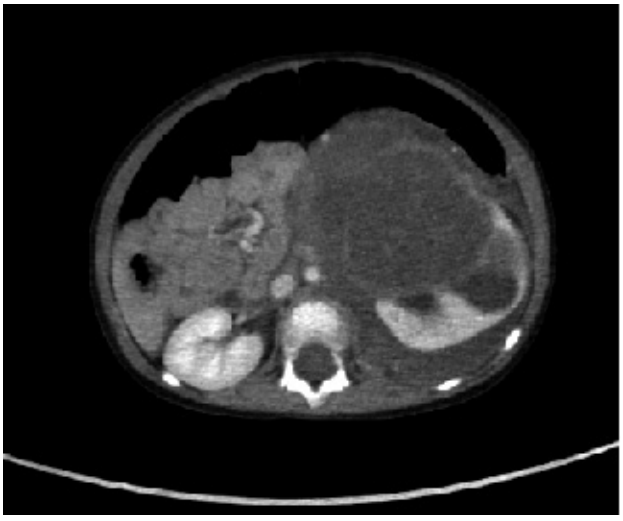

Figure 1a: CT Chest – Abdomen – Pelcis with contrast

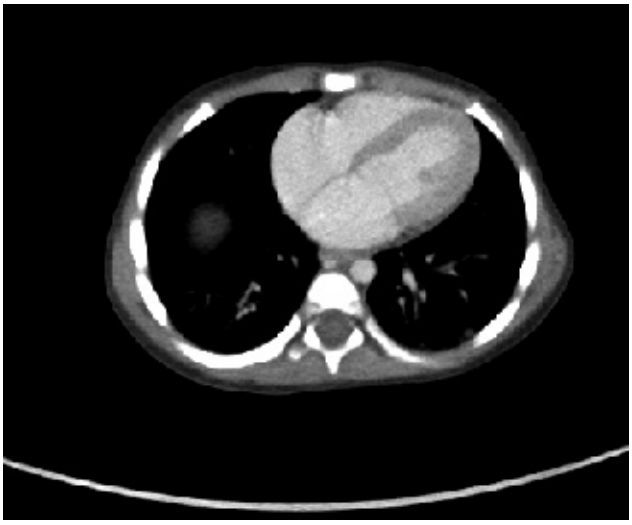

Figure 1b: Large heterogenous left renal mass suspicious for Wilms tumor. Extensive complex free retroperitoneal fluid concerning for haemorrhage secondary to rupture of the tumor capsule. Two calcified pulmonary nodules in the lower lobes and contrast enhancement of the left para-aortic lymph nodes is suspicious for metastatic disease.

Paediatric Hematology-Oncology was consulted and further laboratory work up and imaging was performed per counsel. The results of these investigations are included below. Histopathology confirmed the diagnosis to be a nephroblastoma.

## Discussion

Early diagnosis of paediatric cancers has a great impact on the overall stage dependent mortality and morbidity and thereby on the course and prognosis of the illness. Since the presentation of childhood cancers is not always straightforward, it makes the diagnosis challenging (1).

It goes without saying that all children deserve a thorough evaluation when presenting to the ED. This becomes especially important in a child with cancer, who often presents with vague complaints.

A retrospective study was performed in Singapore to assess the factors that determine the diagnostic delay in paediatric solid tumour. One of the factors studied was the first contact with the health care system and they concluded that when paediatric ER physicians were the first point of contact for evaluation of a child with cancer, the delay to diagnosis was shorter. This was an independent positive determinant with a significant P value.[1]

The incidence of Wilms tumour is 1 in 10000 children[2] constituting about 5% of all paediatric malignant tumours.[3] Wilms tumour is the most common primary renal malignancy in children. It is one of the success stories of paediatric oncology with long-term survival of about 90% for localized disease and 75% for metastatic disease.[4] The most important prognostic variables for a patient with Wilms is the histopathologic tumour classification and surgical stage of the tumour.[5] Meticulous attention must be paid for accurate staging of the tumour to make its management successful.[4] As described above, paediatric ER physicians in fact play an important role in the early identification of childhood solid tumours.[1] Our patient was an unusual case where the diagnosis was incidentally discovered in our evaluation of this child, who had presented with a suspected head injury and was thought to have a splenic rupture.

Most cases of Wilms tumour are seen in children less than five years of age with the peak incidence in children 2–3 years of age. The typical presentation of Wilms tumour is an abdominal mass palpated in an otherwise well child.[5] A five-year retrospective study performed in an inner-city hospital in Michigan identified the incidence of paediatric cancers diagnosis in the ED to be one in every 4,623 annual ED visits of which 22% were abdominal tumours, Wilms tumour, and neuroblastoma being the most common. They described these children to characteristically present with abdominal pain or distention.[6]

It is important to note that acute abdominal pain can be the presenting complaint in 30% of children with Wilms tumour. In some instances, this pain can be severe enough to have a child to be incorrectly classified as an acute surgical abdomen. A study describing acute abdominal pain in children with Wilms discusses the importance of a thorough abdominal examination prior to surgical exploration in any children with such a presentation.[7] Acute pain however is not very specific for tumour rupture and only some of these patients had true tumour rupture noted on surgery[8].

Other atypical presentations can be due to paraneoplastic syndromes, which though uncommon in paediatric cancers, are known to be associated with Wilms tumour.[9]

Isolated polycythemia is one of them and can be a rare presentation of Wilms tumour and occurs due to hypersecretion of erythropoietin by the tumour.[10] Unexplained elevated haemoglobin levels in some patients can be the initial indication of a Wilms tumour and should alert physicians for further work up.[11] Our patient had low haemoglobin at presentation that was thought to be secondary to the fall causing a rupture of the tumour capsule. Hypertension due to release of renin, hypercalcemia, and acquired von Willebrand disease are other atypical presentations due to paraneoplastic syndromes.[4] Atypical presentations can also be due to infiltration and compression of surrounding organs or vascular infiltration for which reason, a Doppler US evaluation of the veins is extremely important.[2]

Routine laboratory tests and imaging for Wilms tumour can be initiated in the ED. Anytime an abdominal mass is suspected, a color Doppler ultrasound is the first modality of investigation performed to characterize the mass and confirm its origin.[8] It also helps to evaluate the contralateral kidney.[4] Since tumour rupture significantly changes management strategies, the outline of the tumour is carefully checked during the ultrasound. A CT scan is then performed to determine tumour extent, including extension into the IVC.[5,8] Pulmonary metastasis is the most common and a chest CT scan

is performed to evaluate the lungs.[5]

The management of Wilms tumour requires a multi-disciplinary team including paediatric oncologist; specialist surgeons; radiologists; pathologists and radiation oncologists[4] and transfer of the patient to an equipped center should be facilitated soon after the diagnosis is made on imaging.

## Conclusion

Wilms tumour is the most common primary renal malignancy in children and constitutes about 5% of all paediatric cancers. It is typically described as an asymptomatic abdominal mass noted by a caregiver, however may have vague and atypical presentations. Paediatric ER physicians are often the first point of medical contact for children with malignancy and play an important role in early diagnosis of paediatric cancers.

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