CLINICAL OBSERVATION OF THE FAMILY CASE OF GENERALIZED MENINGOCOCCAL INFECTION CAUSED BY MENINGOCOCCAL GROUP W STRAIN (MEN W).

Ivanova M.V. ¹, Skripchenko N.V. ¹, Vilnits A.A. ¹, Sidorenko S.V. ¹, Shtykunova E.V. ²

¹ Federal State-Financed Institution “Pediatric Research and Clinical Center for Infectious Diseases” under the Federal Medical Biological Agency, St. Petersburg, Russia

² Pirogov Russian National Research Medical University, Chair of Pediatric Infections, Moscow, Russia

Invasive Meningococcal Disease (IMD) and especially its generalized forms are very dangerous and risky for its fatal outcomes and life-threatening complications. Serogroup landscape of Meningococcal infection is heterogenic, varies from one territory to another and may change over time. [1, 2] Recent epidemiology and clinical data report an endemic rise in IMD caused by W-135 strain (Niger, Saudi Arabia, Australia, The Netherlands, Canada, etc.). [3, 4]

The epidemiology study performed in Russia in 2010-2015 confirmed predominance of A (30%), B (32%) and C (27%) meningococcal strains, while the share of W, Y, X strains did not exceed 5%. At the same time, meningococcal landscape on territories varied significantly [5]. In 2015-2016 in Moscow there were observed an increase in the number of cases of generalized IMD in kids and adolescents that were caused by W strain [6]. Recent retrospective analysis performed for IMD in St. Petersburg (1995-2016) also discovered significant growth of IMD-W cases [7]. (Fig.1.)

Fig 1. Changes in meningococcal landscape in St. Petersburg region in 1995 – 2016

References

1. WHO. Meningococcal meningitis. Fact sheet №141. URL: <http://www.who.int/mediacentre/factsheets/fs141/en/> (as of 09.06.17); 2.Dwilow R, Fanella S. Invasive meningococcal disease in the 21st Century—an update for the clinician. Curr Neurol Neurosci Rep 2015; 15(3): 3.Stephen I., PeltonM.D. The Global Evolution of Meningococcal Epidemiology Following the Introduction of Meningococcal Vaccines, Journal of Adolescent Health, Volume 59, Issue 2, Supplement, August 2016, Pages S3-S11; 4. Lingappa J. R., Al-Rabeah A.M., Hajjeh A., et al. Serogroup W-135 meningococcal disease during the Hajj, 2000 //Emerging Infection Disease. 2003. vol. 9. N6. pp 665–671. ; 5. Meningococcal infection and purulent bacterial meningitis in the Russian Federation in 2016. Information-analytical review. - Federal Service for Supervision of Consumer Rights Protection and Human Welfare. Russian reference center for monitoring bacterial meningitis. Moscow, 2017.; 6. Korozyova I.S., Beloshitskiy G.V., Zakroeva I.M., Korozyova M.A., Meningococcal disease in the Russian Federation. – Medical Alphabet №6 2015, Vol. 1 Epidemiology and Hygiene (RU). 7. Lobzin YU.V., Skripchenko N.V., Vi'nic A.A., Ivanova M.V. i dr. //Infektologia. 2016; 8,1: 19-25 (RU).

Fig 2. IMD Fatality rate depending on the strain of infectious agent, Russia, 2016. Data of National Reference Center for IMD & Bacterial Meningitis

In June-July 2017, in St Petersburg we observed the family case of generalized meningococcal infection caused by Men W strain. Twins of 7 years old were infected by their Mom who had light nasopharyngeal symptoms, sore throat and sub febrile fever two days prior to the disease onset. Boy G. had a 4-year history of glomerulonephritis with nephrotic syndrome (w/o exacerbation at the time of IMD onset). Meningococcal disease had acute manifestation with severe headache, repeated vomiting, increasing apathy and slackness passing rapidly into lethargy turning into stunning. Both boys were admitted to the hospital within first 24 hours from the disease onset. In admission, their condition was assessed as extremal due to clinical symptoms of purulent meningitis, cerebral edema and multiple organ failure. Clinical blood tests verified moderate leukocytosis with a shift in leukocyte formula to young forms. Cerebrospinal fluid analysis confirmed high neutrophilic pleocytosis with significant protein increase (Tab 1).

	Boy A.	Boy G.
Status presence	✓ Condition: critical due to syndrome of cerebral edema ✓ Fever: 37,8°C ✓ Consciousness: 8-9 GCS (The Glasgow Coma Scale), transitory anisocoria ✓ Meningeal symptoms: positive + ✓ Somatic status: unremarkable ✓ Clinical blood test: ER 3,68 x10 ¹² /l, Hb 100g/l, Ley 10,4 x10 ⁹ /l, j 1, stab 21, segmented 62, lymph 13, mon 3, ESR 22 mm/h PCT – 47	✓ Condition: critical due to syndrome of cerebral edema ✓ Fever: 37,4°C ✓ Consciousness: 8-9 GCS (The Glasgow Coma Scale) ✓ Meningeal symptoms: positive ++ ✓ Somatic status: unremarkable ✓ Clinical blood test: ER 4,4 x10 ¹² /l, Hb 126 g/l, Ley 15,1 x10 ⁹ /l, j 8, stab 28, segmented 55, lymph 8, mon 1, ESR 19 mm/h PCT - 28

	Boy A.	Boy G.
Lab tests	✓ CSF test (6 day) : Cytosis – 9 472 /3 Protein – 0.85 g/l Glucose – 7.6 mmol/l ✓ PCR (blood): N.meningitidis positive ✓ RLA (blood): N.meningitidis positive ✓ PCR (CSF): N.meningitidis positive ✓ Bacteriology (Blood) N.meningitidis W	✓ CSF test (4 day) : Cytosis – 40 988 /3 Protein 0,26 g/l Glucose – 4.2 mmol/l ✓ PCR (blood): N.meningitidis positive ✓ RLA (blood): N.meningitidis positive ✓ PCR (CSF): N.meningitidis positive ✓ Bacteriology (CSF) N.meningitidis W

Tab 1. Clinical data & lab test results in two boys at the moment of admission to the hospital

Fig 3. Non-plentiful hemorrhagic rash, cerebral edema and perifocal circulatory disorders in Boy A.

According to the severity of the condition, the patients were immediately put on mechanical lung ventilation. Massive antibacterial and anti-edematous treatment were performed. Against the backdrop of the treatment, we achieved stabilization of patient’s condition on the 5th day from the therapy start. Sanitation of cerebrospinal fluid confirmed on the 16th day. ST-11 sequence-type of Men W strain was isolated from blood and liquor of both patients and from the nasopharyngeal swab of their Mom.

Conclusions

The clinical case shows the peculiar properties of Men W disease course. It demonstrates that the most severe and complicated W-135 cases are registered in children of older age group (here – twins of 7 years of age). Other distinctive clinical features of W-135 infection can be described. As following: a sub-acute onset; non-plentiful hemorrhagic rash that appears later on the 4-6th day from onset of the disease and its predominant localization on extremities and distal parts of the body. Specific changes in lab tests are determined as: neutrophil pleocytosis and high protein level in CSF., insignificant leukocytosis and left leycocyte shift in Blood test. The CT changes in W-135 infection are also specific and considered brain edema and exudative (purulent-necrotic, septic) inflammation with secondary microcirculatory disorders.in the affected tissues.