associated with additional cytogenetic abnormalities and mutations within *BCR-ABL*.⁶ Further studies have also demonstrated that disruption to Ikaros can play a critical role in progression of CML to advanced stages.⁷

A diagnosis of myeloma and CML in the same patient is a rare event, only reported in a small number of case studies.^{2,3,5} The postulated aetiologies of synchronous or metachronous diagnosis of two different haematological malignancies includes a common haematological progenitor stem cell, environmental exposure to carcinogens, presence of pre-existing plasma cell myeloma, treatment of one haematology malignancy predisposing to the second diagnosis and genetic or epigenetic changes associated with myeloma predisposing to other malignancies.^{8–10} Simultaneous diagnosis of myeloma and CML is even rarer and raises a therapeutic conundrum. IMiDs form the backbone of myeloma therapy and are known to cause Ikaros suppression, particularly in myeloma cells; however, Ikaros suppression has been demonstrated to be a potential cause of progression to advanced stages in CML. Whether lenalidomide is safe to use in patients with synchronous CML and myeloma remains unclear. Whilst the risk remains theoretical, due to the paucity of patients diagnosed with concurrent myeloma and CML, there is almost no real-world data to guide therapy. In our case, given the patient did not imminently need myeloma-directed therapy, we elected to control the CML clone with a TKI with the aim of maximal suppression, to allow adequate myeloma therapy if and when he requires it.

This case demonstrates the ongoing critical role that karyotype analysis plays in the diagnosis of haematological malignancies. The incidental finding of CML has real-world therapeutic ramifications for our patient and highlights the ongoing importance of maintaining karyotype analysis, even with the expansion of more sensitive techniques such as FISH and genome sequencing.

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***Enterococcus cecorum* infective endocarditis in a patient with chronic myeloid leukaemia and cirrhosis**

To the Editor,

Enterococcus cecorum is a rare infection in humans, predominantly occurring in immunocompromised patients. We report the second known case of infective endocarditis caused by *E. cecorum* in a patient with chronic myeloid leukemia (CML).

A 70-year-old man presented to the Emergency Department with a 2-week history of dyspnoea, fatigue and intermittent fevers. He had a background history of CML diagnosed in 2003, which was initially in a sustained molecular remission on imatinib. In 2019, after developing an imatinib resistance mutation, therapy was changed to dasatinib 100 mg daily. Other medical background included alcohol-induced hepatic cirrhosis with associated portal hypertension. He underwent an endoscopy for upper gastrointestinal bleeding 4 months prior to presentation with findings of portal hypertensive gastropathy. The patient's only zoonotic exposure was his pet dog.

The peripheral blood count was unremarkable and a chest X-ray demonstrated bilateral moderate sized pleural effusions. Blood cultures were positive for non-motile, Gram-positive cocci in chains growing in both aerobic and anaerobic bottles. The isolate was unambiguously identified as *E. cecorum* on matrix assisted laser desorption-ionisation time of flight (MALDI-TOF) mass spectrometry (Vitek; bioMérieux, France) with a score of 2.2. The patient commenced therapy with intravenous benzylpenicillin and synergistic ceftriaxone. Dasatinib was temporarily ceased. A transoesophageal echocardiogram revealed an 8×8 mm vegetation attached to the right coronary cusp with abscess formation and severe aortic incompetence.

Antimicrobial susceptibility testing was achieved using antibiotic gradient strips and interpreted against European Committee on Antimicrobial Susceptibility Testing (EUCAST) published breakpoints. The isolate demonstrated susceptibility to ampicillin [minimum inhibitory concentration (MIC) 0.38 mg/L], vancomycin (0.75 mg/L), and linezolid (0.75 mg/L). No breakpoint exists for cefotaxime (MIC 0.19 mg/L).

The patient underwent an aortic valve replacement with a bio-prosthetic valve and coronary artery bypass graft. Macroscopic examination of the aortic valve cusp showed vegetation (Fig. 1). Gram stain of the resected aortic valve leaflet revealed numerous Gram-positive cocci in chains. Valve cultures ultimately grew *E. cecorum*. Identification of the organism was confirmed by gene sequencing (16S rRNA PCR). Histopathology of the aortic valve demonstrated a vegetation comprised of necrosis, inflammatory exudate and granulation (Fig. 2). Also notable were nodular calcifications and myxoid changes with thick-walled blood vessels, macrophages and lymphocytes. These findings were consistent with acute endomyocarditis on a background of chronic valvulitis.

The patient completed a total of 6 weeks of ceftriaxone and benzylpenicillin with complete resolution of dyspnoea and fevers. He was recommenced on dasatinib several weeks after the aortic valve replacement.

Enterococcus cecorum was first isolated from the intestines of poultry in 1983 and was subsequently observed to cause infections such as osteomyelitis and endocarditis in broiler chickens.¹ It was originally named *Streptococcus cecorum* but in 1984 reverse transcriptase sequencing of the 16S ribosomal RNA led to reclassification.² *Enterococcus cecorum* is phylogenetically closer to *Streptococcus* species when compared with other *Enterococcus* species, as incubation in carbon dioxide rich conditions is optimal. It does not grow on *Enterococcus* selective media, it grows poorly on bile-esculin agar, and unlike other clinically significant enterococci, *E. cecorum* is Lancefield group D negative.

Early literature describes misidentification based on biochemical/phenotypic identification methods,³ which may have resulted in an underestimation of the true number of infections. However, reports of *E. cecorum* human infections have been sparse, even after the advent of mass spectrometry, and it remains a rare cause of disease in humans.

Close contact with animals is thought to be a major risk factor for *E. cecorum* infection in humans.³ The source of infection was not confirmed in our case, however it is likely to have been exposure to his dog and/or food contamination from handling raw poultry with subsequent inoculation via the enteral tract. Infections via the cutaneous route have been postulated in the literature^{4,5} though this was not apparent in the patient's history.

Since its discovery in 1983, there have been 12 other cases of *E. cecorum* infection documented in the literature. Infection types have included bacteraemia, peritonitis, empyema thoracis, infective endocarditis and a knee joint infection.^{3–10}

Fig. 1 Aortic valve cusps with vegetations.

Fig. 2 Histopathology of aortic valve cusps showing necrosis, macrophage and lymphocyte infiltration and granulation.

Half of the reported cases (6/12 cases) had decompensated liver cirrhosis or gastrointestinal pathology such as Crohn's disease with a common risk factor of chronic compromised local mucosal immunity. It is well recognised that decompensated liver cirrhosis with portal hypertension and ascites is associated with intestinal mucosal oedema, reduced intestinal motility and failure of local host defences.¹¹ Of the remaining cases without a history of cirrhosis or liver disease, 50% (3/6) were immunocompromised in some way (malnutrition, immunosuppressive medication).

The only other reported case of *E. cecorum* infective endocarditis was a previously well 58-year-old male who had undergone dental extraction 5 weeks earlier.³ The aortic valve was affected, eventually resulting in valve replacement for worsening aortic regurgitation and failure to respond to the initial antibiotic regimen of benzylpenicillin and gentamicin. The patient made a full recovery following surgery and received an extended course of vancomycin and rifampicin.

Dasatinib is a potent second-generation multikinase inhibitor used in the treatment of CML and Philadelphia chromosome-positive acute lymphoblastic leukaemia. In addition to BCR-ABL, dasatinib also inhibits several other tyrosine kinases, including c-Kit and has been shown to inhibit the pro-inflammatory functions of mature human neutrophils. As such, dasatinib has been associated with severe prolonged myelosuppression and an increased risk of infection, particularly bacterial infection.¹² It is possible, although difficult to confirm, that the occurrence of *E. cecorum* infective endocarditis following the switch from imatinib to dasatinib in our patient may have been reflective of the potent immunosuppressive impact of dasatinib.

The optimal management of *E. cecorum* invasive infection is unclear. Although non-infective endocarditis infections have been reported to have favourable outcomes after short courses of antibiotics, the two patients presenting with endocarditis eventually required surgery. The isolate would be considered susceptible to cefotaxime if the MIC were referenced against breakpoints for streptococci (e.g., *S. pneumoniae*), a characteristic that has previously been described.⁶ As *E. cecorum* is typically susceptible to cephalosporins, a 4-week course of combined β -lactam treatment (ampicillin and ceftriaxone) may be preferred instead of β -lactam/aminoglycoside combination. In addition, although both cases presented with significant valvular dysfunction, the penicillin MIC is predictably low and phenotypically more similar to *Streptococcus*, raising the possibility that a single antibiotic (penicillin or cephalosporin) may be

sufficient in *E. cecorum* native valve endocarditis if treatment is commenced early.

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Actinomycosis canaliculitis

To the Editor,

Lacrimal canaliculitis is a rare medical condition, commonly undiagnosed or misdiagnosed for long periods of time in up

to 60% of patients.¹ It is an infection of the canaliculus of the lacrimal drainage system, often symptomatic with chronic tearing of the eye and irritation in the medial portion of the infected eyelid.² It accounts for only 1.2–2.0% of all lacrimal disease.^{1,3} Examination reveals erythematous protrusion of the lacrimal punctum, erythema of the surrounding skin and mucopurulent discharge.² *Actinomyces*, belonging to the phylum Actinobacteria, are Gram-positive non-acid fast bacilli that can have a fungal mycelia-like appearance.⁴ First described in 1854 by Von Graefe,⁵ *Actinomyces*, typically *Actinomyces israelii*, is the most common aetiological causative organism of canaliculitis, although several organisms have been shown to cause the disease.² *Actinomyces* are commensal flora of the oral cavity, likely causing canaliculitis by either regurgitation or direct inoculation by saliva.^{1,2} Diagnosis of the actinomycotic infection is confirmed by culture, with *Actinomyces israelii* expressed concretions often having unique yellowish 'sulphur granules' composed of aggregates of filamentous branching microorganisms^{2,3} and showing characteristic stellate radiations of the organism.² Due to its rarity, actinomycosis canaliculitis can present a diagnostic challenge, leading to unnecessary suffering for patients, making it essential to recognise the condition early.²

An 88-year-old female presented to the treating ophthalmologist for a second opinion with a 4-year history of tender, injected conjunctiva associated with purulent discharge with episodes lasting for a period of 2–3 weeks. Previously diagnosed with left blocked nasolacrimal duct in 2014, the patient remained symptomatic after surgery and multiple regimes of oral flucloxacillin and chloramphenicol ointment. Medical history included primary open angle glaucoma managed with latanoprost, timolol and brinzolamide, type 2 diabetes managed with metformin and insulin, and hypothyroidism treated with thyroxine. Previous ophthalmic procedures were left phacoemulsification and insertion of intraocular lens and left dacryocystorhinostomy. Visual acuities were right eye 6/9–1 pinhole (Ph) 6/5–1 and left eye 6/60 Ph 6/18 with bilateral intraocular pressures of 15 mmHg. Anterior segment examination was unremarkable. A tender erythematous lump was present in the left medial canthus inferiorly, with exudate in punctum (Fig. 1).

Fig. 1 Pouting punctum, discharge and medial injection of conjunctiva, typical of canaliculitis.