Authors' Reply: Primary Neonatal Psoas Abscess

This correspondence is in relation to the dilemma regarding primary and secondary psoas abscess as pointed recently [1].

There remains no doubt that primary abscesses are due to a hematogenous spread due to bacteremia and secondary abscess occurs as a result of direct spread of infection from an adjacent structure [2,3]. A case of primary psoas abscess with spondylodiscitis was published earlier by us [4]. Hernández-Ros, *et al*. commented that all authors must use the recent classification criteria and accordingly, that patient should have been diagnosed as secondary rather than primary psoas abscess.

We disagree because our patient, a 26 day-old-neonate, presented with clinical features of septicemia and it was clearly mentioned that the isolates of methicillin resistant staphylococcus aureus (MRSA) was detected in the blood culture. Hematogenous spread of infection following the bacteremia resulted in the psoas abscess. Discitis with vertebral body destruction following primary abscess was a unique presentation in this age group. Referring to the new classification criteria, as also pointed out by Hernández-Ros *et al*., the hematogenous spread of the infection in our case clearly established the diagnosis of primary psoas abscess beyond any doubt. Our diagnosis of primary was not on the basis of organism. It was not the local spread of infection from nearby structure; hence it cannot be labeled as secondary psoas abscess.

RAKESH MONDAL AND *SUMANTRA SARKAR
Department of Pediatrics, NBMCH Darjeeling,
**Department of Pediatrics, IPGMER Kolkata, India.*
ivanrakesh2001@gmail.com

REFERENCES

1. Hernández-Ros R, Hernández-Belmonte A. Psoas Abscess: Primary or Secondary? Indian Pediatr. 2013;50:345-6.
2. Dos Santos VM, Silva Leao CE, Borges Santos FH, Fastudo CA, Machado Lima RL. Iliopsoas abscess and spondylodiscitis by Staphylococcus aureus: successful clinical treatment. Infez Med. 2011;2:120-4.
3. Navarro López V, Ramos JM, Meseguer V, Pérez Arellano JL, Serrano R, García Ordóñez MA, *et al*. Microbiology and outcome of iliopsoas abscess in 124 patients. Medicine (Baltimore). 2009;88:120-30.
4. Mondal R, Sarkar S. Spondylodiscitis with primary psoas abscess in a neonate. Indian Pediatr. 2012;49:681.

Disulfiram Poisoning Causing Acute Encephalopathy

Disulfiram is used for the treatment of alcoholism, exerts its action when taken concomitantly with alcohol. The typical reaction is self-limited, with headache, flushing, dizziness, nausea, blurred vision, tremor and dyspnea [1]. When not associated with alcohol ingestion, its effects are scarce at usual daily doses. Acute intoxication at doses higher than 500 mg/d, may result in severe collateral effects, and can be lethal at doses between 10-30g/d [2]. The presence of this drug in the home makes it a potential agent for accidental poisoning. We report an unusual case of disulfiram poisoning.

A two-year-old healthy boy presented with lethargy, drowsiness and unresponsiveness of one day duration. No history of fever, trauma and parents denied any intoxication. His development was appropriate for age. There was no significant family history. Later the subject developed convulsions, persistent encephalopathy and dystonia. On examination, his anthropometry was normal. He had acidotic breathing with hypotension (blood pressure-60/40mm). His Glasgow coma scale was 6/15. The pupils and fudus were normal. Dystonia was present. There were no signs of meningeal irritations. Differential diagnosis of metabolic encephalopathy, encephalitis and intoxication were considered.

Hematological and biochemical workup including serum electrolytes, serum lactate and ammonia levels were normal. Arterial blood gas was suggestive of severe metabolic acidosis (pH-7.02, PO_2-75, PCO_2-12, HCO_3-6 BE- -16). Cerebrospinal fluid examination was normal. MRI of brain showed swollen and symmetrical hyperintense signal changes involving the globus pallidus and substantia nigra. Additionally both the structures are showing restricted diffusion on DWI images (***Fig.* 1**). Tandem mass spectroscopy (TMS) and gas chromatography and mass spectroscopy (GC/MS) of urine were found to be normal. After 2 weeks the boy's father reported that 8-10 of the disulfiram tablets, that the

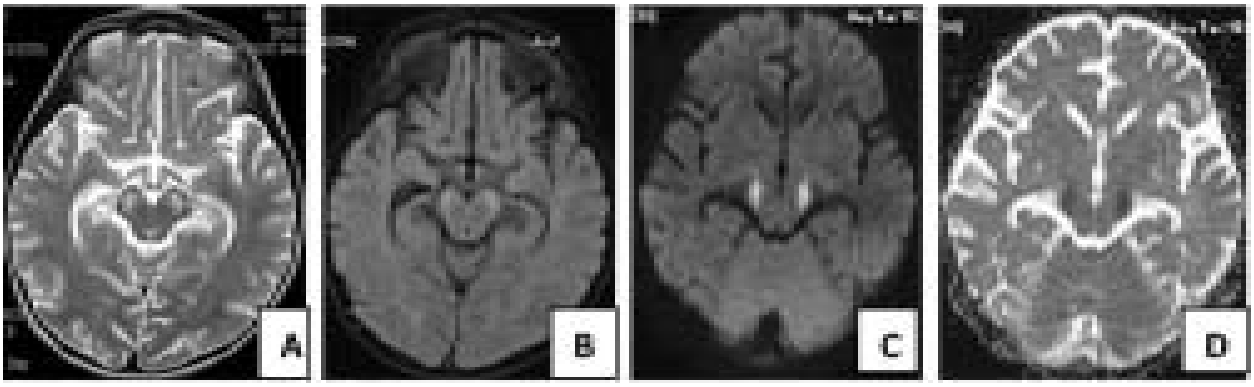

FIG. 1 A,B,C and *D represents axial T2WI, FLAIR and axial DWI and ADC maps taken at the level of crus cerebri respectively. They show swollen and symmetrical hyperintense signal changes involving the globus pallidus and substantia nigra. Additionally restricted diffusion is seen on DWI images.*

grandfather was on, were missing. The clinical findings, acidosis and MRI findings were consistent with disulfiram poisoning. Treatment with megavitamins was started but child had persistent extra-pyramidal symptoms with minimal improvement in sensorium after one month of consumption of disulfiram.

The diagnosis of disulfiram poisoning is difficult as it is rapidly cleared from the circulation; its metabolites can be measured only by highly specialized laboratory techniques, which are not readily available. There is no specific antidote for disulfiram toxicity.

The exact mechanism of disulfiram mediated encephalopathy is not known. However, disulfiram metabolites diethyldithiocarbamate and carbon disulfide have been shown to inhibit the activity of the enzyme dopamine-α-hydroxylase leading to the accumulation of dopamine, producing a relative deficiency of adrenaline and noradrenaline in the area of the basal ganglia. Dopamine-mediated cellular injury may be related to its ability to induce excitatoxic effects of glutamate; - calcium mediated cell death, and impairs the cellular ability to eliminate free oxygen radicals [3].

The possible differential diagnosis of Leigh's Disease, Organic acidurias, and carbon monoxide poisoning were considered but ruled out on history, clinical features and MRI findings. We ruled out extrapontine myelinolysis as his serum sodium levels were within normal.

Disulfiram poisoning should be suspected in any child presenting with unknown encephalopathy with convulsions, extra-pyramidal symptoms with basal ganglia signal changes in MRI of brain in a previously normal child.

***Acknowledgments*:** Dr Kavitha V and Dr Arvind Kasaragod for their help in management of the case.

VYKUNTARAJU KN and *ARVINDA HANUMANTHAPURA RAMALINGAIAH
Division of Pediatric Neurology,
Indira Gandhi Institute of Child Health, Bangalore and
**Department of Neuro-radiology,*
National Institute of Mental Health and Neurosciences (NIMHANS),
Bangalore, Karnataka, India.
drknvraju@yahoo.co.in

REFERENCES

1. Wright C, Moore RD. Disulfiram treatment of alcoholism. Am J Med. 1990;88:647-55.
2. Petersen EN. The pharmacology and toxicology of disulfiram and its metabolites. Acta Psychiatr Scand Suppl. 1992;369:7-13.
3. Rothmann S, Olney J. Glutamate and the pathophysiology of hypoxicischemia damage. Ann Neurol. 1986;19: 105-111.