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Editorial

Gust and Flare-up- Measles Lymphadenitis

Anubha Bajaj*

Department of Histopathology, Panjab University/A.B. Diagnostics, India

***Corresponding Author:** Anubha Bajaj, Department of Histopathology, Panjab University/A.B. Diagnostics, India.

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Measles emerges as preventable, contagious, acutely febrile, viral illness. Alternatively designated rubeola, measles is associated with global mortality and morbidity. Measles virus, engendering measles, emerges as an enveloped, single-stranded, negative-sense ribonucleic acid (RNA) virus pertaining to genus Morbillivirus, family Paramyxoviridae.

Viral genome encodes 6 structural proteins and 2 non-structural proteins V and C. Structural proteins configure as nucleoprotein, phosphoprotein, matrix, fusion, haemagglutinin (HA) and large protein. HA protein ensures viral adhesion to host cell.

Administered live, attenuated measles vaccine may induce regional lymphadenopathy. Measles associated lymphopenia may arise due to apoptosis of uninfected lymphocytes.

Fatal instances of measles may concur with malnutrition and engenders depletion of T cell zones.

Measles virus induces significant immunosuppression of extended duration. Consequently, enhanced susceptibility towards secondary bacterial and various infections is observed.

Of obscure mechanism, measles infection is posited to induce proliferation of measles-specific lymphocytes which replace preceding population of memory cells, thereby engendering 'immune amnesia'. Thus, enhanced susceptibility of host towards secondary infection with consequent emergence of morbidity and mortality associated with measles is encountered [1,2].

Neutralizing IgG antibodies against hemagglutinin may incur lifelong, persistent immunity which impedes host cell receptors from binding to the virus. Viral inhalation from exposed droplets may preliminarily infect the respiratory tract with activation of inflammatory cells as lymphocytes, dendritic cells and alveolar macrophages [1,2].

The virus disseminates into adjacent lymphoid tissue and bloodstream with consequent occurrence of viremia and viral spread into distant organs [1,2].

Virus residing into dendritic cells and lymphocytes transmits into respiratory tract epithelial cells which may be exfoliated and extruded as respiratory droplets during manoeuvers as coughing or sneezing. Thus, infection of various humans and perpetuation of infective cycle may occur [1,2].

Prodromal phase of measles virus decimates host immunity by suppressing production of interferon through non-structural proteins V and C. Enhancing viral replication activates humoral and cellular immunological responses [1,2].

Preliminary humoral response is comprised of production of immunoglobulin (Ig)M antibody as discerned within three days to four days following the emergence of rash and may persist for 6 weeks to 8 weeks.

Subsequently, immunoglobulin (Ig)G antibodies are produced which are principally directed against viral nucleoprotein [2,3].

Cellular immune response appears essential for recovering health, as demonstrated by elevated T helper (Th)1-dependent plasma interferon-gamma levels discerned during acute phase. Subsequently, elevation of Th2-dependent interleukin 4, interleukin 10 and interleukin 13 levels may be enunciated [2,3].

Initial inflammation of respiratory tract may induce clinical symptoms as coryza, conjunctivitis and cough. Emergence of pyrexia concurs with occurrence of viremia. Cutaneous rash ensues following viral dissemination on account of perivascular and lymphocytic inflammatory cell infiltrate [2,3].

Upon microscopy, polykaryocytes are encountered. Germinal centres display a population of Warthin-Finkeldey giant cells imbued with significant, enlarged nuclei. Aforesaid giant cells appear immune reactive to B cell markers [3,4].

Inter-follicular areas are pervaded with T lymphoid cells impregnated with miniature, hyperchromatic nuclei.

Lymph node architecture displays diffuse hyperplasia of immunoblasts confined to paracortical region, as predominantly encountered with viral lymphadenitis [3,4].

Morphological assessment of lymph node characteristically expounds fused lymphocytes commingled with foci of paracortical hyperplasia.

Characteristically, diffuse proliferation of immunoblasts and depletion of miniature lymphocytes induces a mottled or 'moth-eaten' pattern.

Severe reaction to measles vaccine may incur vascular thrombosis and partial haemorrhagic necrosis of regional or inguinal lymph nodes [3,4]. Multinucleated giant cells or Warthin and Finkeldey may appear within prodromal phase of measles. Besides, the giant cells may appear within diverse hyperplastic lymphoid tissues as tonsils, adenoids, lymph nodes, spleen, appendix or thymus.

Giant cells may disappear with elevating antibody titres or in concurrence with cutaneous eruption [3,4].

The polykaryocytes are spherical, lobulated, impregnated with abundant, eosinophilic cytoplasm and four to fifty, centric, darkly stained nuclei disseminated as 'grapelike' clusters, thereby articulating 'mulberry cells'. The giant cells configure enlarged syncytia and display a magnitude of 25 μm to 150 μm. Infrequent, intracytoplasmic cellular inclusions may be encountered within the giant cells or endothelial cells [3,4].

Polykaryocytes morphologically identical to measles associated Warthin-Finkeldey giant cells may be encountered in reactive lymphoid hyperplasia, neoplastic lymphoid lesions, benign lymphadenopathies as pattern A of acute phase of human immunodeficiency virus(HIV) lymphadenitis [6,7].

Lymphocyte-predominant subtype of Hodgkin's lymphoma and well-differentiated or poorly differentiated lymphocytic non-Hodgkin lymphomas may depict a population of polykaryocytes. Alternatively, the cells may be associated with viral lymphadenitis or low grade lymphomas [6,7].

TNM staging of Sinonasal B Cell Lymphoma [5].

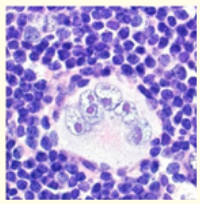

Figure 1: Warthin Finkledey cells displaying abundant, eosinophilic cytoplasm and several fused nuclei surrounded by a population of small, mature lymphoid cells [9].

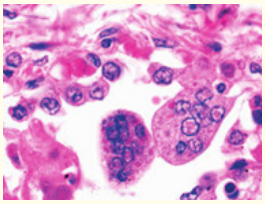

Figure 2: Warthin Finkledey cells enunciating abundant, eosinophilic cytoplasm and several, overlapping nuclei commingled with a population of small, mature lymphocytes [10].

Primary tumour

- T1: Tumour confined to nasal cavity
- T2: Tumour extension into maxillary sinus, anterior ethmoid sinus or hard palate
- T3: Tumour extension into posterior ethmoid sinus, sphenoidal sinus, orbit, superior alveolar bone, cheeks or superior buccinators space
- T4: Tumour incrimination of inferior alveolar bone, inferior buccinators space, infratemporal fossa, nasopharynx or cranial fossa.

Regional lymph nodes

- N0: No regional lymph node involvement
- N1: Unilateral regional lymph node involvement
- N2: Bilateral regional lymph node involvement

Distant Metastasis

- M0: Distant metastasis absent
- M1: Distant metastasis present

Measles requires segregation from conditions as drug eruptions, erythema infectiosum, Kawasaki disease, meningococcemia, Rocky mountain spotted fever, infectious mononucleosis, parvovirus B19 infection, paediatric enteroviral infections, paediatric rubella, paediatric sepsis, paediatric toxic shock syndrome, scarlet fever, multisystem inflammatory syndrome in children, dengue, Zika virus infection or syphilis [7,8].

Prevention of measles is a superior mode of therapy. Measles-mumps-rubella (MMR) vaccine is efficacious in preventing the infection.

Specific antiviral therapy for measles appears absent. Supportive therapy includes control of pyrexia, prevention and correction of dehydration, control of viral infection along with appropriate isolation. Supplementation with vitamin A is beneficial for decimating complications of measles in malnourished children [7,8].

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9. Image 1 Courtesy: Wiley online library.

10. Image 2 Courtesy: Academic oup.