The amplification identified 94,717,3416 (7.98log)copies/ 10×5 cells of MCPyV.

MCC can often be confused with melanoma or lymphoma. Although the literature suggests MCC is more common in men, in this case the patient's age and the features and location of the lesion led to MCC being considered, and confirmed: patient over 50 years; painless, fast growing lesion on upper limb;[3] lesion measuring more than average tumour size at diagnosis (1.7 cm), alongside presence of MCPyV.

In most cases, cell transformation occurs through virus replication in mechanoreceptors[4] and other cell types.[5] However, the specifics of MCPyV host cell tropism(s) remain unclear, and it may be that the mechanism is mediated by oncogenes. One known trigger is UVA light although other trigger mechanisms have been identified in experimental trials,[3] including, as in this case, trauma injury. That said, only one other clinical similar case has been described in the literature, in a patient who received a blow to an area affected by Bowen's disease.[6]

In general MCCs produced by MCPyV have a better prognosis[1] when detected and diagnosed early, mortality being 30% at 2 years in such circumstances compared to 50% in patients diagnosed at advanced stages, where life expectancy can be as little as 9 months following diagnosis.[7]

There is no consensus in terms of treatment for MCC, although surgery is recommended at diagnosis, which may be complemented with radiotherapy and/or chemotherapy.

With this patient, two excisions were carried out as the first showed the tumour edges were affected. The second was thus made employing a safety margin of 1–2 cm.[8] Since no metastasis was found, radiotherapy or any other therapy was delayed.

The high viral load found suggests very active viral reproduction, and could imply rapid clinical progression (as is usual with this type of tumour). It also indicates the lesion was at an early stage, as does the absence of any metastasis in the axillary ganglion, which is observed in 70% of patients at diagnosis, and thus surgery was considered sufficient treatment to eradicate the tumour.

In conclusion, in undifferentiated skin lesions, when other common pathologies can be discounted and the patient has experienced a trauma, MCC should be considered and MCPyV tested for early.

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Conflict of interest

No.

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Hemolytic anemia in pediatric patients treated with artesunate for severe malaria☆

Anemia hemolítica tardía en niños tratados con artesunato intravenoso por malaria grave

Dear Editor:

Intravenous artesunate is currently the recommended first-line treatment in cases of severe malaria, with a reduction in death rates of 23% in children compared with quinine. Although it demonstrates an acceptable safety profile compared with other drugs, the potential for haemolytic anaemia associated with its use has been described.[1–3]

Three cases of paediatric patients treated with artemisinin derivatives for severe imported malaria who subsequently developed haemolytic anaemia have been described. The three patients were born in Spain, with no personal history of previous malaria.

Two sisters aged 6 and 4 years old were diagnosed with severe *Plasmodium falciparum (P. falciparum)* malaria at our centre, with parasitaemia of 5% and 15% respectively. There was history of a recent stay in Senegal without antimalarial prophylaxis. They received treatment with intravenous artesunate (2.4 mg/kg/dose, every 12 h for the first two doses, and subsequently every 24 h), with a cumulative number of 5 doses for the older sister and 4 doses for the younger, followed by piperaquine-artenimol for 3 days. Both were afebrile with disappearance of the parasitaemia during the first 48 h. In laboratory test follow-up, parameters compatible with haemolytic anaemia were observed at 10 days after start of treatment: drop in haemoglobin to 7.4 mg/dl (in the follow-up 5 days before, haemoglobin was 9 g/dL) in the older sister and 6.3 mg/dl (in the follow-up 5 days before, haemoglobin was 8.8 g/dL) in the younger, increase in LDH to 751 U/L (previous follow-up 674 U/L) and 1831 U/L (previous follow-up 738 U/L)

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respectively, undetectable haptoglobin levels and negative Coombs test in both cases. In the face of suspected haemolytic anaemia secondary to artemisinins, and once haemoglobinopathies had been ruled out, treatment was started with prednisolone at 1 mg/kg/day for 3 days with good clinical and lab test evolution although one of the patients, the older sister, required a packed red blood cell transfusion due to haemoglobin of 6.3 g/dL.

A 6-year-old boy is transferred to our site with a diagnosis of malaria due to *P. falciparum* and *Plasmodium vivax* in light of the need for intensive care (parasitaemia 25%). There was history of a recent extended stay in Gambia, without antimalarial prophylaxis. At his site of origin he was treated with proguanil/atovaquone, with a poor clinical and lab test response at 24 h after admission. In ICU he was treated with cefotaxime and intravenous artesunate (2.4 mg/kg dose, 0.12 and 24 h) followed by piperaquine-artenimol 3 days with a favourable response. At 8 days of evolution haemolysis findings were detected: haemoglobin of 7.5 g/dL (drop of 2 g/dL compared to previous follow-up, 9.9 g/dL) was detected, hyperbilirubinaemia of 2.08 mg/dL (previous follow-up 1.5 mg/dL), elevation of LDH to 1831 IU/L (previous follow-up 354 IU/L), undetectable levels of haptoglobin and positive Coombs test), haemoglobinopathies test within normal range. In the face of suspected haemolytic anaemia secondary to artemisinins, prednisolone was started at 1 mg/kg/day for 3 days with favourable clinical and lab test evolution.

Artemisinin derivatives have become the first-line option for the treatment of severe malaria. Their most common side effects are mild, although haemolytic anaemia secondary to their use has also been described, with an estimated incidence of between 7–21% of cases treated with an intravenous artesunate, especially in patients with a higher degree of parasitaemia,[4] although cases of haemolysis associated with treatment with oral artemisinins have also been described.[5] It usually appears between the first and fourth weeks after the start of its administration, and it is a different process from blackwater fever associated with treatment with quinine, which usually appears earlier.[6] The ultimate cause of haemolysis is unknown, although several hypotheses have been put forward. One of these is the removal at the splenic level of the previously infected red blood cells, with inclusion bodies, due to the phenomenon of «pitting».[7,8] An immune mechanism has also been put forward, since in some patients a positive Coombs test has been observed,[9] such as the third case presented, and a favourable response to corticosteroids, although the role of this therapy has not been clearly established.[10] Other approaches have also been posed, such as direct toxicity of an artesunate metabolite, dihydroartemisinin. There are also factors that favour a greater individual predisposition, such as haemoglobinopathies (sickle cell disease or glucose-6-phosphate dehydrogenase deficiency) or due to interindividual variability in drug metabolic pathways.

Haemolytic anaemia is a potential complication associated with treatment with intravenous artesunate in paediatric patients, for which reason its appearance must be monitored for during the weeks following its administration, especially in patients with high parasitaemias.

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Severe bradycardia probably associated to Oseltamivir in a pediatric patient with acute renal injury

Bradicadia severa probablemente asociada a Oseltamivir en un paciente pediátrico con fracaso renal agudo

Dear Editor,

Children constitute a high-risk population for the development of severe influenza regarding adult population. Current recommendations state that antiviral treatment should be provided to all children hospitalized with influenza or underlying medical conditions or those suffering from a severe illness.[1] Oseltamivir, a neuraminidase inhibitor (NI), represents the most widely used antiviral in children with influenza viral infection. We report a 10-year-old previously healthy female admitted to our PICU due to an acute kidney injury (AKI) (creatinine clearance < 30 ml/min/1.73 m^2), anemia (7.9 g/dl) with schistocytosis of 3.5% and thrombocytopenia (29,000/mm^3). She was conscious and did not require respiratory or hemodynamical support (SpO_2 100%, blood pressure 107/68 mmHg and heart rate (HR) 110 bpm).