antituberculosis therapy and fever resolved after 45 days. A nurse is going to her house daily to check the patient's adherence during the one year treatment.

Abstract 953 Figure 1 Chest radiography of miliary tuberculosis

Conclusion The diagnosis of MT can be clearly invoked with a simple and inexpensive investigation, even in an immunocompetent child. The typical image in the chest radiography is the most important reason to report this case.

954 PLEURAL EFFUSION AND ASCITES: AN UNUSUAL COMPLICATION OF HEPATITIS A

doi:10.1136/archdischild-2012-302724.0954

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Background and Aims To the best of our knowledge, we are reporting the first case from United Arab Emirates in the literature of Hepatitis A associated with pleural effusion and ascites. Hepatitis A is the most common cause of viral hepatitis in childhood and a major health problem in the developing world. Pleural effusion and ascites are very rare extrahepatic complications of hepatitis A. There have been only a few case reports in the literature of the two complications. The etiology is not clearly understood, but they tend to undergo spontaneous resolution and do not warrant specific diagnostic or therapeutic measures.

Methods Information collected from the hospital electronic case notes (Cerner).

Results Our case is a three years old Afghani boy who presented with fever and jaundice for two days. There was no history of recent travel abroad. Investigations revealed a high DIRECT bilirubin level and elevated liver enzymes. Hepatitis A virus immunoglobulin M (HAV IgM) was detected. During the course of hepatitis A, the child developed clinically significant and symptomatic bilateral pleural effusion associated with ascites. The diagnosis was confirmed with chest x-ray and ultrasonography. Lowest Albumin level was 23 g/l. Both pleural effusion and ascites resolved spontaneously without intervention.

Conclusions In patients with jaundice and pleural effusion and/or ascites, Hepatitis A is an important differential diagnosis. Both conditions are self-limited. Pleural and/or peritoneal diagnostic tapping is not warranted. Research is required to explore the underlying pathogenesis of the association.

955 FIRST-EVER CASE OF CEREBRAL TOXOPLASMOSIS IN HYPER IGE SYNDROME WITH DOCK8 MUTATION

doi:10.1136/archdischild-2012-302724.0955

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Background and Aims A ten year-old girl with hyper IgE syndrome caused by DOCK8 mutation was admitted to our hospital due to neuropsychiatric symptoms. Cranial MRI revealed multifocal cerebral lesions. Our aim was to clarify the etiology of these lesions by extended microbiology tests and comprehensive search in the literature then provide her with proper treatment options.

Methods Multiple blood and cerebrospinal fluid samples and were examined for bacterial and fungal culture, Aspergillus and Cryptococcus antigen, HSV, CMV, Mycobacterium and Toxoplasma PCR, panfungal PCR and for Toxocara and E. histolytica serology. Brain biopsy was also done for histology, bacterial and fungal culture.

Results All diagnostic assays showed negative results therefore causative agents could not be identified. For treatment, ceftriaxone and metronidazole combination was initially used accompanied by slight clinical and neuroradiological progression. Considering the possible presence of vascular brain lesions, high dose parenteral steroid treatment was introduced together with preemptive parenteral voriconazole therapy. Further progression in the clinical and radiological status was observed. Although there is no report of cerebral toxoplasmosis in this disorder, empirical anti-toxoplasma treatment was initiated with significant clinical improvement and radiological regression after 6-week therapy. Retrospective tests of CSF for Toxoplasma serology showed IgG titer increment.

Conclusion To our knowledge this is the first paper on cerebral toxoplasmosis in hyper IgE syndrome to date. In case of cerebral lesions in these patients Toxoplasma reactivation should be considered inspite of negative Toxoplasma PCR and antitoxoplasma treatment could be introduced in the absence of other etiologic factor.

956 INFANT BOTULISM DUE TO HONEY INGESTATION

doi:10.1136/archdischild-2012-302724.0956

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Background and Aims Botulism is a neuroparalytic disease caused by neurotoxins produced by the bacteria *Clostridium botulinum*. This neurotoxin inhibits the normal release of acetylcholine in the synaptic cleft, inducing presynaptic neuromuscular blockade. Infant botulism results from the absorption of heat-labile neurotoxin produced in situ by ingested *Clostridium botulinum*. Honey and environmental exposure are the main sources of acquisition of the organism.

Patient The patient was a 6-month-old girl with bilateral ptosis, muscle weakness, constipation and history of honey consumption. Lumbar puncture (LP), electromyography (EMG), nerve conduction study (NVS) and brain magnetic resonance imaging (MRI) were performed that all were normal. Stool evaluation for botulinum toxin was positive for toxin-A. Due to delayed diagnosis and improvement of general condition with conservative management botulinum anti-toxins did not started for her. After several days symptoms gradually improved and she was discharged with good general condition.

Conclusion Botulinum should be considered in every patient with weakness and ptosis. Botulism evaluation and appropriate management should be done.

957 MULTIPLE HUGE HYDATID CYSTS OF LIVER

doi:10.1136/archdischild-2012-302724.0957

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Background and Aims Hydatidosis, caused by *Echinococcus granulosus*, is an endemic parasitic disease. The most frequent anatomic locations are liver and lung. Dogs and other canines are the primary definitive hosts for this parasite. Hydatid cyst may develop after accidental ingestion of tapeworm eggs, excreted with the feces of these animals. Diagnosis is usually based on radiological and serological findings.

Patient We present a 6 Year-Old-Boy with abdominal pain since months ago. He had history of travelling to village and exposure to cattle and dog. Physical examination of head and neck, chest, extremities, and neurologic exam were normal. In abdominal Physical examination he had mild right upper quadrant tenderness without rebound tenderness. Liver was palpable 3 Cm below costal margin. Abdominal sonography and CT scan showed multiple huge cysts in liver that occupied almost all the liver space. Serum hydatid cyst antibody was positive. With diagnosis of hydatosis surgery was done and albendazole started for him. The patient improved after a while and he was discharged with good general condition.

Conclusion Hydatid cyst should be considered in every patient with liver cystic lesion in endemic area.

958 FATAL HUMAN BOCAVIRUS INFECTION IN A BOY WITH IPEX-LIKE SYNDROME AND VACCINE-ACQUIRED ROTAVIRUS ENTERITIS AWAITING STEM CELL TRANSPLANTATION

doi:10.1136/archdischild-2012-302724.0958

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We report about a 21-month-old boy presenting with chronic diarrhea and obstructive lung disease since infancy. His older brother, the first male child of non-consanguine Egyptian parents, had died at the age of 20 months suffering from BCGitis and severe CMV infection, suggesting a severe primary immunodeficiency syndrome.

We found immunological dysregulation, endocrine dysfunction and enteropathy compatible with IPEX syndrome, therefore the child was listed for stem cell transplantation (SCT). *FOXP3* gene showed no IPEX associated mutations, but sequencing *IL2RG* we found an mutation in Exon 2 associated with x-linked IPEX-like SCID (c.252C>A, p.N84K).

The pulmonary condition of the boy deteriorated and he was admitted on the ICU, where he was mechanically ventilated ever since. After common respiratory infections were excluded, chronic human bocavirus (hBoV) was identified by multiplex-PCR as the primary causative pulmonary agent causing his respiratory failure. hBoV was found in decreasing quantity in respiratory material, blood and stool specimen.

Interestingly, chronic rotavirus shedding was notified in repetitive stool specimen. As the boy had been vaccinated against rotavirus, vaccine-acquired chronic infection was suspected and confirmed by typing of a vaccine virus specific gene variant.

The boy died before SCT could be performed.

Since its discovery, there is an ongoing discussion if hBoV can cause serious infections or represents only a harmless "bystander". This case report shows that hBoV can result in lethal respiratory infections in immunocompromised children.

959 MYCOPLASMA PNEUMONIAE ASSOCIATED WITH STEVENS JOHNSON SYNDROME IN TWO CHILDREN

doi:10.1136/archdischild-2012-302724.0959

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Background Mycoplasma pneumonia (MP) infections are often asymptomatic but can involve multiple organ systems. Secondary skin reactions are common, although few patients infected develop Stevens Johnson syndrome (SJS).

Results We describe 2 cases of Mycoplasma pneumoniae chest infection associated with Stevens Johnson syndrome. The two patients had prodromal symptoms of an upper respiratory tract infection before the onset of the eruption. The patients had extensive epidermal bullous vesicles, oropharyngeal and genital ulceration, injected conjunctivae and sclera and swollen lips with flaccid bullae. The mycoplasma IgG and IgM titers returned positive, and blood cultures and other titers were negative. They were successfully treated with macrolides and glucocorticoids.

Conclusion Although the clinical course may be severe and prolonged, the prognosis is uniformly good with complete recovery.

960 SECONDARY PSEUDOHYPOALDOSTERONISM DUE TO PYELONEPHRITIS: TWO CASES REPORT

doi:10.1136/archdischild-2012-302724.0960

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Secondary pseudohypoaldosteronism occurs due to transient resistance in renal tubules causing renal sodium loss, hyponatremia and hyperkalemia. This may resemble congenital adrenal hyperplasia. This is to be considered specially when a urinary tract infection is present as treatment and prognosis varies.

We report two infants who developed pseudohypoaldosteronism secondary to acute tract urinary infection and urologic malformations.

Patient 1 27 days old infant, weight: 4056 gr. He suffers severe dehydration, hyponatremia 119 mmol/L, K 7 mmol/L and metabolic acydosis, normal serum creatinine and normal 17 OH progesterone. Pathological urine analysis and high levels of serum rennin and aldosterone. Urine culture: E.Coli. We can also appreciate left Ultrasonography hidronefrosis IV/V and primary megaureter. He was treated with Ampiciline + gentamicine and ions stabilisation within the following 36 hours after hospital admission.

Patient 2 14 days old infant, 2510 gr. Admitted at emergencies with a severe dehydration. Hyponatremia and hyperpotasemia that needed urgent treatment and severe metabolic acydosis. Electrolytic disorders were amended, then a urine culture was done with positive results in E. Coli test. He was treated with ampiciline + gentamicine. The renal scan evinced a bilateral ureteral hydronephrosis. The infant showed a recovery thanks to antibiotics treatment.

Conclusions An endocrinological evaluation is necessary to a diagnosis but we mustn't forget the urinary cultures.

Most probable cause is the immature renal responsiveness to aldosterone in three first months of life when exist urinary tract anomalies and/or urinary tract infection.

This secondary pseudohypoaldosteronism is reversible with early treatment.

961 THE DIAGNOSTIC VALUE OF NEUROIMAGING IN RECOGNIZING AND THERAPY MONITORING OF ENCEPHALITIS AND MENINGITIS IN CHILDREN

doi:10.1136/archdischild-2012-302724.0961

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