nodule, presumed site of the tick bite occurred two months prior. She did not develop fever, arthralgias or other systemic symptoms. In order to exclude autoimmune diseases and infectious etiologies, we performed laboratory exams, such as anti-thyroid, antinuclear, anti-transglutaminase, TORCH and anti-Borrelia antibodies, resulted negative. A punch biopsy specimen from the scalp (0.4×0.3×0.2 cm) revealed fibrosis of the derma and the peripheric areas of pili-sebaceous annexes. The following month, we observed a rapid centrifugal progression to total alopecia. Thus, we decided to attempt therapy with topic corticosteroids followed by a progressive hair regrowth during the following four months.

Tick bite alopecia was first described in 1921. Since then, a few other cases have been reported in the international literature. The characteristic manifestation is a single zone of alopecia, often with a centrifugal spread, that appears 1–2 weeks after the tick removal. Sometimes, it can be associated with a central eschar, representing the site of tick bite. The nonscarring forms of alopecia manifest as ‘moth-eaten’ patches or, in alternative, as nodular or blood-crusts lesions. Clinically, patients may present with pain, pruritus or swelling. The precise mechanism for hair loss is not well understood but it is assumed to be caused by the host inflammatory response to tick saliva antigens. The result is the destruction of hair follicles or the alteration of the catagen/telogen phase. Histologic findings may show a heterogeneous inflammatory infiltrate and areas of fibrosis. The international literature does not report effective therapy for tick bite alopecia, while treatment with topic corticosteroids for alopecia areata is recognized. Prognosis is favourable with a complete hair regrowth usually within 3 months, although in some cases alopecia is reported to persist for 5 years.

GP90

VACCINE INDUCED PERSISTENT SKIN REACTION WITH LOCALISED ACQUIRED HYPERTRICHOSIS

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Background Vaccine induced persistently itchy granulomas are described in the setting of aluminium adsorbed vaccine preparations.^{1–2} Acquired localised hypertrichosis (ALH) has been attributed to inflammation yet rarely reported at vaccination sites². A Swedish longitudinal study estimated the incidence rate of vaccine induced granulomas as 0.8%³. 77–95% of those where contact allergic to aluminium with sensitivity to aluminium decreasing over time³. The granulomas are reported to resolve spontaneously over a number of years.³ The Meningococcal B ‘Bexsero’ vaccine is aluminium adsorbed and given by intramuscular injection.

Case We present the case of a 3 year old female with a vaccine induced persistent subcutaneous nodule and ALH. At age 12 months she received an intramuscular injection of Bexsero to her left anterolateral thigh. Within 2 weeks she developed a subcutaneous nodule at the injection site with an associated intensely localised pruritus causing her discomfort. Examination revealed a non tender subcutaneous nodule with an overlying patch of lichenified eczema with prominent growth of fine brown hair. Unfortunately, the pruritus was recalcitrant to

treatment with occlusive dressings, emollients or application of 1% hydrocortisone cream. There was a good response however to topical Clobetasol but on ceasing application the pruritus flared immediately.

Her parents were reassured of the benign nature of the nodule and likelihood that a contact allergy to aluminium had developed. Caution with future aluminium exposure was recommended. They were advised that it may take up to a number of years, if at all, for the nodule to fully resolve and that studies into long term outcomes of the reaction are lacking. Nevertheless, their experience did not deter them from the national vaccination schedule. The patient has not experienced any further adverse reactions to vaccinations received.

Discussion Vaccine induced persistently pruritic subcutaneous nodules with ALH have been associated with aluminium adsorbed vaccines. The long term sequelae, if any, to this reaction is unknown. It is important for clinicians to recognise this reaction as a potential side effect so as to avoid unnecessary medical investigation.

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GP91

SCREENING FOR CYSTIC FIBROSIS RELATED LIVER DISEASE WITH ULTRASONOGRAPHY

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Introduction Respiratory manifestations of cystic fibrosis (CF) have historically been the factor limiting prognosis. As there have been improvements in management of respiratory issues, increased screening and management of other manifestations of CF is required. CF related Liver Disease (CFLD) usually presents before or during adolescence and 5–10% of patients develop clinically significant disease. Diagnosis of CFLD is defined by two of the following; abnormal clinical examination, persistent liver function test (LFT) derangement or ultrasonographic evidence of disease. Available clinical guidelines for infants with CF recommend screening for CFLD. The Royal Brompton Care of Children with CF guideline and the CF Trust Standards of Care suggest routine abdominal ultrasound (US) screening from age 5. In contrast, the NICE guidelines do not specify an age at which to commence screening. Given the high treatment burden for patients with CF, we examined whether routine abdominal ultrasound screening for CFLD at age 5 leads to intervention or changes in clinical management.

Method We conducted a retrospective analysis of routine abdominal US screening conducted in CF patients at age 5, at the Royal Children's Hospital (Melbourne, Australia). The primary outcome measure was whether the US results led to a clinical intervention defined as treatment with ursodeoxycholic acid. Additional data collected included patient demographics,