IAP BANGALORE-BPS
NEWSLETTER

Term: 2019 -20 | Issue 1 | January 2019

PRESIDENT'S MESSAGE

Greetings from IAP-BPS, Bangalore. Happy new year and Happy Makara Sankranthi to all .

It is my duty to congratulate my predecessor Dr Natesh and secretary Dr Kushal for their wonderful and memorable work last year. I am indebted to Dr Jayoji Rao and Dr PC Bopaiah's family for their hospitality and great response towards our association. I am really very fortunate to have a great team with a dynamic secretary, Dr Sumitha Nayak, and Treasurer, Dr Shivaprakash Sosale. I am sure we can make a remarkable contribution to our association this year. My dream is to construct the IAP Bhavan at our site in Rajarajeswarinagar and celebrate the Valedictory function in this building this year. I request all our seniors to bless us in this project.

We have lined up a year full of academic activities. Besides this we are honoured to have five major National conferences being held in our city, for each of which IAP-BPS is the co host. I request each of you to actively participate in every monthly meet of IAP-BPS which will be highly educative sessions and will be beneficial for all. We look forward to your support to make this an academically successful year. Happy reading!
Jai Hind. Jai IAP

Dr Srinivasa S
President, IAP Bangalore-BPS.

SECRETARY SPEAKS..

It gives me great pleasure to pen these lines, as I present to you the first edition of our e-journal. In order to make this an interesting read, we will carry both academic and non-academic articles. We have many extremely talented members in various areas of arts. I request you to send in your contributions ,which can be published in this e journal.

Plenty of interesting scientific programs and workshops have been planned for this year. Besides all the National Conferences which IAP BPS will be partnering , we have planned smaller focused interactive sessions, that will provide invaluable learning for all. I request each of you to kindly support these initiatives by attending these programs in large numbers.

May you have a great year ahead.....

Dr Sumitha Nayak

Hon Sec, IAP-BPS 2019

EDITORIAL MESSAGE

Happy New Year and Warm Greetings!

It is indeed a privilege to be associated with IAP-BPS as journal editors. It is a proud moment for us as we release the first edition of the E-Pediscan newsletter for 2019. We assure you of the best quality articles and look forward for contributions from each one of you for the future issues.

With warm regards,

Dr Nandeesh B.

Dr Priya Shivalli

UPCOMING EVENTS

Feb 2, 2019 - One day Workshop on Child Sexual Abuse, IMA Hall 8.30 AM - 5.00 PM

Feb14 - 17, 2019 - Indian College of Allergy, Asthma and Applied Immunology Conference

Feb 24, 2019 - IAP ID KTK Inaugural CME

PHOTO GALLERY

Dr Natesh, Past President, at RR Nagar site

Dr Srinivasa, Dr Jayoji Rao with Mrs P. C. Bopiah and sons

First Executive Committee meeting

Free health checkup camp at RR Nagar site

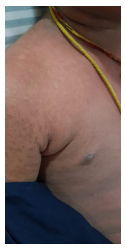
CASE REPORT

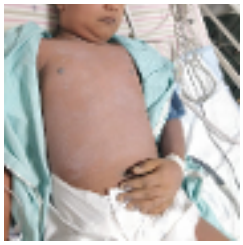
Fever, Rash & Shock - Medicine or Microbe - Who is the culprit?

Dr.Sindhu M V, Dr.Supraja Chandrasekar, Dr.Gurudutt, Dr.Nanditha, Dr.Bhavya KB; PEOPLE TREE Hospitals;

A 11-year-old male child presented with high-grade intermittent fever and a generalized rash for 10 days. He was becoming progressively sick with generalised oedema lethargy and increasing nature of fever. He presented a month ago with unprovoked generalised seizures diagnosed as Neurocysticercosis and started on Eptoin, Albendazole, and Steroids. The latter was tapered and stopped in 3 weeks.

On presentation, child was sick looking with 104 F fever, tachypnea, tachycardia, flushed with bounding pulses and generalised oedema suggestive of hypotensive warm shock. Distinct diffuse erythematous, maculopapular rash with normal





mucosae, was noted all over the body including palms and soles associated with hepatomegaly and ascites. No obvious focus of infection. Initial impression was viral exanthema in septic shock. Child required multiple fluid boluses and significant dose of Noradrenaline followed by steroids to correct shock. Broad spectrum antibiotics were initiated and other supportive medications. Platelets and Total counts were unremarkable except Eosinophilia. Initial septic workup including CRP, blood culture and urine culture were negative. Procalcitonin was high. Mild transaminasaemia noted with normal renal function tests and coagulation. Dengue serology Typhidot and Weil Felix test were negative. USG abdomen revealed mild hepatomegaly with minimal ascites. As fever and rash persisted despite all infective markers negative, non-infectious aetiology was sought. Dermatologist strongly considered drug rash; eosinophils noted in retrospect were significantly high [19 % AEC 2300]. A clinical diagnosis of DRESS SYNDROME was made and Phenytoin was withdrawn. Bone marrow aspirate showed a normocellular marrow. Rheumatologist confirmed the same and started child on pulse dose of methyl prednisolone for 5 days. Child dramatically responded with absence of fever and complete resolution of rash and general condition improved. Gradually the steroids were tapered and stopped in next few weeks with the child making a complete recovery.

DISCUSSION:

DRESS (Drug reaction with eosinophilia and systemic symptoms) is a rare, potentially life-threatening, drug-induced hypersensitivity reaction that includes skin eruption, hematologic abnormalities (eosinophilia, atypical lymphocytosis), lymphadenopathy, and multi organ involvement1-3. The skin involvement can range from maculopapular rash to Stevens-Johnson syndrome and erythema multiforme. Laboratory tests typically show eosinophilia and atypical lymphocytes4-5.

Drugs incriminated commonly for DRESS syndrome include aromatic anticonvulsants (phenobarbital, carbamazepine, phenytoin, lamotrigine), sulfonamides, vancomycin, allopurinol, antiretroviral drugs etc. It has been suggested that concomitant Human Herpes Virus-6 (HHV-6) infection increases the risk to develop DRESS syndrome 1-2.

There are 7 J-SCAR criteria for diagnosis of DRESS syndrome:(1) maculopapular rash developing 3 weeks after beginning treatment with the causative drug; (2) prolonged clinical symptoms after discontinuing the causative drug; (3) fever >38°C (4) hepatic abnormalities (5) leukocyte abnormalities: leucocytosis, eosinophilia (6) lymphadenopathy (7) HHV-6 reactivation. Late onset of symptoms (2-6 weeks following culprit drug ingestion) with delayed resolution after withdrawal of causative drug is an important feature of DRESS4-6.Immediate withdrawal of the causative drug and initiation of systemic corticosteroids is the mainstay in the management of DRESS syndrome 6.

Conclusion:

DRESS syndrome is a drug hypersensitivity reaction with cutaneous and systemic manifestations.

DRESS syndrome is potentially fatal with a 10% mortality rate hence clinicians must be vigilant when using common culprit medications and be aware of typical delayed (after 2-4 weeks) presentations.

DRESS can mimic any acute infectious disease and can present with septic shock with SIRS response which fails to respond to broad spectrum antibiotic. Hence a high index of suspicion is required for diagnosis and management.

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ACTIVITY REPORT

FREE HEALTH CHECK UP CAMP CONDUCTED AT PATTANAGERE, RR NAGAR, BANGALORE ON 13.1.2019

BPS MEMBERS PRESENT:

1. Dr S Srinivasa - President
2. Dr Sumitha Nayak - Secretary
3. Dr Sunil BM - EC Member
4. Dr Harilal Naik - EC Member
5. Dr Jayoji Rao - Member Building Committee

The free health check up camp began at 10.30 AM.

About 50 children, all below the age of 12 years, who are from the weaker sections of society, were examined. The most common issues noted were the presence of anemia, multiple vitamin deficiencies and upper respiratory infection.

These children were examined by the doctors of IAP Bangalore BPS who were present. They were given free medicines which had been donated by the IAP Bangalore members.

The residents of the society were very appreciative of our initiative. They have requested IAP Bangalore to conduct this event on a regular basis at the same location.

CULTURAL CORNER

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CASE REPORT

Seizures and cerebellar calcification in a child with autoimmune polyendocrine syndrome 3A.

Rachel Ranitha Peterson, Asha Kuruvilla

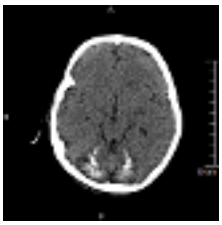
Bangalore Baptist Hospital, Bangalore.

A six-year-old girl, born to consanguineous parents presented with polyuria and weight loss for two weeks. Six months prior, she had multiple episodes of generalized seizures for which she was on anticonvulsants. She had history of developmental delay in all domains after the age of one year and at presentation she had a developmental age of about three years.

On examination she was found to have proportionate short stature with bone age of three years and six months. Her speech was dysarthric and she had a wide based ataxic gait with increased tone and exaggerated deep tendon reflexes in the lower limbs. Investigations showed random blood sugar of 369 mg/dL, HbA1C of 10.5 % and urine ketones was positive. TSH was > 100 (0.3-4.5100 mIU/ml), and Free T4 was 0.364 (0.97-1.67 ng/dL). Anti TPO (thyroid peroxidase) antibody was elevated at 813.59IU/ml and anti-GAD (glutamic acid decarboxylase) antibody was >2000IU/ml confirming autoimmune origin. Serum calcium, phosphorus, parathormone and cortisol were normal. Non-enhanced CT scan of the brain showed bilateral symmetrical calcification and mild atrophy of the cerebellar folia [Fig 1]. MRI brain showed mild cerebral atrophy, marked cerebellar atrophy and bilateral symmetrical cerebellar calcification. She was diagnosed to have Autoimmune Polyendocrine Syndrome Type 3A due to the presence of hypothyroidism and Type 1 diabetes mellitus [1].

The neurological signs, seen in this child namely developmental delay, seizures, dysarthria, ataxia, spasticity along with cerebellar atrophy and calcification seen on neuroimaging can be attributed to long standing untreated hypothyroidism with onset in early childhood. The common neurological manifestations seen in early onset hypothyroidism include mental retardation, impaired motor development with involvement of the pyramidal and extrapyramidal systems, cerebellar dysfunction, strabismus, sensorineural hearing loss and intracranial calcification. There have been reports of children with hypothyroidism presenting with intellectual impairment, spasticity and deformity of lower limbs mimicking cerebral palsy with spastic diplegia (2). The severity of these symptoms has been linked to delay in diagnosis and treatment of hypothyroidism especially in the early childhood or post -natal period. These manifestations do not show an improvement even after initiation of treatment although progression is halted. Hypothyroidism is known to cause calcification in the basal ganglia, subcortical areas and rarely in the cerebellar folia probably due to metabolic derangements [3]. The differential diagnosis for this pattern of calcification includes hypoparathyroidism, hypothyroidism, pseudo-hypoparathyroidism and idiopathic disorders such as Fahr disease. Low levels of thyroid hormones may contribute to epileptogenesis by mitochondrial dysfunction, oxidative stress and due to their role in the development and function of GABAergic neurons [4].

Hypothyroidism being a treatable condition should be ruled out in any child presenting with symptoms like developmental delay, seizures, spasticity, cerebellar dysfunction with neuroimaging findings such as intracranial calcification and cerebellar atrophy.



Non-contrast CT scan of the brain showing bilateral symmetrical calcific foci in the cerebellum.

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