

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.



Fig 2. Incidental finding of gas within stomach wall

Keywords: pneumobilia, blunt trauma, portal venous gas

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JOHN FAGAN AND THE PNEUMATIC TYRE

Editor,

It cannot be said of many presidents of the Ulster Medical Society that they helped to change the world but if a claim by John Fagan is true then he was one. The pneumatic tyre was patented in 1845 by Robert William Thomson who, despite demonstrating its advantages on heavy horse-drawn vehicles, could not make it a commercial success. John Boyd Dunlop, a veterinary surgeon with a large practice in May Street, Belfast, filed his own patent in 1888 but acknowledged

that it was doubtful it was valid when, in 1890, he learnt of Thomson's prior art. The efficiency and comfort of the pneumatic cycle tyre and its success in cycle races lead to a huge demand for it especially after Charles Kingston Welch made it detachable.

Sir Arthur du Cros published a history of the pneumatic tyre in 1938^[1] and was well placed to do so as he had been a director of the Pneumatic Tyre and Booth's Cycle Agency (his father's company with Dunlop on the Board) and of its successors, the first being the Dunlop Pneumatic Tyre Company. John Fagan (later Sir John Fagan), twice President of the Ulster Medical Society, had suggested that Dunlop's son, Johnnie, should take up cycling as it was an excellent form of exercise. The granite setts in the streets of Belfast made riding on solid tyres a jarring experience and Dunlop began to experiment with non-solid ones, initially filling them with water. Fagan had experience of air mattresses in his medical practice and du Cros states that Fagan frequently claimed to family and friends that he had suggested to Dunlop that he would be better to use air. Du Cros knew Dunlop very well from 1892 onwards and does not record any denial by him which perhaps lends credence to Fagan's claim. Thus Fagan would seem to have had a significant influence on the re-discovery of the pneumatic tyre on which modern road and air transport depends.

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PARANEOPLASTIC VITELLIFORM MACULOPATHY – ASSOCIATION WITH PRIMARY CANCERS

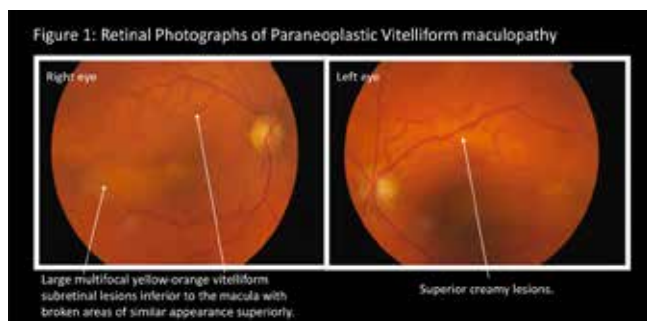
Editor,

We wish to highlight an important potential ophthalmic paraneoplastic presentation, that should trigger further investigations to diagnose underlying malignancy.

Paraneoplastic disorders are conditions related to systemic malignancy, but the effects occur at a site remote from the original tumour or metastases.¹ It is estimated that paraneoplastic syndromes affecting the nervous or visual systems occur in about 0.01% of patients with cancer.² A systematic review in 2013 by Rahimy and Saraf listed the 23 cases of paraneoplastic vitelliform maculopathy (PVM) reported in the literature at that time. Most of the cases described were associated with cutaneous or choroidal melanoma and only rarely with carcinoma. All the cases in the review were either associated with metastatic disease at the time of presentation to ophthalmology or metastases were discovered within the following months.³ The average age of onset was 59 years, with equal sex distribution.³

We present two cases of PVM; one associated with underlying cutaneous melanoma and one with primary breast carcinoma.

The first case was a 68-year-old lady (Figure 1) who presented with a one-year history of gradually decreasing vision bilaterally. She had a background of grade III ductal breast carcinoma, diagnosed five years previously. This was treated with mastectomy, chemotherapy and radiotherapy. At presentation her vision was RE 6/12, LE 6/15.



The second case was a 48-year-old lady who presented with bilateral visual distortion and gradually increasing blurred vision over 1 year. She had a medical history of malignant melanoma diagnosed 4 years before and treated with surgical resection and chemotherapy. At presentation to the ophthalmic team her vision was 6/6 right eye, 6/12 left eye. On retinal examination both cases revealed multifocal yellow-orange vitelliform lesions.

Prompt recognition of the clinical appearance of PVM can facilitate early investigation of underlying malignancy and metastases and it must be remembered that PVM may be the presentation of a distant primary malignancy. In the first case described here, there is currently no evidence of underlying metastases; this patient remains under close monitoring. Unfortunately, there is evidence that PVM is a poor prognostic indicator, with most patients having metastatic disease diagnosed shortly after presentation and succumbing to this from months to four years after presentation with PVM³.

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THE CHALLENGE OF ACHIEVING ADEQUATE ORAL IMMUNOSUPPRESSION IN A RENAL TRANSPLANT RECIPIENT WHO DEVELOPS SHORT BOWEL SYNDROME (SBS)

Editor,

A 39-year-old male with a renal transplant was admitted to

hospital with abdominal pain and vomiting. A computed tomography (CT) scan of abdomen showed ischaemic large bowel. He proceeded to a laparotomy with ileocaecal resection and right hemicolectomy. 2 days later he had worsening abdominal pain and a repeat CT abdomen demonstrated ischaemic small bowel. He had a further laparotomy, small bowel resection and end ileostomy, leaving only 1 metre of small bowel distal to the duodenal-jejunal flexure. 12 days later there was recurrence of small bowel ischaemia and a further 20cm of distal ileum was removed, leaving only 80cm of small bowel. Initial post-operative immunosuppression was established with intravenous (IV) hydrocortisone and IV cellcept, with no impairment in graft function.

The clinical challenge was how to achieve adequate oral immunosuppression in a patient with only 80cm of small bowel, presuming drug absorption from the gastrointestinal tract is significantly reduced.

Animal studies demonstrate tacrolimus absorption is predominantly in the upper part of the small intestine¹ and the colon². On review of the literature there are multiple cases which describe the use of tacrolimus in SBS, in both kidney³ and other solid organ transplants^{4,5}. Interestingly, adequate tacrolimus levels can be achieved in the presence of a jejunostomy⁴ and even in complete absence of small bowel⁵.

We stopped cellcept, commenced oral tacrolimus (Prograf) and converted IV hydrocortisone to oral prednisolone. Tacrolimus absorption was monitored with blood trough levels (target trough 5-10 µg/L). The patient was initially commenced on Prograf 5mg BD (0.15mg/kg). The first trough level was 12µg/L. After a period of elevated levels the dose was reduced to a maintenance dose of 1.5mg BD and this remained stable for many months

7 months later he underwent surgery to reverse the ileostomy. After reversal surgery, tacrolimus trough levels rose to 14-18 µg/L and Prograf dose was reduced to 1mg BD, maintaining stable trough levels 4-8 µg/L. There were no concerns regarding medication compliance with this patient. It is noteworthy that with ileostomy reversal, trough levels rose significantly. This supports observations in animal studies of further tacrolimus absorption in the colon².

This case reminds us of the challenge of attaining adequate oral immunosuppression in renal transplant recipients who develop SBS. Tacrolimus can be used in this situation. Trough levels should be monitored and the dose adjusted in line with the surgery performed.

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