



FIG. 1 ECG at presentation indicative of paroxysmal supraventricular tachycardia.

inversion and high serum CK level. Blood and peripheral culture(s) together with serology, were negative. There was no histological evidence of inflammation on biopsy. The patient was gradually weaned off inotropic support and mechanical ventilation, while antiarrhythmic medication with amiodarone with digoxin and propranolol was initiated due to rhythm disturbances. Ventricular function improved gradually and myocardial biopsy after cardiac catheterization revealed dilated cardiomyopathy, without histological evidence of inflammation. At one year follow-up the patient remains asymptomatic with normal left ventricular size on echocardiography and a LVEF of 67%, confirming the diagnosis of tachycardia induced cardiomyopathy.

In children, SVT is the most common tachyarrhythmia. When SVT is incessant, it can lead to tachycardia induced cardiomyopathy which presents as heart failure [1]. The condition is characterized by significant cardiac enlargement, reduced ventricular wall thickness, and impaired ventricular contraction that resembles to dilated cardiomyopathy but are after control of tachyarrhythmia and management of heart failure [2,3]. A high index of suspicion is mandatory for the diagnosis of tachycardia induced cardiomyopathy. On ECG, the arrhythmia becomes evident. Chest X-ray is also helpful but it is the echocardiogram that reveals LV and left atrial (LA) dilatation with reduced LVEF. Differential

diagnosis includes other causes of reversible cardiac dysfunction such as coronary artery disease hypertension, myocarditis, alcohol, Takotsubo–stress cardiomyopathy, and sepsis. LV dimensions can be used to differentiate dilated cardiomyopathy accompanied by supraventricular tachycardia. Restoration of a normal heart rate improves LV systolic function and reverses clinical manifestations of heart failure in patients with tachycardia induced cardiomyopathy [4].

This is a rare and potentially treatable cause of acute heart failure. A high index of clinical suspicion is mandatory for prompt diagnosis and immediate initiation of treatment.

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Maria Kourti and Maria Sdougka
*Pediatric Intensive Care Unit,
 Hippokraton General Hospital,
 Greece.
 makourti@med.auth.gr*

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Assessment of Iodine in Salt Samples at Retail Trader and Household Levels

Chudasama, *et al.* [1] have mentioned the taluka specific assessment of iodine in salt samples by spot kit at retail trader level. Have the authors combined 840 samples collected from household with those obtained from the

retail shop? If yes, how can the household sample be labelled as the retail trader level sample as method of storage can affect content of iodine. Moreover, the total number of salt samples tested comes to 672, while iodization of salt >15ppm has been mentioned as 775.

In a survey conducted at GMCH, Chandigarh, 1849 school children (6-12years) were studied from 10 schools. Every child was asked to bring the household salt sample. All the samples had > 5 ppm iodine level and 71.8% of salt sample had adequate iodine content of >15

ppm(unpublished data). Patro, *et al.* [2] have also shown 64.2 % household samples and 70.9% at the retail shops ($n=55$) in Jharkhand as optimally iodized samples.

**JASBINDER KAUR AND
SEEMA GUPTA**

*Department of Biochemistry,
Govt. Medical College and Hospital,
Chandigarh, India.
jasbinderkaur@yahoo.co.in*

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Migraine Variant

I read with interest the case reported by Chakravarty and Mukherjee labeled as a migraine variant [1]. They describe a 4 year old boy with delayed expressive language development and episodic focal motor and language deficits lasting 10 minutes to 3 days. Many inborn errors of metabolism can have such a presentation in childhood even in the absence of overt changes on brain magnetic resonance (MR) imaging including organic acidemias (methylmalonic, propionic and isovaleric), maple syrup urine disease, ornithine transcarbamylase (OTC) deficiency, and, hydroxyl-methyl-glutaryl CoA lyase deficiency [2]. Particularly in boys, OTC deficiency is an important consideration. Lack of appropriate metabolic investigations to screen for these disorders and MR spectroscopy, together with absence of headache in the child, makes the diagnosis doubtful, at best.

RAVINDRA ARYA

*Division of Neurology, Cincinnati Children's Hospital
Medical Center, Cincinnati, Ohio
Ravindra.Arya@cchmc.org*

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REPLY

The child is under review for several months now and is doing well. His speech has also improved. There has been no further episode of any focal neurological problem. These would exclude any underlying metabolic disorder.

AMBAR CHAKRAVARTY
saschakra@yahoo.com

Routine Immunization: Campaign or Routine?

Goel, *et al.* provide an information report on effectiveness of a campaign to strengthen routine immunization in Bihar [1]. The exercise involved massive organizational inputs in infrastructure, management and manpower. *Anganwadi* and ANM workers, ASHAs and vaccinators were mobilized and budgetary support and political commitment were forthcoming.

The chief reason for poor coverage of routine (and other) immunizations is ignorance of the parents and the family of the benefits of immunizations. If that were clearly understood there would be a demand for vaccinations. Literacy rates are high in Indian states with

impressive immunization coverage. Whereas campaigns are useful to achieve short term benefits, they are very difficult to sustain. Communities need to be educated and informed of the value of immunizations and other aspects of health care, with the help of *Anganwadi* and ASHA workers, school teachers and panchayat bodies and others. Minor reactions following DPT vaccine administration, often responsible for dropouts, should be properly managed and the family reassured. Supply of vaccines, maintenance of cold chain and safe injection practices must be ensured. Completeness of immunizations can be monitored with the help of a health card for every child, which would have records of vaccines administered and other vital health parameters [2]. Local officials should be charged with the responsibility of immunization and other health interventions and made accountable.