Fatal meningococcaemia in a SARS-CoV-2-positive adolescent

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Novel coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection has been spreading world-wide since December 2019. Initial reports described children as largely spared from severe manifestations; however, since April 2020, multisystem inflammatory syndrome in children (MIS-C) has been described associated with SARS-CoV-2 infection.2

Bacterial co-infection in patients infected with SARS-CoV-2 remains little understood.3,4 The authors report a case of meningococcaemia in a SARS-CoV-2-positive adolescent and highlight the importance of differential diagnosis, as well as the impact of an emergent disease.

Case Report

A 14-year-old adolescent male, whose psoriasis had been treated with prednisolone 0.2 mg/kg/day for 2 months, presented to the emergency department with a 12-h history of fever, dyspnoea and vomiting. He lived in an area with a high prevalence of SARS-CoV-2 infection. He had no recent travel history, exposure to sick contacts or any significant family history.

Upon presentation, he was poorly responsive and hypotensive, tachypnoeic, tachycardic and febrile. A prolonged capillary refill time and purpuric rash were evident. Cultures were collected; intravenous ceftriaxone and time and purpuric rash were evident. Cultures were collected; intravenous ceftriaxone and

Laboratory tests revealed thrombocytopenia, high C-reactive protein, acute renal failure and metabolic acidosis with hyperlacticacidaemia. Nasopharyngeal swab was positive for SARS-CoV-2 by polymerase chain reaction (PCR).

In this early phase, toxic shock syndrome and therefore MIS-C were evaluated as a possible cause together with our initial hypothesis of meningococcal or pneumococcal invasive disease.

Clinical deterioration with refractory shock and multi-organ dysfunction was observed, non-responsive to all treatment measures including mechanical ventilation, resuscitation guided by invasive hemodynamic monitoring, renal replacement therapy, large broad antibiotics (meropenem and vancomycin), blood products and intravenous immunoglobulin, resulting in his subsequent death 18 h post-admission.

Blood cultures grew Neisseria meningitidis serogroup B after 48 h of incubation, sensitive to penicillin and ceftriaxone. The patient had not received vaccination for this strain of meningococcus, which only became routinely available in Portugal in October 2020.

Discussion

Despite a downward trend in recent years, N. meningitidis serogroup B has been responsible for the majority of reports of invasive meningococcal disease (IMD) in Portugal and Europe. The incidence of IMD is highest in infants, with a second peak occurring in adolescents and young adults. IMD depends on various factors related both to bacterial pathogenicity and to host immunity.

N. meningitidis spreads via respiratory droplet transmission and the human nasopharynx remains its only known reservoir. Asymptomatic colonisation (carriage) of the nasopharyngeal mucosa is higher during adolescence. The relationship between carriage and infection remains unclear.2

Infection with SARS-CoV-2 results in viral replication in the nasopharynx and viral detection is possible there, as well as in sputum, faeces, urine and blood. Internationally, the prevalence of confirmed COVID-19 is significantly lower in children than in adults.6

Although data on the severity of COVID-19 show more significant disease among adults and the elderly, a clinical manifestation called multisystem inflammatory syndrome was described in children. MIS-C is characterised by shock and features of macrophage activation resembling other known illnesses, mainly Kawasaki disease.2

In our patient, the diagnosis of meningococcaemia was suspected by the typical clinical course. However, living in a high-prevalence COVID 19 area, and the detection of SARS-CoV-2 by PCR swab being positive, led to the postulation that the presence of nasopharyngeal viral infection may have predisposed...
to IMD. It is known that viral infections can enhance bacterial colonisation of the airway by damaging the respiratory tract mucosa. Frequently, IMD is preceded by viral upper respiratory tract infections that may promote invasion.9

The possibility of N. meningitidis co-infection is of concern among children and young adults with SARS-CoV-2 infection, as higher rates of meningococcal carriage are known in this age range.7

Moreover, the fact that our patient was on prednisolone therapy raised the question whether this could have caused some degree of immunosuppression that may have contributed to this aggressive clinical course, although the low dosage administered argued against it.

MIS-C hypothesis as a cause of this fulminant disease was excluded due to both clinical and laboratory findings.

To our knowledge, there is only one other case report of meningococcaemia and COVID-19 co-infection.4 This case demonstrates the challenge around the diagnosis and management of a critical patient with suspected bacterial co-infection, in the COVID-19 pandemic era, with limited scientific literature and knowledge.

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