RARECARE project. Cancer Epidemiol 2014; 38: 670–8).
Although KS remains an AIDS-defining illness, the advent of combination antiretroviral therapy in 1995 has dramatically reduced the risk of KS among HIV-positive patients. In this study, two-thirds of new diagnoses occurred in HIV-negative patients. Awareness of this evolving epidemiology among dermatologists is important to facilitate early diagnosis and appropriate treatment.

**B13**
Abstract withdrawn.

**B14**
**Painful cutaneous nodules in chronic neutrophilic leukaemia**
Christopher Phillips, Olivia Espinosa, Gorav Wali, John Reed and Rubeta Matin
Churchill Hospital, Oxford, UK
Chronic neutrophilic leukaemia (CNL) is a very rare, often clinically aggressive myeloid malignancy with few effective treatment options. Neutrophilic dermatosis of the dorsal hands (NDDH) is an uncommon, localized variant of Sweet syndrome (SS), first described in 1995. It is histologically identical to SS and characterized by tender violaceous nodules or plaques on the dorsal hands. We present the first case, to our knowledge, of NDDH in a case of CNL highlighting the challenge of histopathological differential diagnosis of leukaemia cutis (LC). A 74-year-old man with a 2-month history of CNL presented with a 10-day history of firm, tender, erythematous nodules and plaques over the dorsal aspects of both hands. He was systemically well and reported a similar milder episode 3 months earlier that had spontaneously resolved over 3 weeks. Blood tests revealed a neutrophilia of $57 \times 10^6$ cells L$^{-1}$, in keeping with known CNL. A skin biopsy demonstrated squamous syringometaplasia and focal papillary dermal oedema, which merged with a pandermal infiltrate of mature neutrophils and scattered larger histiocytoid cells but no admixed blasts and no immunohistochemical evidence of neoplasia. The histopathological differential diagnosis lay between NDDH with histiocytoid features and LC. The lesions spontaneously resolved within 3 weeks with topical clobetasol propionate, which supported the diagnosis of NDDH. To the best of our knowledge, this is the first reported case of NDDH presenting in the context of CNL, highlighting the importance of clinicopathological correlation. Histologically, it is challenging and almost impossible to distinguish CNL and NDDH. The clinical course of disease is relevant. NDDH is characterized by an abrupt onset of tender skin lesions with good response to corticosteroids or spontaneous resolution within several weeks; LC can comprise nontender lesions of more gradual onset that worsens as leukaemia progresses and requires systemic chemotherapy. Histopathology of NDDH is that of a neutrophilic dermatosis, characterized by a dense dermal infiltrate of mature neutrophils with associated dermal oedema and sometimes prominent leucocytoclasia in the absence of bona fide vasculitis. In contrast, neutrophils in CNL are immature, on occasions manifesting in atypia, often arranged in a perivascular location with no significant dermal oedema. Immunophenotypic and molecular studies can also establish the presence of blasts in the skin. This case highlights that clinicopathological correlation is essential to establish this distinction, where a more conservative approach can be taken for NDDH, but the poor prognosis associated with LC would require systemic chemotherapy.

**B15**
The impact of COVID-19 and ruxolitinib on extracorporeal photopheresis in chronic graft-versus-host disease: a retrospective observational study
Libin Mathew, Sukran Saglam and Fiona Child
St John’s Institute of Dermatology, London, UK
Chronic grafi-versus-host disease (GvHD) is one of the major complications following allogenic haematopoietic stem cell transplantation. Second-line treatment options in refractory chronic GvHD include extracorporeal photopheresis (ECP) (Dignan FL, Clark A, Amrolia P et al. Diagnosis and management of acute graft-versus-host disease. Br J Hematol 2012; 158: 30–45). ECP is a cell-based immunomodulatory therapy, available in a limited number of centres across the country. Patients often have to travel to reach their nearest centre. In 2020, with the onset of the COVID-19 pandemic, all patients with GvHD were considered clinically extremely vulnerable and advised to shield. Janus kinase (JAK) signalling pathways are known to be activated in GvHD. Ruxolitinib is a selective inhibitor of JAK1 and JAK2, available as a tablet that can be taken at home (Zeiser R, von Bubnoff N, Butler J et al. Ruxolitinib for glucocorticoid-refractory acute graft-versus-host disease. N Engl J Med 2020; 382: 1800–10). At the onset of the pandemic, ruxolitinib did not have a European Medicines Agency licence for GvHD. NHS England prepared a rapid policy development process that allowed for specialized centres to initiate ruxolitinib with the aim of reducing hospital attendance for those deemed extremely vulnerable. We carried out a retrospective analysis of our ECP patient cohort to assess the effect of COVID-19 on patients stopping their ECP therapy and the impact of ruxolitinib. Our cohort consisted of 118 GvHD patients (mean age 52 years). Thirty-eight patients stopped ECP in the last 2 years, with 26 patients stopping in order to shield. Of the patients who stopped, 21 subsequently restarted ECP owing to deteriorating GvHD. Thirteen patients were commenced on ruxolitinib with nine continued on both ECP and ruxolitinib. Of the nine patients on combination therapy, two stopped ruxolitinib because of side-effects, while another found no benefit and two patients had no reduction in their ECP frequency. Our results show that although 22% of our cohort stopped ECP at the beginning of the pandemic in order to shield, the majority chose to continue their therapy. Eighty per cent of those who stopped to shield subsequently restarted ECP to manage worsening symptoms or as a result of side-effects from alternative therapies. Ruxolitinib...
was introduced in 11% of patients, four of whom were able to continue it as an effective alternative to ECP. Our data illustrate that we were able to continue to deliver a safe and effective ECP service during the pandemic and that ruxolitinib provides an alternative second-line option in the management of refractory chronic GvHD.

**B16**

**Severe epidermolysis bullosa acquisita in a patient with graft-versus-host disease: causal or coincidental?**

Jaimie Oldham, Sasha Dhoat, Catherine Harwood and Rebeca Goiriz

Department of Dermatology, Barts Health NHS Trust, London, UK

Graft-versus-host disease (GvHD) is common after haematopoietic cell transplantation (HCT). Mucocutaneous manifestations are variable and may simulate autoimmune bullous dermatoses. However, the association of GvHD with autoimmune disorders, including bullous dermatoses, is also well recognized. We describe a patient with GvHD in whom severe and relapsing epidermolysis bullosa acquisita (EBA) was diagnosed 3 years after transplant and propose a causal association with GvHD. A 66-year-old woman developed GvHD following allogeneic HCT for acute myeloid leukaemia in 2016. This affected her gastrointestinal tract and skin but improved with oral corticosteroids and ciclosporin. In 2019 she presented with a widespread rash consisting of large, tense, haemorrhagic blisters. Histological features were in keeping with EBA. Direct immunofluorescence was also consistent with EBA, demonstrating linear positivity for IgG and C3 confined to the blister base, as was detection of collagen VII antibodies on indirect immunofluorescence. She was admitted and treated with high-dose oral steroids, ciclosporin and intravenous immunoglobulin (IVIg) with eventual resolution of blistering. Although further IVIg administration was planned as an outpatient, this coincided with the start of the COVID-19 pandemic and she elected not to attend and also stopped all medication. Despite this, her EBA remained quiescent until September 2021 when she was readmitted with a severe deterioration in blistering and significant dysphagia due to an oesophageal stricture, with a weight of 31.7 kg. Once again, she responded rapidly to oral prednisolone and IVIg. Dapsone was considered but precluded by G6PD deficiency and there were clinical and adherence concerns about using mycophenolate mofetil. Upon discharge she was again nonadherent to medication and failed to attend for planned IVIg. She flared and was admitted for a third time in December 2021, requiring gastrostomy for nutritional support; her weight at this time was 26.4 kg. Her EBA is currently well controlled on prednisolone and IVIg. EBA is a rare, acquired blistering disorder secondary to autoantibodies targeting type VII collagen. Previous studies have found circulating basement membrane zone (BMZ) antibodies in 24% of chronic GvHD patients, possibly generated in response to chronic BMZ damage (Hofmann SC, Kopp G, Gall C et al. Basement membrane antibodies in sera of haematopoietic cell recipients are associated with graft-versus-host disease. J Eur Acad Dermatol Venereol 2010; 24: 587–94). Corresponding clinical manifestations are rare, with bullous pemphigoid the most frequently reported. EBA is much less common with four previously reported cases [Brassat S, Fleury J, Camus M, et al. (Epidermolysa bullosa acquisita and graft-versus-host disease). Ann Dermatol Venereol 2014; 141: 369–73 (in French)]. As a fifth case of EBA, our patient provides further evidence of a likely pathophysiological relationship between GvHD and autoimmune subepidermal bullous dermatoses, and highlights the significant challenges of managing these vulnerable patient groups during the COVID-19 pandemic.