Outbreaks of Rubella Indicate Epidemiological Shift in Age

The report on outbreak of rubella in Chandigarh by Madhanraj, *et al*. [1], exposes the inadequate disease surveillance mechanism for most communicable diseases in our country. Such outbreaks elicit knee-jerk reactions which are soon forgotten over a period of time. Diseases like rubella which are mild and self-limiting are likely to be missed in day-to-day practice. The only serious consequence is in the first trimester of pregnancy when the infection can lead to abortions and Congenital Rubella Syndrome (CRS). Because of this serious complication, we need to beef up the surveillance for rubella in the community.

Earlier, we published about an outbreak of rubella in a population of adolescents in a military training centre [2]. Out of the 163 suspected cases, we confirmed rubella in 117 (72%) by presence of anti-rubella IgM antibodies. This outbreak, occurring more than a decade ago, indicates that there is already an epidemiological shift in susceptible age for rubella towards young adolescents and young adults. This shift in age may be due to mass use of Measles, Mumps and Rubella (MMR) vaccination during infancy without any subsequent doses in the older age groups that makes women of child bearing age susceptible to rubella during pregnancy which can lead to the dreaded CRS.

All outbreaks of rubella and all cases of CRS should be investigated and reported in addition to building and maintaining a robust surveillance system to provide essential inputs for planning and implementing prevention programs [3]. Two approaches are recommended to prevent the occurrence of CRS [4]. The first is prevention of CRS only by immunization of adolescent girls or women of childbearing age. The other approach is elimination of rubella as well as CRS through universal immunization of infants and ensuring immunity in women of child bearing age. We need a well thought-out strategy commensurate to our resources. As an immediate measure we should aggressively go for control of CRS by targeting women of child bearing age. We should also consider mandatory rubella vaccination for all medical and nursing staff to limit nosocomial spread of rubella to pregnant women in the health care setting.

AMITAV BANERJEE
Department of Community Medicine,
Dr DY Patil Medical College, Hospital and Research Centre,
Pune, Maharashtra, India.
amitavb@gmail.com

REFERENCES

1. Madhanraj K, Singh N, Gupta M, Singh MP, Ratho RK. An outbreak of rubella in Chandigarh, India. Indian Pediatr. 2014;51:897-9.
2. Banerjee A, Sahni AK, Gupta RM, Grewal VS, Singh Z. Outbreak of rubella among cadets in an academy. Medical Journal Armed Forces India (MJAFI). 2007;63:2:141-3.
3. Sachdeva S, Gupta P. Time to target rubella elimination. Indian Pediatr. 2014;51:872-4.
4. Duclos P, Reef S. Rubella. *In*: Control of Communicable Diseases Manual. Heymann DL (ed). 19th edition. Washington; American Public Health Association. 2008. p. 529-34.

Klebsiella Brain Abscess in an Infant With Hereditary Spherocytosis

Streptococcus is the principle causative organism of brain abscess but widespread use of antibiotics, immunization and frequent neurosurgical intervention seems to have changed the epidemiology and clinical spectrum of this entity in recent years [1,2].

A 5½-month-old boy presented to neurosurgical emergency of our hospital with complaints of progressively increasing head size. He had severe anemia for which he was referred to pediatric emergency. There was no fever, seizures or focal neurological deficit. This child had jaundice within 24 hours of life, and received blood transfusion for severe anemia on day 8 of life. He later developed fever and abscess at the site of intravenous cannulation for which he received oral drugs from a local practitioner. At 4 months of age, child developed severe pallor and parents noticed an increased head size for which he was referred to our hospital.

Blood investigations of the child suggested hereditary spherocytosis. Computed tomography (CT) of head showed multiple brain abscesses. No primary or secondary immunodeficiencies were identified. Serum

levels of immunoglobulins (IgA, IgG and IgM) were normal; mother's HIV ELISA was negative.

Patient was started on empirical antibiotics (Ceftriaxone, vancomycin and metrogyl) and surgical drainage was performed. No organism was identifiable in pus culture and gram stain but CSF culture grew *Klebsiella pneumoniae* sensitive to Gentamicin, Ciprofloxacin, Cotrimoxazole, Amikacin, Ceftriaxone, Ceftazidime and Piperacillin–tazobactum. Drugs were changed according to the sensitivity pattern to Ciprofloxacin and Amikacin and continued for 6 weeks. Non-contrast CT after completion of treatment showed significant decrease in size of the abscess.

The spectra of clinical presentations of brain abscess range from insidious to fulminant. In a previous study, the classic triad of headache, fever, and focal neurologic deficit was present in fewer than half of patients with brain abscess [3]. Our patient presented as hydrocephalus without any other constitutional symptom or features of raised intracranial tension. Klebsiella is a rare cause of cerebral abscess in infants. In our patient, the cause of the abscess is likely to be hematological spread from skin abscess due to intravenous cannulation at day 8-9 of life.

Unlike other hemolytic anemias like sickle cell anemia and thalassemia, cerebrovascular events and their complications are rare with hereditary spherocytosis [4]. An increased propensity of thrombosis in these patients is due to increased aggregability and reduced deformability of red blood cells, and increased viscosity of blood. An area of infarct thus formed could later on become a site for abscess formation. Apart from that, brain abscess is a known complication in meningitis and septicemia, and association of hereditary spherocytosis could be coincidental.

***DEEPAK SACHAN AND AMLIN SHUKLA**
Department of Pediatrics,
PGIMER,
Dr RML Hospital, New Delhi.
**deepaksachan26@yahoo.com*

REFERENCES

1. Su TM, Lin YC, Lu CH, Chang WN, Liliang PP, Rau CS, *et al*. Streptococcal brain abscess: Analysis of clinical features in 20 patients. Surg Neurol. 2001;56:189-94.
2. De Louvois J, Gortavai P, Hurley R. Bacteriology of abscesses of the central nervous system: A multicentre prospective study. BMJ. 1977; 2:981-4.
3. Tunkel AR, Wispelwey B, Scheld WM. Brain abscess. *In*: Mandell GL, Bennett JE, Dolin R, eds. Mandell, Gouglas, and Bennett's Principle and Practice of Infectious diseases. 5th ed. Vol 1. Philadelphia: Churchill Livingstone; 2000. p. 1016-28.
4. van Hilten JJ, Haan J, Wintzen AR, van de Nes JC, Heuvelmans JH, Aarts PA, *et al*. Cerebral infarction in hereditary spherocytosis. Stroke. 1989;20:1755-6.