Letters

RECURRENT VENOUS THROMBOSES AND ANTI-GLOMERULAR BASEMENT MEMBRANE ANTIBODY

Editor,

We wish to record an association between a severe thrombophilic state and the presence of anti-glomerular basement membrane (anti-GBM) antibody.

Case Report: An active man aged 41 noticed chest pain, haemoptysis and dark urine after a two-week lecture course late in March 2004. On 18 April he developed headache, dim vision, memory loss and epileptic seizures. Tests showed multiple cerebral haemorrhages and thrombosis of the anterior half of the superior sagittal sinus. Thromboses of the left brachial, axillary and subclavian veins and superior vena cava followed the insertion of a long line and he later developed a left femoral DVT and pulmonary emboli. He was anti-coagulated and transferred for convalescence on 28 May with an INR of 2.6 to Musgrave Park Hospital, Belfast. Despite continued anti-coagulation he developed a right below-knee DVT and pulmonary emboli in early June, extension of the right DVT to above the knee in mid June, and a recurrence of the left leg DVT in late July. His mother had multiple sclerosis and a cousin a lupus-like illness. A benign right adrenal phaeochromocytoma had been removed in 2000.

CRP was 38 mg/L (normal < 10) on 2nd April, 223 on 2nd May, and 111 on 28th June. Anti-GBM antibody titre was 193 (normal < 10) on 8th June, 108 on 17th June and 33 on 29th June. Plasma homocysteine was 21.3 micromol/L (normal 5.5–13.6). No other haematological, biochemical, or immunological abnormality was found, with thrombophilia screen, anti-cardiolipin antibody, convalescent urinary catecholamines, creatinine clearance and urinary protein in particular being normal. MIBG, CT and PET scans showed no phaeochromocytoma or other tumour, and OGD and colonoscopy were normal. He was given prednisolone from 1st July to 12th August. CRP was normal from 24th July onwards. Anti-GBM antibody titre was normal from 9th July onward apart from one reading of 27 on 27th July. He made an excellent recovery. He was advised to remain on warfarin for life.

Discussion: Anti-GBM antibody is not normally associated with recurrent venous thrombosis and its presence may simply have reflected an underlying immunological abnormality of unknown origin. Both it and the CRP responded promptly to steroid treatment. Only one thrombosis occurred after the prednisolone was started but the thrombotic tendency was probably already diminishing. Perhaps anti-GBM antibody should be looked for in other patients with severe thrombophilic states and high CRP.

We acknowledge the excellent care he received in the Lagan Valley Hospital, Lisburn, and the Royal Victoria Hospital, Belfast, and thank all who contributed to the good clinical outcome.

The authors have no conflict of interest.

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A CASE REPORT OF 5-FLUOROURACIL-INDUCED CORONARY ARTERY VASOSPASM

Editor,

5-Fluorouracil (5-FU) is a chemotherapeutic agent, frequently used in the treatment of solid tumours, including colorectal, breast and upper gastrointestinal cancers. 5-FU can cause angina (effort-related or at rest)[(1)], myocardial infarction, supraventricular and ventricular arrhythmias, acute pulmonary oedema, cardiogenic shock, cardiomyopathy, cardiac arrest, and sudden death, all recorded in association with intravenous (IV) infusion or bolus administration of the drug. We proceed to document a case of 5-Fluorouracil-induced coronary artery vasospasm.

Case History

A 76 year old gentleman diagnosed with adenocarcinoma of the low rectum and a coincidental left upper pole renal tumour, underwent abdomino-perineal excision of rectum (APER) and left nephrectomy. Pathology confirmed a PT1a papillary variant renal cell carcinoma requiring no further treatment and a rectal adenocarcinoma staged ypT3N1M0, for which adjuvant 5-Fluorouracil and Folinic acid (5-FU/FA) chemotherapy was planned.

The past medical history included supraventricular tachyarrhythmia (on verapamil), hypertension and hypercholesterolaemia. The adjuvant chemotherapy schedule comprised the administration of four cycles of weekly IV bolus 5-FU/FA chemotherapy.

After his first dose of 5-FU, oncology notes confirmed one episode of chest pain, with a history suggestive of angina. After cycle 3 IV bolus of 5-FU, he was admitted under the medical team with a history of two episodes of exertional ‘aching’ central chest pain, each lasting one hour, over a 24 hour period. Troponin ‘T’ at twelve hours was elevated at 0.07µg/l. However, the medical team conferred a diagnosis of ‘atypical chest pain’ and he was subsequently discharged within 24 hours.

In view of his persistent complaint of chest pain, 5-FU was discontinued and he was switched to oral Capecitabine at 25% dose reduction. Despite this, a further admission to the medical team occurred following an episode of central crushing chest pain on the day following Capecitabine treatment. 12-lead ECG confirmed transient antero-lateral ST-segment elevation and/or hyperacute T waves on presentation to Casualty, (figure 2 and 3). Cessation of chest pain was