The 2nd baby had grunting, and respiratory distress soon after birth. She was admitted in NICU and started with iv fluid, iv antibiotics and O₂ through nasal prong. Investigations revealed covid 19 RT-PCR negative but covid 19 IgG-7.89, IL6-184.0 pg/ml, D Dimer - 919, LDH-593, Trop-I- 155.1 ng/L, NTproBNP-567.1, procalcitonin- 0.866 ng/ml with B/L haziness on CXRay. The mother also had covid IgG -7.89. The baby was treated with IVIg, SC LMWH, and supportives. The baby responded to treatment well and discharged.

Results Both the babies responded well to IVIg, iv antibiotics and supportives. We have use iv dexona in 1st case.

On follow up all the laboratory parameters of the 1st baby was normal and she was gaining weight. The 2nd baby was on LMWH as her d Dimer, APTT and covid IgG were high. Clinically she is asymptomatic, feeding well and gaining weight.

Conclusions Though cytokine storm is dangerous consequence of covid 19 in Preterm newborns timely diagnosis and appropriate treatment can reduce the morbidity and mortality of the disease.

372 USAGE OF HIGH FLOW NASAL CANNULA IN PAEDIATRIC PATIENTS OUTSIDE ICU SETTING

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Background High-flow nasal cannula (HFNC) is a non-invasive positive pressure ventilation which delivers adjustable mixture of heated and humidified air and oxygen at rates that exceed spontaneous inspiratory flow. It's easy to initiate, relatively safe, and usually well tolerated by children (1). A lot of studies have suggested that HFNC may reduce the work of breathing (1). Early initiation of HFNC has been associated with reduced rate of endotracheal intubation.

Objectives This study aims to provide an overview of HFNC usage in paediatrics general ward setting, outside ICU setting.

Methods A retrospective study was carried out to evaluate the usage of HFNC in paediatric patients in HSIP general ward settings from April 2019 to March 2020.

Results A total of 177 children's record (boys: 112, girls: 65, age: 1 month to 12 years old) were analysed. A total of 112 patients were referral from district hospital, while 55 patients were referral from our emergency department.

From the analysis, we noted the age group of 12 to 24 months had the highest number of admissions requiring HFNC. Most of the patients were put on HFNC immediately upon admission to ward. The duration of its usage ranges from 2-4 days [interquartile range (IQR)], with median of 3 days. Length of hospital stay were 6 days (IQR: 4 to 8 days). Multiple linear regression analysis showed that duration of HFNC usage and delaying its initiation >6 hours were associated with significantly longer hospital stay (p-value <0.001).

Among the indications for the usage of HFNC, pneumonia is the main cause, followed by acute bronchiolitis, heart failure, sepsis and laryngomalacia. Almost one third (38%) of the patients that required HFNC had underlying disease(s), mainly respiratory disease. SpO₂ on arrival were mostly 96% (IQR: 92 to 99%). Respiratory rates were analysed according to age group as well.

The median white cell count (WCC) is $12.96 \times 10^3/\mu\text{L}$ (IQR: 9.67 to 17.27), while C-reactive protein (CRP) is 22 mg/L (IQR: 10.91 to 52.62). Linear regression was used to analyse the correlation between these two parameters, which showed that WCC and CRP are two independent variables.

Nasopharyngeal aspiration (NPA) for viruses sent yielded 24% positive culture. Among the viruses detected, RSV accounted for 66% of the positive culture.

As for outcome, 98% (N=174) of them recovered well while 3% (N=3) required escalation of therapy to BIPAP and intubation. Unfortunately, 3 patients succumbed to death in which one had severe malnourished and the other two patients with had congenital cyanotic heart with prolonged hospital stay. No major adverse effect was reported but 1 patient had minor burn on cheeks.

Conclusions HFNC is an excellent choice of NIV in providing respiratory support in district hospital with no or very limited intensive care unit. Our study showed that it is relatively safe to use with regular vital signs monitoring, with occasionally some patients requiring continuous SpO₂ monitoring. Detailed studies on its indication, safety protocol and cost effectiveness are needed to improve the outcome of patients.

375 HEPATOPATHY-THROMBOCYTOPENIA SYNDROME (HTS) AFTER ACTINOMYCIN-D THERAPY IN WILMS TUMOUR: A RARE COMPLICATION WITH FAVOURABLE OUTCOME

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Background Hepatopathy-thrombocytopenia syndrome (HTS) is characterized by fever, hepatopathy (hepatomegaly with abnormal liver function tests), ascites, weight gain, jaundice, and thrombocytopenia (platelet count less than $25 \times 10^3/\mu\text{L}$). The incidence is about 1.4% in a child with Wilms tumour receiving actinomycin D. Its mortality is 5-20%.

The mechanism is not well understood. Histopathological markers of HTS are obliteration of small hepatic venules and damage to endothelial cells

One of the prominent features of HTS is severe thrombocytopenia. It could be the first sign of detecting 'at risk child' of developing full-blown syndrome. Thrombocytopenia related to actinomycin D has been well documented.

Risk factors are younger age, administration of actinomycin D, radiotherapy, GSTM1-null genotype and MDR1 gene mutations.

Objectives To report a case of HTS post actinomycin D in a patient with underlying Wilms tumour.

Methods This is a case of a 4-year-old girl, newly diagnosed left Wilms tumour COG stage II, underwent left nephrectomy and started on weekly chemotherapy. Three days after she received her 3rd cycle of vincristine and actinomycin D, she crashed to emergency department in encephalopathic state with symptomatic hypoglycemia. Physical examination revealed hepatosplenomegaly and gross ascites but there was no jaundice. Her blood investigations showed low platelet count of $7 \times 10^3/\mu\text{L}$, severe transaminases AST 5381U/L, ALT 2035U/L, and coagulopathy but normal renal profile. Her serum ammonia was 125 $\mu\text{mol/L}$ with compensated metabolic acidosis. CT

Abstract 375 Table 1 Table of investigations

	Prior chemotherapy	D2 post Chemotherapy	D3 post chemotherapy	D6 post chemotherapy	D11 post chemotherapy	D19 post Chemo therapy
Platelet	33	7	42	142		152
AST	40	5381	7978	938	100	79
ALT	16.4	2035	3524	1829	610	127
Bilirubin	4.9	29.6	49.9	34.5	37.8	19.9
Ammonia		125	84			
PT		33.7	34.5	18.8		

brain revealed no intracranial bleed and ultrasound abdomen showed gross ascites. Multidisciplinary teams discussion led to diagnosis of HTS.

She was closely monitored in intensive care unit with vigorous supportive care included high flow nasal cannula, judicious fluid and urine output monitoring.

She showed tremendous improvement, regained full GCS within 24 hours of admission. Her platelet count had marked increment by second day of admission and normalized within one week. All the blood parameters showed improvement within a week.

She received subsequent chemotherapy with reduction of actinomycin D dose with no recurrence of HTS.

Results The onset of HTS occurred within 10 weeks of diagnosis and usually occurred 1 to 7 days post actinomycin D administration. HTS can last up to 12 days and resolve with supportive treatment. After recovery, most children can tolerate actinomycin D at a reduction dosage without recurrence of the syndrome.

Therapy of HTS is mainly vigorous supportive care. However, defibrotide, which is an antithrombotic has emerged as an effective therapy in view of histopathologic marker of HTS is obliteration of hepatic vein.

Conclusions Early detection and recognition of syndrome will prompt immediate action resulting extremely favourable outcome. Further studies regarding risk factors and promising drugs should be carried out to reduce mortality of HTS.

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POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN ACUTE POST-STREPTOCOCCAL GLOMERULONEPHRITIS

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Background Acute post streptococcal glomerulonephritis (PSGN) is well known sequel of group A β -hemolytic streptococcus infections and may be complicated with hypertensive encephalopathy followed by Posterior reversible encephalopathy syndrome (PRES). PRES is also known as acute hypertension encephalopathy, usually present with seizures, hypertension, headache, encephalopathy and/or visual disturbance. The pathophysiology behind PRES is poorly understood. It is usually associated with hypertensive encephalopathy, kidney disease, autoimmune disease, immunosuppressive drug and organ transplantation. Contrast-enhanced computed tomography imaging allows for fast assessment which shows features

of edema of the posterior cerebral hemisphere, especially at the parieto-occipital region. PRES can be a presenting sign of PSGN and vice versa.

Objectives To study relationship between PSGN and PRES

Methods Two case reports PSGN with PRES, based on contrast-enhanced computed tomography (CECT) brain.

Results We reported two cases of PSGN with PRES.

First case is a 11 years old boy, who presented with status epilepticus, following one-day history of headache and vomiting. He was afebrile, no history of epilepsy or recent trauma and any drug intake. He had history of left facial cellulitis secondary to odontogenic infection one-week prior the onset of seizure. He was initially diagnosed with PRES after CECT brain.

Second case is a 6 years old boy, who presented with multiple infected impetigo lesions over bilateral lower limb, fever and vomiting. He also developed blurry vision and recurrent vomiting, requiring ICU care due to hypertensive crisis. CECT brain was performed and reported as PRES.

Similarly, both children had persistent microscopic hematuria and hypertension, and investigations show low C3 and normal C4 levels with raised Anti-streptolysin O titer (ASOT), fulfilling the criteria of PSGN.

Conclusions The incidence of PRES is rare in children, but should always be a diagnosis to be considered in children with PSGN presenting with hypertensive crisis and/or rapidly progressive neurological manifestations, e.g. status epilepticus. Early recognition and prompt treatment are important measures in reducing morbidity and mortality.

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THINK OUT OF THE BOX! LYMPHADENOPATHY, SKIN RASHES AND SKULL LESIONS; ALL ENDED UP WITH LANGERHANS CELL HISTIOCYTOSIS ?

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Background Langerhans cell histiocytosis (LCH), previously known as histiocytosis X, is a rare paediatric disorder. The abnormal proliferation of specialized dendritic cells found in skin and mucosa, which resembles langerhans cells, account for the underlying pathology of LCH (1). The vast clinical presentations of LCH, from self-limiting lesion to fulminant disseminated disease, make its diagnosis a challenge for doctors. Its incidence stands at around 1–5 per million children. Some literatures showed that children of parents who were exposed to certain chemicals such as benzene have an