Pedia Companion

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Dear Doctor,
Greetings from Micro Labs Limited. We are happy to bring you "PEDIA COMPANION" which contain latest and interesting news in the field of Paediatrics.

This issue contains:

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Evaluation of the clinical characteristics of pediatric patients with anaphylaxis

Introduction

Food was the most triggering factor for anaphylaxis, followed by drugs.

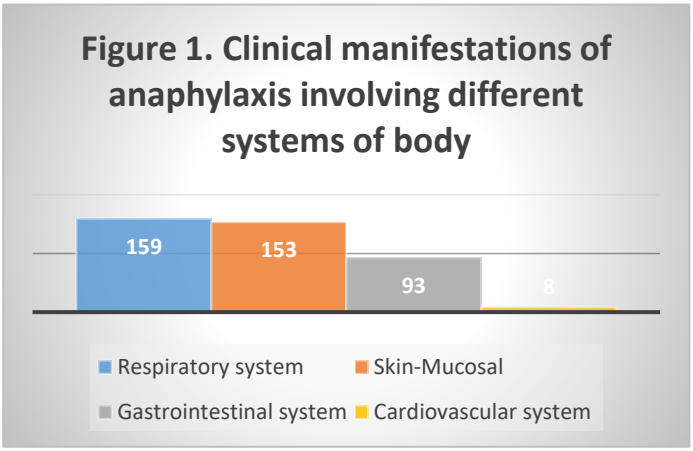
Anaphylaxis is a severe and sometimes fatal hypersensitivity reaction characterised by sudden onset of multisystem involvement.[1] Anaphylaxis includes respiratory and airway difficulties, circulatory collapse, mucosal inflammation, and other consequences.[2] Although anaphylaxis affects people of all ages, diagnosing and treating it in children presents more difficulties, making pediatric cases even more critical. The most important elements for diagnosing anaphylaxis in a patient are the patient's medical history and clinical findings. As the incidence of anaphylaxis are increasing worldwide, understanding the clinical characteristics and causes in children is important to improve patient care and clinical outcomes. [1] Hence, this study aimed to investigate and analyse the clinical profiles of pediatric patients diagnosed with anaphylaxis, with the goal of understanding and improving the management of the potentially life-threatening condition.

Methods

A retrospective review of data on patients aged between 0-18 years with the history of anaphylaxis were included in this study. A total of 186 patients with the history of anaphylaxis were included. The study included an evaluation of the patients' age at diagnosis, gender, allergy history, probable allergen-causing anaphylaxis, and clinical symptoms during anaphylactic episodes.

Results

Exposure to a potential allergen occurred through oral contact was seen in majority of patients (74.7%, n=139). Food accounted for 41.9% of cases (n = 78) of potential allergens for anaphylaxis, drugs for 40.3% (n = 75), bee venom for 7.5% (n = 14), and idiopathic/other reasons for 10.2% (n = 19). Among the patients, the respiratory system involvement was seen in 85.5% (n = 159), the skin and mucosa in 82.3% (n = 153), the gastrointestinal system in 50% (n = 93), and the cardiovascular system in 4.3% (n = 8) (Figure 1). Adrenaline was administered to all patients upon admission. Of the patients, 24.7% (n=46)



had a prior history of allergies. 80.4% (n = 37) of these patients with a history of allergies who had documented food allergies, 17.4% (n = 8) had inhaled allergies (dust mites, pollen, pets), and 2.2% (n = 1) had drug allergies. Most common probable allergens triggering anaphylaxis was food items. Tree nuts were the most common likely trigger among food allergies, accounting for 35.9% (n=28) of cases. Anaphylaxis may be caused by hen's eggs in 14.1% of cases (n=11) and milk in 16.7% of cases (n=13). Among tree nuts, hazelnuts (32.1%) and walnuts (32.1%) were the most frequent causes of anaphylaxis followed by peanut (21.5%), almond (7.1%), pistachio (3.6%), and cashew (3.6%). Antibiotics were the most often occurring allergens among drugs (n=42, 56.0%). Amoxicillin/Clavulanic acid was the most common likely allergen among antibiotics (n=16, 21.3%).

Discussion

Food items accounted for 41.9% of patients who had anaphylaxis, drugs for 40.3%, and bee venom for 7.5% of cases. Tree nuts ranked first among food allergies with 35.9%, followed by milk (16.7%) and hen's egg (14.1%).[1] According to research conducted in many countries, food is the most common cause for anaphylaxis, which is consistent with the results of this study. In a study evaluated at a multicentre anaphylaxis registry in Korea, among 284 cases, hen's eggs were the most common cause of FIA in children, followed by cow's milk, peanuts, wheat, and walnuts.[3] In this study, Amoxicillin/Clavulanic acid was the most frequently triggering drug for anaphylaxis.[1] The most often implicated antibiotic was amoxicillin/clavulanic acid, which was consistent with the findings of this investigation. Overall, among drugs causing anaphylaxis, NSAID-induced anaphylaxis ranked second in prior research.[4]

Conclusion

Assessing the clinical features of individuals suffering from anaphylaxis is important for improving the understanding and therapeutic strategy for this complex hypersensitivity reaction in children. This approach seeks to optimise the identification, appropriate treatment, and prevention of anaphylactic responses, which will eventually enhance children's health and quality of life.

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Assessment of polyp burden differences in paediatric Peutz-Jeghers syndrome through Capsule Endoscopy

Introduction

Video Capsule Endoscopy directed double balloon enteroscopy is an effective strategy to reduce the polyp burden and decrease the need for surgical intervention

Peutz–Jeghers Syndrome (PJS) is characterised by a genetic propensity to develop small intestine hamartomatous polyps that first appears in early childhood and an elevated risk for cancer in multiple organs, mostly at adulthood.[1] PJS is caused by heterozygous germline pathogenic variants (PV) in the serine threonine kinase 11 tumour suppressor gene (STK11/LKB1 gene). The majority of childhood symptoms, such as bleeding, anaemia, and obstructive symptoms, are caused by polyp related complications.[2] The pediatric population lack detailed description of the polyp burden, growth characteristics over time, and impact of therapeutic intervention on polyp burden. Hence, this study aimed to describe and evaluate polyp burden and evaluate in pediatric patients with Peutz– Jeghers Syndrome.

Methods

Retrospective longitudinal cross-sectional analysis of video capsule endoscopy (VCE) studies were carried out on pediatric patients with the clinical diagnosis of PJS. This study included 15 patients. Using linear mixed models adjusted for clustering, polyp burden variables were modelled as functions of patient and study characteristics. Study outcome is to examine total polyp count, maximum polyp size, and maximum luminal occlusion. Multivariate models were fit to examine each outcome variable as a function of patient gender, age at visit, total VCEs, duration of study visit, and procedure.

Results

When VCE occurred, the patients were asymptomatic in the majority of cases (74%) (Figure 1). The polyp burden, as determined by the total number of small bowel polyps and the largest polyp size, resulted in less than 30 polyps and <20 mm. Only four studies detected more than 10 polyps and one or more polyps > 21 mm in size. Small bowel involvement was more common in the proximal region (77%) than the distal region

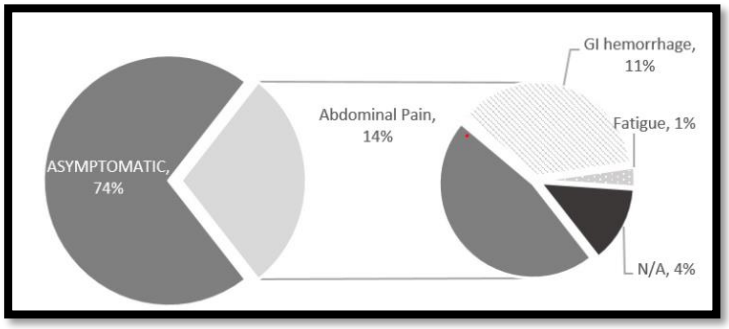

Figure 1. Symptoms at the time of VCE in pediatric PJS

(66%). Males had larger adjusted largest polyp sizes. The use of double balloon enteroscopy (DBE) has been associated to a lower polyp load.

Discussion

When determining the load of polyps, VCE has proven to be the most accurate method. This is especially true for polyps that can be removed using device-assisted enteroscopy (DAE), such as DBE (Double Balloon Enteroscopy). Most of the patients were asymptomatic at the time of VCE and if symptomatic (32%), abdominal pain and anemia were the most frequent symptoms.[1] Findings suggests that a patient's genotype, more particularly, truncating mutations of STK11, predisposes them to an increased incidence of intussusception.[3] Reduced maximal luminal blockages, reduced maximal polyp sizes, and comparatively lower polyps were observed in VCE investigations conducted following DBE. Nakayama et al. reported a patient 5 hours after the onset of acute abdominal symptoms, a DBE was conducted. The results revealed a large polyp in the jejunum with an intussusception stalk, and both endoscopic mucosal excision and reduction of the intussusception were performed successfully at the same time.[4]

Conclusion

The proximal two-third of the small intestine has a comparatively low polyp burden in paediatric PJS patients, with a size that can be removed via enteroscopy. A greater polyp load was correlated with male gender and older age.

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Association of preterm birth at very low birth weight with adult cardiometabolic biomarkers

Introduction

Preterm birth at very low birth weight is a risk factor for impaired glucose and fatty acid metabolism.

Preterm birth, which is defined as a birth at <37 weeks of gestation, affects 10.6% of pregnancies worldwide, accounting for 14.9 million preterm births annually. Of all preterm births, 75% are late preterm births, which are commonly classified as births between 34 and 36 weeks of gestation. Early life exposures in the prenatal and early postnatal stages can influence the susceptibility to chronic diseases that develop in adulthood.[1] Prematurity is linked to the components of metabolic syndrome and adult-onset cardiovascular disease, and it has an impact on subsequent cardiovascular health. When comparing preterm and term-born individuals, for example, insulin resistance, increased fat mass, blood pressure, cholesterol, fasting glucose, and insulin levels have all been linked to prematurity.[2] This study aimed to assess glucose regulation and cardiometabolic biomarkers in adult very low birth weight (VLBW, <1500g) survivors, using siblings as controls.

Methods

Biochemical tests were performed among 74 VLBW adults and 70 siblings; 66 of the subjects were matched sibling pairs, and the remaining 12 were unpaired provided blood samples and underwent oral glucose tolerance test. After an overnight fast, peripheral venous blood samples were taken, and a 2-hour, 75 g oral glucose tolerance test (OGTT) was conducted. Blood samples were taken at baseline, 30, 60, and 120 minutes after consuming the glucose solution in order to measure the levels of glucose, insulin, and free fatty acids (FFA). Assessment was also done for cardiometabolic biomarkers.

Results

23 (31%) VLBW and 11 (16%) sibling-controls met World Health Organization criteria of impaired glucose regulation. VLBW participants had 9.2% higher 2-h glucose than their siblings after adjusting for age and sex. Additionally, VLBW subjects had greater fasting (13.4%, −0.3% to 29.0%) and 2-hour free fatty acid levels (15.6%, −2.4% to 36.9%) (Figure 1). Both fasting free fatty acids (FFA) and 2-h free fatty acids were higher in VLBW-adults. 2 VLBW and 1 sibling met the WHO criteria for Type 2 diabetes, while 3 VLBW and 1 sibling had impaired fasting glucose (IFG), and impaired glucose tolerance (IGT) was found in 18 VLBW and 9 siblings. After confounders were further taken into consideration, the differences were statistically significant. Regarding other tested indicators, such as insulin resistance, atherogenic lipid profiles, or liver tests, no statistically significant differences were seen.

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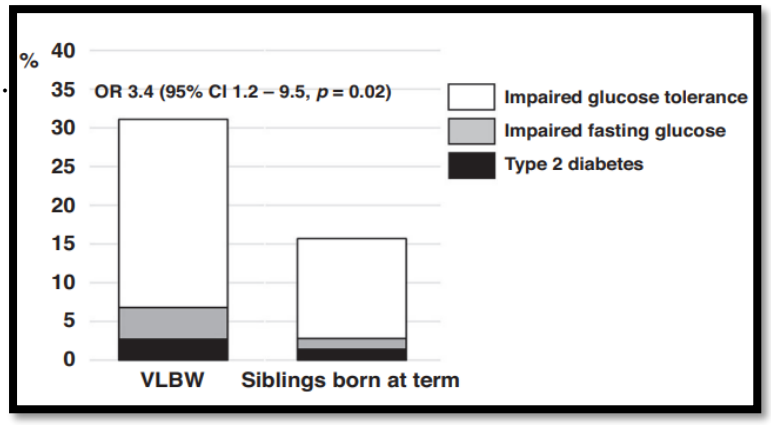

Figure 1. Impaired glucose regulation in adults born preterm at very low birth weight (VLBW) and term-born siblings

Discussion

In comparison to siblings, VLBW adults showed 3.4 fold higher odds of having impaired glucose regulation. Additionally, results showed higher 2-h glucose concentrations, fasting and 2-h FFA concentrations. However, there were no variations observed in the 2-hour insulin concentration, fasting glucose, or fasting insulin levels. There were similarities across the groups for several cardiometabolic biomarkers, such as lipid profiles, liver testing, inflammatory markers, and hyperandrogenism measurements.[2] Preterm birth is considered as risk factor for metabolic syndrome and cardiovascular disease in adult life.[3] Yun et al. showed children's aged 6-8years who were born very preterm/ very low birth weight showed lower weight and fat mass, they also showed significantly higher blood pressure, fasting glucose, homeostatic model assessment of insulin resistance (HOMA-IR) and leptin levels.[4]

Conclusion

Adults of VLBW demonstrated more impaired regulation of glucose and fatty acid metabolism. Compared to previous non-sibling investigations, there were less differences in cardiometabolic indicators. A portion of this could be explained by common environmental, genetic, or familial variables.

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Development of plastic bronchitis in an immunocompromised child with respiratory syncytial virus infection: A rare case report

Introduction

Bronchoscopy should be considered in patients with plastic bronchitis

Plastic bronchitis (PB) is caused by an endogenous foreign body in the airway which causes extensive bronchial blockage, resulting in pulmonary ventilation dysfunction, respiratory insufficient, and life-threatening respiratory failure.[1] The condition can aggravate over time if the obstruction is not found and removed in a timely manner.[2] This case report describes an immunocompromised child with respiratory syncytial virus (RSV) infection who developed fatal PB.

Case presentation

A 2-year-old female patient was admitted to hospital with the complaints of a cough, fever for 5 days, and worsening shortness of breath for 1 day. The child presented with paroxysmal productive cough, cyanosis, and fever five days prior to admission. Her body temperature was 40 ℃ and four days later the child experienced cough and significant shortness of breath then admitted to hospital with presumptive pneumonia. Past medical history showed that she was diagnosed with acute myeloid leukemia (M5, CR1) and was in the induction phase of chemotherapy. Throat swab and alveolar lavage fluid PCR for respiratory pathogens showed positive for RSV only. Chest CT scan suggested consolidation of both lungs with segmental atelectasis (Figure 1).

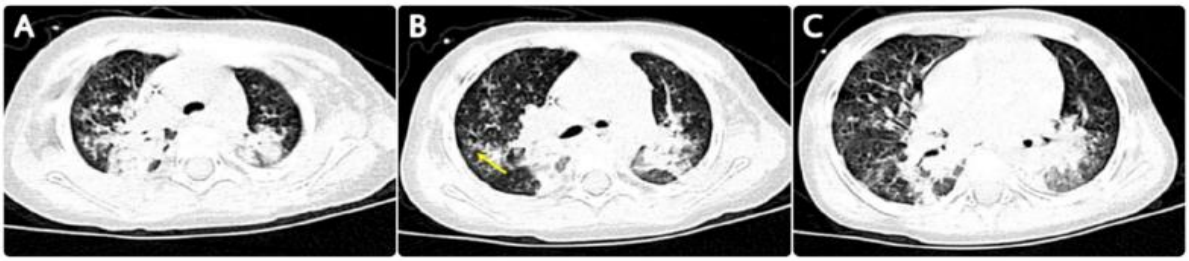

Figure 1. Chest CT on admission showing consolidation of both lungs with segmental atelectasis, centrilobular nodules, and a sign of tree-in-bud (yellow arrow)

As the patient was on induction phase of M5 chemotherapy with neutropenia and fever treatment includes intravenous meropenem (20 mg/kg q8h), with high flow nasal catheter oxygen (HFNC), nebulization (with budesonide 2 ml, ipratropium 2 ml. salbutamol aerosol 1.25 ml q8h), rehydration and antipyretic. On the 6th day of hospitalisation patient oxygen

saturation was 88% so intubation and mechanical ventilation was given after which the oxygen saturation was less than 90%. Due to mechanical ventilation, lung injuries such as subcutaneous emphysema and mediastinal pneumatosis occurred. Repeated chest radiographs showed signs of white lung. Considering airway obstruction, bronchoscopy was performed on the 10th, 13th, 15th, and 19th days of ECMO operation. After which, the tidal volume increased. Pathology of the plastic plugs showed fibrinous secretions, as well as red blood cells, lymphocytes, and neutrophils. Withdrawal of EMCO was done 35 days later. During discharge, patient was on HFNC (FiO2 34%, flow 13 L/min) with, an oxygen saturation maintained above 95%. Six months after the patient's discharge, a CT scan of the chest showed interlobular septal thickening, hyperinflation, atelectasis, and uneven inflation in both lungs. Consequently, bronchiolitis obliterans (BO) was diagnosed. Budesonide nebulization, low-dose azithromycin, and oral montelukast sodium were administered. 19 month's post discharge, a follow-up CT scan revealed atelectasis, interlobular septal thickening, and a mosaic attenuation pattern (Figure 2). Pulmonary fibrosis was first treated with oral pirfenidone. After more than two years of observation, no BO aggravation was noted.

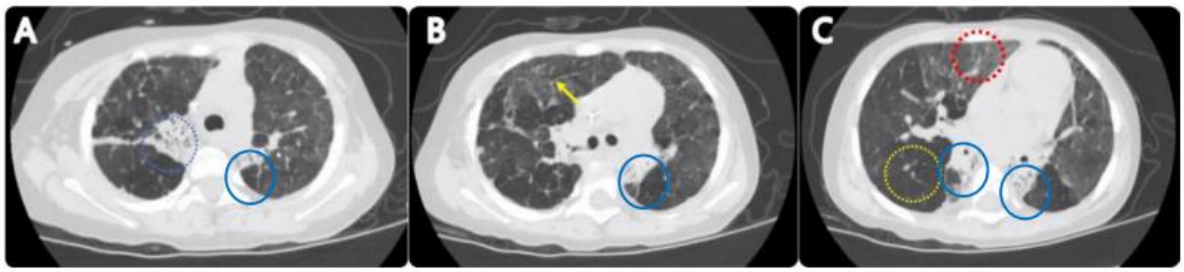

Figure 2. CT scan after 19months of discharge showing mosaic attenuation pattern, (hyperinflation (yellow circle) and ground glass opacity (red circle)), atelectasis (blue circle), and interlobular septal thickening (yellow arrow) in both lungs.

Discussion

The patient's main complaints were a sudden onset of cough, fever, and progressive dyspnoea. The patient had a past medical history of acute myeloid leukaemia M5, which progressed rapidly and severely. The patient had high ventilator parameters, and even with pure oxygen supplementation, it was difficult to keep the arterial oxygen saturation above 80%. Then the patient showed signs of air leakage and their oxygenation index (OI) was greater than 40, ECMO operations were carried out. After numerous bronchoscopy and early lavage to remove a large amount of bronchial plastic secretions, the patient's dyspnoea considerably improved while on ECMO.[1] Li et al. stated bronchoscopy and chest physiotherapy for airway clearance are among the most-utilized therapies for the treatment of PB in paediatric patients.[3]

Conclusion

Respiratory syncytial virus particularly in immunocompromised patients, may be a possible etiological factor for PB. In such a case, a bronchoscopy procedure and the diagnosis of PB

should be taken into consideration if treatments like mechanical breathing and ECMO have failed to alleviate symptoms. Treatment requires immediate elimination of the plastic secretions from the airway.

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