Fig. 1. *P. monteilii* colonies cultured on Columbia agar under aerobic conditions for 24 h. Colonies are white, shiny and mucoid.

tion, evidence of infection is not clearly demonstrated in many of the cases described and this could suggest that *P. monteilii* may have low pathogenicity, act as a coloniser and only be a source of infection in critically ill or immunocompromised patients or those who have biomedical devices.[10]

Conclusions

Based on the above, the growth of *P. monteilii* on culture media was associated with a nosocomial infection of the external drain valve, either due to colonisation from the patient's skin or handling of the device by healthcare staff, with this acting as a portal of entry for this microorganism into the ventricular fluid.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Varicella monoarthritis in an immunocompetent woman

Monoartritis varicelosa en una mujer inmunocompetente

Dear Editor,

A woman in her early 30s with no relevant medical record came to A&E with a 24-h history of a fever (37.8 °C) and generalized pruritic cutaneous lesions. She presented with vesicles and erythematous papules, some scabby after recent scratching, distributed in torso and extremities. She also mentioned arthralgia in her right knee associated with a discrete swelling without erythema or ecchymosis, which caused her functional impotence. She had no history of previous traumatism or arthritis in the knee. Weight bearing X-ray of the knees revealed an increase in intra-articular fluid and volume of soft tissue.

Peripheral white blood count showed $6.9 \times 10^6/\mu L$ leukocytes [normal values: $3.9–10.2 \times 10^6/\mu L$], with $4.56 \times 10^6/\mu L$ neutrophils, $1.4 \times 10^6/\mu L$ lymphocytes, $12.4 \times 10^6/\mu L$ monocytes—slightly high—[$2.0–9.5 \times 10^6/\mu L$], $1.1 \times 10^6/\mu L$ eosinophils and $0.0 \times 10^6/\mu L$ basophiles. Hepatic enzymes were increased, with AST and ALT values of 121 IU/L [0–40 IU/L] and 187 IU/L [0–35 IU/L) respectively, and a GGT of 254 IU/L [0–38 IU/L] and LDH of 560 IU/L [100–190 UI/L]. RCP had risen to 35.1 mg/L [0–5 mg/L].

Enquiring about her epidemiological environment, her stepson had at that moment the chickenpox. She did not recall contracting it as a child. Suspecting a VZV primary infection with a post-exanthematous arthritis, she started treatment with acyclovir (800 mg IV/4 h) and NSAIDs (dexketoprofen and acetaminophen).

Serology for several microorganisms returned positive IgG for Parvovirus, measles and Herpes simplex virus (HSV) 1/2 whereas their respective IgM were all negative. Both IgM and IgG for HSV,

rubella, syphilis and VVZ returned negative. Serology was also negative for HIV.

Before starting with acyclovir, a sample of joint fluid was taken. It was a transparent and yellowish liquid with no crystals but plenty of red blood cells and 4030/mm^3 nucleated cells. It had a glucose of 29 mg/dL, total proteins of 5.5 g/dL and 1854/mm^3 polymorphonuclear cells (46% neutrophils, 13% lymphocytes, 36% macrophages and 5% mesothelial cells). Bacterial culture after five days was sterile. However, the two different nucleic amplification tests (Clart® Entherpex, Genomica, and VHSI, VHSII and VZV Real Time Progenie Molecular) performed on the synovial fluid confirmed the VZV diagnosis.

After two doses of acyclovir IV and once confirmed the patient was clinically stable, she was discharged. She continued treatment with acyclovir 800 mg/4 h for 7 days. She had a follow-up serology repeated 10 days later: both VZV IgM and IgG returned distinctively positive (Varicella-Zoster ELISA IgM/IgG, Vircell). At the same time, a sample of the skin lesions was taken, and VZV was detected by PCR (Clart® Entherpex, Genomica).

VZV arthritis has a low incidence even in children.[1] In adults, it is usually seen in patients diagnosed with autoimmune diseases and subsequently treated with immunosuppressive therapy.[2] It is not known if the increased risk of VZV complications is due to the immunosuppression, the underlying disease or a combination of both.[3] It is extremely rare for an immunocompetent adult to develop this presentation.[4]

Detection of the virus has been reported before the exanthem but is usually seen at the onset or within the first 72 h.[5] It usually presents with a monoarticular pattern, being the knee the most commonly affected joint,[1] where it produces pain, swelling and limitation of motion.

The pathogenic path of the joint infection is not clear. Several hypotheses have been postulated, including direct invasion of the joint, immune complex formation or immune dysregulation.[6] Isolation of the DNA virus had only been achieved a few times before in the literature.[7]

Our patient presented an IgM rise and subsequent seroconversion. Clinical symptoms were compatible with chickenpox and VZV DNA was detected in the skin lesions and the joint fluid. We can safely assume that the arthritis was reactive to a primary infection by VZV.

Bacterial exclusion must always be performed in VZV acute arthritis since it mimics a septic joint. Septic arthritis is provoked by the haematogenous spread of the bacteria from infected lesions, so it occurs after the onset of the exanthem.[8] Detection of VZV DNA by PCR reduces the time of diagnosis and allows us to rule out septic arthritis. VZV arthritis usually has a short duration and it generally do not persist or recur. However, providing prompt acyclovir treatment should be assessed to see if it reduces the duration of the symptoms.

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