

with desaturation/bradycardia, majority requiring stimulation. There was no identifiable cause for apnea in this neonate despite extensive work-up (septic work-up/biochemistry/echocardiography/CSF analysis/neuroimaging/EEG/gastroesophageal reflux evaluation/upper airway study/ metabolic screening). The neonate was then labelled as having primary/idiopathic apnea, and was started on trial of caffeine therapy after discussion with parents. Caffeine citrate (20mg/kg) was injected intravenously, followed by 10 mg/kg every 24 hourly. There was noticeable improvement in symptoms, and the neonate was weaned off from CPAP support after 3 days; he was discharged at 3 weeks of postnatal life on oral caffeine. Post-discharge home monitoring with pulse-oximeter recorded no apneas. Follow-up of the infant showed lag in weight velocity which caught-up after stopping caffeine at four month of age. Developmental milestones were appropriate for age.

Respiratory pauses of >20 seconds or if associated with bradycardia and cyanosis are labelled as apneas [1]. Apnea is a grave sign in term neonates and could result from sepsis, meningitis or severe brainstem dysfunction in hypoxic neonates. A term neonate with temporal lobe hemorrhage can also present with apneic seizures [2,3].

The neonate in our care was well looking with no features of encephalopathy. Caffeine therapy is extensively used in preterm neonates with apnea of prematurity [1]; however, use of this drug in term neonates is not well known. Its use is described for post-extubation management, and also for bronchiolitis related apnea [4]. We presume that the neonate had primary/idiopathic central apnea requiring intervention that gradually resolved over a period.

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REFERENCES

1. Jing Z, Fernando G, Dezhi M. Apnea of prematurity: From cause to treatment. *Eur J Pediatr.* 2011;170:1097-105.
2. Jeffrey JT, Howard PG. Temporal lobe hemorrhage in the full-term neonate presenting as apneic seizures. *J Perinatol.* 2004;24:726-9.
3. Sirsi DI, Nadiminti L, Packard MA, Engel M, Solomon GE. Apneic seizures: A sign of temporal lobe hemorrhage in full-term neonates. *Pediatr Neurol.* 2007;37:366-70.
4. Cesar K, Iolster T, White D, Latifi S. Caffeine as treatment for bronchiolitis related apnoea. *J Pediatr Child Health.* 2012;48:619.

Grave's Disease Following Aplastic Anemia: Predisposition or Coincidence?

A 3-year-old boy, whose mother had Grave's disease for 9 years, was diagnosed with severe aplastic anaemia in October, 2010. Bone marrow aspiration showed hypoplasia. and bone marrow cytogenetic studies were normal. He was diagnosed to be having severe aplastic anemia and was treated with immunosuppressive therapy of anti-thymocyte globulin at 5 mg/kg/d intravenously for 5 days, prednisone 1 mg/kg/d orally for 1 month and cyclosporine A 3 mg/kg/d orally. Because of neutropenia, recombinant Human granulocyte colony-stimulating factor therapy was initiated. Erythrocyte and thrombocyte infusions were also given moderately because of low blood counts. Four months after immunosuppressive therapy, cyclosporin was continued orally. The blood work-up was normal after 6 months of immunosuppressive therapy.

On follow-up, the parents informed us that the patient had a voracious appetite but had poor weight-gain, and palpitations and excessive sweating. Grave's disease was confirmed by a low TSH, elevated total thyroxine (T4), triiodothyronine (T3), free triiodothyronine (FT3) and free thyroxine (FT4). Thyroid-associated antibodies TRAB(+), ATG(+) and ANTI-TPO(-) were present. Thyroid ultrasound showed bilateral diffuse thyroid lesions. The patient was put on 0.5 mg/kg/d of prednisone and methimazole, which successfully improved the thyroid function later on.

Severe aplastic anemia has now been identified as a kind of bone marrow failure caused by T lymphocyte hyper-function, which induces the apoptosis of hematopoietic cells by excessive secretion of Th1 lymphokines such as IL-2 and interferon-gamma (IFN- γ) [1]. Grave's disease involving the thyroid gland is typically characterized by the presence of circulating auto-antibodies that bind to and stimulate the thyroid hormone receptor, resulting in hyperthyroidism and goiter. It is postulated that the failure of T-suppressor cells allows expression of T-helper cells, sensitized to the TSH antigen, which interact with B cells. These cells differentiate into plasma cells, which produce thyrotropin receptor-

stimulating antibody. Thus, both aplastic anemia and Grave's disease appear to have altered T-cell function.

A previous report details the appearance of Grave's disease after cyclosporine was discontinued for several months. As GD is also considered an autoimmune disorder with abnormal T lymphocytes, it is interesting to speculate that cyclosporine CSA was keeping the disease under control by immune modulation. Grave's disease is not commonly seen in pediatric patients, less so in boys, and the association with severe aplastic anemia in a child has not been frequently described. Our patient's mother had Grave's diseases for 9 years and was put on propylthiouracil treatment, but neonatal Grave's disease is rare, and it is probable that Grave's disease may be hereditary [3].

We propose that both diseases might be related in the autoimmune pathology under certain genetic backgrounds, which needs to be further studied.

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REFERENCES

1. Young NS, Scheinberg P, Calado RT. Aplastic anemia. *Curr Opin Hematol.* 2008; 15:162-8.
2. Kumar M, Goldman J. Severe aplastic anaemia and Grave's disease in a paediatric patient. *Br J Haematol.* 2002;118:327-9.
3. Levy-Shraga Y, Tamir-Hostovsky L, Boyko V, Lerner-Geva L, Pinhas-Hamiel O. Follow up of newborns of mothers with Graves' disease. *Thyroid.* 2014; 24:1032-9.

Can Rashtriya Bal Swasthya Karyakram Give Impetus and Direction to Japanese Encephalitis Vaccination Program?

Japanese encephalitis (JE) is endemic in almost all States of India except Jammu and Kashmir, Himachal Pradesh and Uttarakhand [1]. The States with highest number of encephalitis cases include Assam, Bihar, Tamil Nadu, Uttarpradesh and West Bengal [2]. Every year JE accounts for 10000 to 15000 deaths, and neurological sequelae in an equal proportion [2]. As there is no known specific treatment, prevention is the key to overcome mortality and severe neurological disability that is associated with this infection.

A vaccination program using cell culture derived live attenuated strain (SA 14-14-2) is in place in selected districts of India as a pilot project to prevent this disease. The Indian Academy of Pediatrics, Committee on Immunization recommends one dose of vaccine to be administered to all infants in endemic areas along with measles vaccine with catch-up vaccination administered ahead of anticipated outbreaks in campaign mode [3]. It also recommends the vaccine for travellers to India who intend to stay for longer than four weeks in the endemic districts. The recent launch of the indigenously developed JE vaccine, using an Indian strain of the virus, as a trilateral venture between National Institute of

Virology, Indian Council of Medical Research and Bharat Biotech, is a shot in the arm for control and prevention of JE in India. However, JE vaccination program should be further strengthened to provide nationwide coverage to prevent neurological disability. Nationwide JE vaccination can be implemented through Rashtriya Bal Swasthya Karyakram (RBSK) [4], a flagship program of the Union Health Ministry, to combat disability, which is one of the four health conditions targeted under the RBSK scheme.

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REFERENCES

1. Kakoti G, Dutta P, Ram Das B, Borah J, Mahanta J. Clinical profile and outcome of Japanese encephalitis in children admitted with acute encephalitis syndrome. *Biomed Res Int.* 2013;2013:152656.
2. Potula R, Badrinath S. Japanese encephalitis in and around Pondicherry, South India: A clinical appraisal and prognostic indicators of the outcome. *J Trop Pediatr.* 2003;49:48-53.
3. Vashishtha VM, Choudhury P, Kalra A, Bose A, Thacker N, Yewale VN, *et al.* Indian Academy of Pediatrics (IAP) Recommended Immunization Schedule for Children Aged 0 through 18 years – India, 2014 and Updates on Immunization. *Indian Pediatr.* 2014;51:785-800.
4. Rashtriya Bal Swasthya Karyakram. Available from: <http://nrhm.gov.in/nrhm-components/rmch-a/child-health-immunization/rashtriya-bal-swasthya-karyakram-rbsk/background.html>. Accessed January 14, 2015.