mind in the diagnosis of children patients who had complaints that fever, weakness, weight loss and especially joint pain, where pediatric clinics.

P624 HOSPITAL-ACQUIRED NEONATAL MENINGITIS: EXPERIENCE OF THE NEONATOLOGY DEPARTEMET OF SFAX (TUNISIA)

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Background The past decade brought with it both highly sophisticated neonatal intensive care with improved perinatal mortality rates and increased risk for nosocomial septicemia and meningitis among survivors. Although most of these infections were caused by multiple antibiotic-resistant bacteria. The absence of specific clinical signs makes diagnosis of meningitis more difficult.

Methods We enrolled neonates who were admitted to neonatal unit at Hedi Chaker hospital, Sfax from 1 January 2007 to 31 December 2016 and had suspected nosocomial infection with abnormal cerebrospinal fluid examination.

Results Five cases were reviewed, Among these 4 were girls (80%), All neonates were preterm between 27 and 34 weeks of pregnancy, with Low birth weight in 2 cases and very Low birth weight in 3 cases, the average age of diagnosis was 14.2 days after hospitalization, 4 (80%) of them had concomitant bacteremia, suspected bacterial infection was confirmed in 4 cases (80%), by positive results on blood culture in 4 cases but only one cerebrospinal fluid (CSF) culture was positive, *Klebsiella pneumoniae* was the leading pathogen. The case mortality rate (CMR) was 0.8. Four patients died and one case was cured but he developed a sequellae of psychomotoric retardation.

Conclusion Preterm neonates have a high risk of developing nosocomial infections and especially Nosocomial Meningitis wich is related to a high case mortality rate, Definitive diagnosis is made by cerebrospinal fluid examination (CSF) via lumbar puncture (LP), which should be performed in any neonate suspected of having sepsis or meningitis. Future efforts should be directed toward better definition of bacterial virulence, host susceptibility and preventive measures.

P625 IMPACT OF ANTIBACTERIAL DRUGS' OVERUSE IN DURATION OF HOSPITALIZATION AMONG PATIENTS WITH ACUTE DIARRHEAL DISEASES IN 'NORK' ICH, ARMENIA, 2018

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Background Antibiotics were announced as life-savers when they became widely available in the middle of 20th century. Nowadays, antimicrobials are fast becoming killers themselves. The more any given antibiotic is used, the greater the chances

that bacteria will develop antimicrobial resistance (AMR) that renders the drug ineffective.

Although we don't have exact number of deaths from antibiotic-resistant infections in Armenia, we give importance to its prediction and prevention of developing multidrug resistant strains of bacteria. Our goal is to describe the low cost-effectiveness of treatment in accordance with national and international guidelines to hospitalized patients with acute diarrheal diseases. It is an option why not to use antibiotics inappropriate.

Objective The purpose of this study was to evaluate the cost-effectiveness of treatment with national guidelines and so to create a premise for prevention of inappropriate antibiotic use.

Methods A retrospective economic analysis was performed using 45-day (01.04.-15.05.2018) data from "Nork" ICH. The analysis was conducted from a third-party payer's perspective.

During the above mentioned period 156 patients were admitted from which 125(80.1%) had watery and 31(19.9%) bloody diarrhea. Antibacterial treatment was given to 80 patients, but only in 23(28.75%) cases it was indicated. Widely used antibacterials were nifuroxazide 55.6%, azithromycin 4.4%, amoxicillin 1.1%, metronidazole 3.3%, ciprofloxacin 13.3%, TMP-SMX 6.7%, cefotaxime 1.1%, ceftriaxone 14.5%.

Patients with appropriate antibacterial treatment were hospitalized 6.8 days on average. The patients without any antibacterial treatment and patients with inappropriate antibacterial treatment were hospitalized 4.7 and 6.3 days on average respectively.

Average Total Cost of treatment was calculated as follows:

a – average duration of hospitalization b* – average cost of treatment per patient per day

* - it is fixed cost – 36.7EUR

Eventually, average costs of treatments have been determined.

Results Since average cost of treatment is known- 36.7EUR per patient per day, totally 172.49EUR per patient, it can be compared with inappropriate treatment cost-totally 231.21EUR per patient. The difference in the costs of two treatment options is 58.72 EUR per patient. Multiplying by the number (57) of patients managed with inappropriate treatment we will have total difference 3347EUR during 45 days.

Conclusions Treatment in accordance with national guidelines is cheaper in comparison with inappropriate antibacterial treatment as the last requires longer duration of hospitalization.

The implementation of guidelines is important from both clinical and financial points of view.

P626 INFLAMMATORY MARKERS OF ANTIPSYCHOTIC WEIGHT GAIN AND CARDIOMETABOLIC DYSFUNCTION IN YOUTH MENTAL HEALTH DISORDERS

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Introduction Second generation antipsychotics (SGAs) are prescribed to treat mental health disorders in children. However, there are concerns about these medications due to

cardiometabolic side effects including weight gain with a risk of Type 2 Diabetes Mellitus. There is limited understanding of the factors increasing susceptibility to these side effects. It is established that increased adiposity associated with weight gain is mediated by the emergence of a persistent low-grade inflammatory state. However, there has been no research investigating the relationships between pro-inflammatory states in children and the cardiometabolic side-effects of SGAs and there are no clinical indicators of those at risk.

Aims

1. To determine whether there is a subgroup of patients at baseline who present with a profile of immune dysregulation.
2. To investigate how SGA medication impacts on inflammatory markers and cardiometabolic function in children.
3. To investigate the potential to predict those at greater risk of developing adverse metabolic outcomes in response to the treatment with SGAs.

Methods We are recruiting children and adolescents (5-18 years) who are commencing SGA medication. We assess the cardiometabolic profile clinically and biochemically and obtain serum and peripheral blood mononuclear cells (PBMCs) to measure levels of inflammatory markers. Through comparison with biobanked healthy control samples, we will determine if there is a subgroup at baseline with a pro-inflammatory profile. The patient groups are assessed longitudinally at 3, 6 and 12 months to measure BMI percentile and cardiometabolic function. Changes in immune cells and inflammatory markers are measured including IL-1, IL-10, IL-17, sCD-163, TNF- α and IFN- γ as well as leptin, ghrelin and adiponectin in response to treatment with antipsychotics.

Results Twelve patients have been recruited and are being followed up longitudinally. Of the data analysis that has been completed, it appears that leptin and TNF- α levels have increased in the participants with the most weight gain (6–9 kg) between baseline and 3 months after commencing SGAs.

Discussion Identification of a high-risk group for weight gain is vitally important and it would allow clinicians to work with these patients to minimise the metabolic side effects of the medications; through rational SGA choice, promotion of intensive monitoring and implementation of preventative treatment regimes. Antipsychotic use is a prominent cause of obesity in young people with mental health disorders. Through this study, we aim to modify clinical practice, which we hope will lead to better guidelines to reduce the risks.

P627 MITOCHONDRIAL DISEASE MIMICS

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A diagnosis of primary mitochondrial disease was traditionally arrived at on the basis of clinical and biochemical features including abnormal respiratory chain analysis on muscle biopsy and/or identification of other 'mitochondrial disease markers'. With the increased availability of genetic testing, in particular massive parallel sequencing, alternative primary diagnoses which result in secondary mitochondrial dysfunction are being identified

We present a cohort of six cases who previously had a diagnosis of mitochondrial disease. Alternative primary diagnoses have recently been identified which includes Andersen-Tawil syndrome (gene: *KCNJ2*), COL4A1-related brain small-vessel disease (gene: *COL4A1*), cardiofaciocutaneous syndrome (gene: *BRAF*), autosomal recessive spinal cerebellar ataxia-10 (gene: *ANO10*), facioscapulohumeral muscular dystrophy (gene: *DUX4*) and IGSF1 deficiency syndrome (gene: *IGSF1*).

Conclusion The reported cohort highlights the important point that many genetic conditions may mimic mitochondrial disease and, although the phenotype and biochemical tests may indicate mitochondrial disease, we suggest that genetic confirmation is required to secure a diagnosis. Establishment of an accurate diagnosis is important, not just prognosis and planning of management and treatments regimes, but also for appropriate genetic counseling and the identification of other at-risk family members for possible cascade analysis. The link between many of these primary diagnoses and secondary mitochondrial dysfunction is poorly understood but reporting such cases will allow these pathways to be elucidated and understood.

P628 WILSON'S DISEASE IN CHILDREN: ABOUT 8 CASES

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Introduction Wilson's disease is a rare disease characterized by its clinical heterogeneity that causes diagnostic difficulties.

Patients and methods This is a retrospective study in 8 patients with Wilson's disease.

Results The study is about 4 girls and 4 boys. The mean age at diagnosis was 8 years and 8 months. The average diagnostic delay was 58 days. All our patients had liver damage at the time of diagnosis. An anxiodepressive symptomatology was noted in one case. The peri-corneal ring of Kayser-Fleischer was objectified in 2 cases. Hepatocellular insufficiency and cholestasis were observed in 4 cases. Thrombocytopenia was present at the time of diagnosis in 3 cases and hemolytic anemia with Coombs test negative in one case. The genetic study revealed the presence of 2 mutations in the homozygous state in 1 case, 2 mutations in the heterozygous state in 1 case and the absence of mutations in 2 cases. Thanks to the family survey, 2 patients were identified at the presymptomatic stage.

The evolution under treatment with D-penicillamine was favorable for 7 patients. Neurological symptoms occurred in one case. The average decline is 5 years and 4 months.

Conclusion Wilson's disease is a curable genetic condition, its prognosis is all the better when the diagnosis is made in time and as a result the treatment is initiated early.