

and promote normal growth. Vomiting and failure to thrive were the most common clinical presentation seen in both genders in our series, similar to an earlier observation by Begum, *et al.* [4].

To date there are approximately 100 different mutations reported in *CYP 21* gene including deletions, point mutations and insertions. Severe mutations are associated with classical CAH whereas milder mutations are associated with non-classical CAH [5,6]. Marumudi, *et al.* from New Delhi reported Intron 2 mutation as the most common mutation in patients with CAH [7]. Mathur, *et al.* [6] from New Delhi reported Ile173Asn followed by intron 2 splice and Gln 319 stop mutations in children with classical CAH. In our series, 8 bp deletion in exon 3 of the *CYP21A2* gene was the most common (66.7%) followed by 12 g mutation in Intron 2 of the *CYP21A2* gene [IVS2-13A/C>G]. As complete sequencing of *CYP21A2* gene is expensive and is available only in select laboratories, knowledge of common mutations prevalent in our population helps us to make a reliable pre-, peri- and post-natal diagnosis, and also to offer genetic counseling to the affected families.

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REFERENCES

1. Huynh T, McGown I, Cowley D, Nyunt O, Leong GM, Harris M, *et al.* The clinical and biochemical spectrum of congenital adrenal hyperplasia secondary to 21-hydroxylase deficiency. *Clin Biochem Rev.* 2009;30:75-86.
2. Dolzan V, Stopar-Obreza M, Zerjav-Tansek M, Breskvar K, Krzisnik C, Battelino T. Mutational spectrum of congenital adrenal hyperplasia in Slovenian patients: A novel Ala15Thr mutation and Pro30Leu within a larger gene conversion associated with a severe form of the disease. *Eur J Endocrinol.* 2003;149:137-44.
3. Bajpai A, Kabra M, Menon PS. 21-Hydroxylase deficiency: Clinical features, laboratory profile and pointers to diagnosis in Indian children. *Indian Pediatr.* 2004;41:1226-32.
4. Begum JA, Sarker AK, Hoque M, Mamun MAA, Mobarak MR, Biswas R, *et al.* Clinical profile of congenital adrenal hyperplasia(CAH): A treatable disease. *Dhaka Shishu (Children) Hospital Journal.* 2010;26:108-12.
5. Forest MG. Recent advances in the diagnosis and management of congenital adrenal hyperplasia due to 21-hydroxylase deficiency. *Hum Reprod Update.* 2004; 10:469-85.
6. Marumudi E, Sharma A, Kulshreshtha B, Khadgawat R, Khurana ML, Ammini AC. Molecular genetic analysis of *CYP21A2* gene in patients with congenital adrenal hyperplasia. *Indian J Endocrinol Metab.* 2012;16:384-8.
7. Mathur R, Menon PS, Kabra M, Goyal RK, Verma IC. Molecular characterization of mutations in Indian children with congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency. *J Pediatr Endocrinol Metab.* 2001;14:27-35.

Pediatric Melioidosis in Southern India

Melioidosis in children is increasingly detected from the coastal region of Southern India during monsoon. We present 11 cases of melioidosis, ranging from localized to disseminated, treated successfully, barring one death. It calls for awareness and upgrading laboratory facilities for better diagnosis and management of pediatric melioidosis.

Keywords: *Burkholderia pseudomallei*, Child, Lymphadenitis.

Melioidosis, a disease caused by the soil-dwelling bacterium *Burkholderia pseudomallei*, has varied clinical spectrum ranging from mild localized illness to fulminating sepsis. Southern part of India is apparently a new 'hot spot' in the global map of melioidosis [1,2]. Childhood infections are increasingly being recognized, and are more localized affecting immunocompetant

population [3,4]. This case series highlights the occurrence and presentation of the culture-confirmed cases of melioidosis among children, diagnosed at our institute between January 2007 and June 2014.

Pediatric melioidosis accounted for 8% of 140 cases of melioidosis diagnosed during this period. The median age was 7.5 years (range 3-18 y). Fever was the commonest presentation (100%) with a median duration of 10 days (range 2-90 d). Ten children presented with acute disease (≤ 2 mo), while one child had fever for three months. Melioidosis was restricted to head and neck region in five children (two submandibular abscesses, two suppurative cervical lymphadenitis and one suppurative parotitis), whereas six had disseminated disease. Hepatomegaly and splenomegaly were observed in three and two cases, respectively. Two children had diabetes mellitus, both of whom presented with severe systemic illness, but recovered. One child, who presented with septic shock, encephalopathy and acute respiratory

distress syndrome (ARDS), died before blood culture report was available. All except one child presented during monsoon season (May to October). Nine children were from coastal regions, and two from around Western Ghats. All children had history of contact with soil and water while playing outdoor. Cultures (BacT/ALERT system) showed 100% susceptibility (Kirby Bauer disc diffusion method) to amoxicillin-clavulanic acid, ceftazidime, meropenem, sulphamethoxazole-trimethoprim (TMP/SMX) and doxycycline. Six children were treated with amoxicillin-clavulanic acid, alone or in combination with ceftazidime or TMP/SMX, while four were treated with ceftazidime or meropenem. Hospital stay ranged from 3 to 14 days. Ten children showed clinical improvement by the time of discharge; two completed 3 months of maintenance therapy with TMP/SMX, and eight were lost to follow-up.

Melioidosis still remains an underdiagnosed entity in India, especially in children [4,5]. Acute and localized clinical presentations involving head and neck as suppurative lymphadenitis is consistent with other reports from South East Asian countries [2,6-10]; in Australia, suppurative parotitis is more common [1]. Severe systemic melioidosis in adults or localized melioidosis in children is treated with intravenous ceftazidime for 10-14 days followed by oral therapy with TMP/SMX alone or in combination with doxycycline (only in children >8 years) for 20 weeks. Mild localized infection may be treated with oral TMP/SMX for shorter duration of 4-5 weeks. Localized melioidosis in children responds well to drainage of pus supporting better recovery with short course antibiotic therapy [2,8]. Majority of the children in our series belonged to rural and semi-urban settings, and presented with acute disease during rainy season which suggests that waterlogged soil possibly increases chance of acquiring this infection. However, the ecology of soil and environmental distribution of *B. pseudomallei* is yet to be studied in India.

We conclude that melioidosis should be an important differential diagnosis in suppurative lesions of head and neck, and soft tissue infections in children. Active

microbiological search would enhance the accuracy of presumptive diagnosis and widen the knowledge on this emerging bacterial agent, especially in coastal areas.

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REFERENCES

1. Currie BJ, Ward L, Cheng AC. The epidemiology and clinical spectrum of melioidosis: 540 cases from the 20 year Darwin prospective study. *PLoS Negl Trop Dis.* 2010;4:e900.
2. Lumbiganon P, Chotechuangnirun N, Kosalaraksa P, Teeratakulpisarn J. Localized melioidosis in children in Thailand: Treatment and long-term outcome. *J Trop Pediatr.* 2011;57:185-91.
3. Edmund KM, Currie BJ, Brewster D, Kilbum C. Paediatric melioidosis in tropical Australia. *Pediatr Infect Dis J.* 1998;17:77-80.
4. Raghavan KR, Shenoi RP, Zaer F, Aiyer R, Ramamoorthy P, Mehta MN. Melioidosis in India. *Indian Pediatr.* 1991;28:184-8.
5. Shivbalan S, Reddy N, Tiru V, Thomas K. Systemic melioidosis presenting as suppurative parotitis. *Indian Pediatr.* 2010;47:799-801.
6. How HS, Ng KH, Yeo HB, Tee HP, Shah A. Pediatric melioidosis in Pahang, Malaysia. *J Microbiol Immunol Infect.* 2005;38:314-9.
7. Lumbiganon P, Viengnondha S. Clinical manifestations of melioidosis in children. *Pediatr Infect Dis J.* 1995;14:136-40.
8. Lumbiganon P, Chotechuangnirun N, Kosalaraksa P. Clinical experience with treatment of melioidosis in children. *Pediatr Infect Dis J.* 2004;23:1165-6.
9. Stoesser N, Pocock J, Moore CE, Soeng S, Chhat HP, Sar P, *et al.* Pediatric suppurative parotitis in Cambodia between 2007 and 2011. *Pediatr Infect Dis J.* 2012;31:865-8.
10. Dance DA, Davis TM, Wattanagoon Y, Chaowagul W, Saiphan P, Looareesuwan S, *et al.* Acute suppurative parotitis caused by *Pseudomonas pseudomallei* in children. *J Infect Dis.* 1989;159:654-60.