Acute myelitis as presentation of measles, a reemerging disease

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Introduction

Over the last few months Italy has witnessed an outbreak of measles mostly in adolescents and young adults.¹ According to the last weekly report on measles from Italian Ministry of Health, in 2017 4575 cases with 4 deaths have been reported: 88% of cases were unvaccinated, about a third presented complications, the median age was 27 years.² Though vaccination was introduced in national schedule about 20 years ago, after 2007 a decreasing trend in coverage has been observed.³ Measles vaccination is one of the most effective health interventions ever developed. Without the vaccine, 5 million children would die each year from measles—assuming an estimated case-fatality rate of 2%–3%.4 In adults respiratory and neurological complications account as main causes of hospitalization in acute phase.

Discussion and conclusions

Neurological complications of measles are rare but severe and include subacute sclerosing panencephalitis (SSPE), measles body encephalitis (MIBE) inclusion and post-infectious encephalomyelitis (**PIE**).4

SSPE is caused by the persistence of virus in central nervous system even for several years, causing multifocal infection and demyelination. Children initially present decreased school performance and behavioral disorders, often misdiagnosed as psychiatric problems. Subsequently, myoclonic seizures develop with progression until vegetative state. Incidence in the USA is 1 per 8.5 million persons previously affected with measles.4 **MIBE** occurs in immunocompromised patients fews weeks to six months after acute measles. At the beginning mental-status changes and seizures are observed; the disease is lethal within weeks in the 80% of patients.4

Case-report

On January 2017, a 32-year-old Romanian woman permanently resident in Italy presented at emergency department with acute urinary retention and limbs weakness. She was febrile with maculopapular rash at neck and face (more prominent behind the ears). During the previous week she had pharyngitis and fever, treated with clarithromycin, while her 4-years-old daughter had been hospitalized for pneumonia fews weeks before. Neurological examination of the patient was suggestive of acute myelitis, showing normoreflexic tetraparesis with distal weakness, walk only with double support, loss of pain sense at both forearms and hands and below mammillary line.

At admission: blood exams were unremarkable with the exception of a mild increase of GPT/GGT (respectively 3/2 times above the normal range); at spine MRI extensive hyperintensity at T2-weighted sequences was observed at antero-central portion of spinal cord from C4 to conus medullaris, with enhancement at C6-C7 (Fig. A-B-C); cerebro-spinal fluid (CSF) examination revealed 225/mmc white cells (neutrophils/lymphocytes ratio 52/48), 115 mg/dl proteins, normal glucose.

With the suspicion of infective myelitis, empiric therapy with ceftriaxone, azithromycin and acyclovir was started since the admission and then discontinued when CSF culture and serological testing (including Treponema pallidum, HIV, Mycoplasma pneumoniae, Parvovirus B19, Borrelia Burgdorferi, herpetic viruses, toxoplasma, rickettsia and QuantiFERON-TB) resulted all negative. Oligoclonal bands in CSF were absent too.

Due to the coexistence of fever, skin rash, pharingitis and myelitis, serum anti-measles antibodies were tested: on day 2 their results came and were suggestive of acute infection (IgM 1.9, n.v. 0.0-1.1; IgG > 300, n.v. 0-16 UA/ml). Retrospectively, the patient revealed that she and her daughter were both unvaccinated for measles and had recently got in touch with an affected child. Therefore, the case was notified to the

PIE occurs in approximately 0.1% of cases of measles. Higher rates of PIE featured by a worse prognosis occur in adolescents and adults than in school-aged children. PIE begins with the abrupt onset of fever, seizures, altered mental status, and focal neurological signs. Lethality is 25% and 33% of survivors have long-term disability as paraparesis and bladder dysfunction. MRI lesions and severe neurological manifestations of PIE resemble those of acute disseminated encephalomyelitis (ADEM). Thus, PIE is likely related to an autoimmune reaction against myelin basic protein during the stage of recovery from infection, generally within 2 weeks of the exanthema. The virus usually is not found in the central nervous systems. Steroids and immunoglobulin may provide a relatively good outcome, nevertheless therapeutic protocols have not been established.4-5-7

This case outlines that prompt diagnosis of measles and its neurological complications is challenging as more frequent symptoms (fever, pharyngitis, skin rash) may be misjudged expecially in adults.

In Italian adult population an increase of cases with neurological presentations is likely to be observed due to recent measles outbreak caused mostly by opposition to vaccination. This remarks that vaccine remains the most potent tool against measles and its severe complications.₃₋₄

proper authority.

To reduce inflammation at spinal cord i.v. 8 mg dexamethasone was administered since the first day, and then tapered after two weeks when a new spine MRI revealed a definite resolution of spinal cord inflammation (Fig. D-E-F). Brain MRI performed on day 7 was normal.

After two-months rehabilitation the patient could walk independently, after six months intermittent self-catheterization was interrupted due to improvement of bladder function.



References

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Figure

At onset T2-weighted images showed hyperintense signal in the anterior portion of all spinal cord from C4, without edema (B), more pronounced at C6-7 level (A); at this level a mild gadolinium enhancement was observed at T1-weighted images (C).

Resolution of spinal cord inflammation was observed at a new MRI performed on day 10 (D-E-F).





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