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Case Report

The diagnostic maze of synchronous mass lesions in brain and lung

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published case reports of SR of lung cancer with brain metastasis. Furthermore, there are other pathological possibilities with higher probability of SR on steroids compared to lung cancer. This case report highlights the diagnostic maze surrounding the presence of synchronous mass lesions in the lung and brain and underscores the importance of pursuing histopathological confirmation in these circumstances.

Introduction

We report on an interesting case of spontaneous-regression of synchronous mass lesions in the lung and brain. The patient was discussed in a multi-disciplinary environment and the diagnosis of lung primary with brain metastasis was made based on the radiological and clinical presentation and the patient was commenced on steroids for symptomatic relief in the absence of histopathological confirmation. However, the lesions spontaneously regressed more than 15 months after initial presentation. Spontaneous regression (SR) of malignancy is an intriguing phenomenon that has been described particularly in the context of renal, melanomas and haematological malignancies. However, SR of lung cancer is an unusual phenomenon and there are no

Case report

In January 2013, an 81-year-old female patient presented with few weeks history of vomiting, unsteadiness and difficulty in writing. There was no recent history of any head trauma, seizures or loss of consciousness. She denied any systemic symptoms of fever, cough and shortness of breath, alteration of bowel habit, weight loss, or rash. Past medical history included hypertension, iron deficiency anaemia, hiatus hernia, and osteoarthritis. In July 2010, she was diagnosed with sero-negative rheumatoid arthritis and was commenced on methotrexate. The patient was investigated three months earlier for self-limiting back pain with weight-loss and underwent comprehensive screening for probable infection with negative cytomegalovirus IgM, Hepatitis B surface antigen and Paul-Bunnel test excluding acute Epstein–Barr virus (EBV) associated infection. During her admission she was taking methotrexate 25 mg/week and her other medications included amlodipine, bendroflumethiazide, lansoprazole, enalapril, gaviscon, codeine, paracetemol, cyclizine and furosemide. She never smoked.

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Table 1 – Temporally ordered display of laboratory investigations.

September 2012	CMV IgM antibody	Not detected
	Toxoplasma IgG	Not detected
	Hepatitis B surface antigen	Not detected
	Hepatitis C antibody	Not detected
	Paul Bunnell	Negative
February 2013	Haemoglobin	9.7 g/dl
	White cell count	17.0×10^9 L^{-1}
	Neutrophils	$14.89 \times 10^{*}9$ L^{-1}
	Platelets	$585 \times l0^{*}9$ L^{-1}
	MCV	88.3 fl
	Sodium	134 mmol/L
	Potassium	4.4 mmol/L
	Urea	9.7 mmol/L
	Creatinine	106 μmol/L
	Corrected calcium	2.41 mmol/L
	Serum iron	5.0 μmol/L
	Serum ferritin	184 μg/L
	Serum B12	453 ng/L
	Serum folate	13.8 μg/L
	Total bilirubin	7 μmol/L
	Alanine-transaminase (ALT)	16 IU/L
	Alkaline-phosphatase (ALP)	69 IU/L
	Albumin	34 g/L
	CRP	5.4 mg/L
	TSH	0.58 mU/L
	Plasma viscosity	1.74 (normal)
January 2015	EBV EBMA IgG	Positive
	AMCA	Negative
	Anti-nuclear Ab	Negative
	Cardiolipin IgG	<2.0 GPL (0.0–19)
	Anti-β2 Glycoprotein (IgG)	<1 μ/mL (0–19)
	IgA	1.45 g/L (0.80–4.00)
	IgG	6.4 g/L (6.0–16.0)
	IgM	0.50 g/L (0.50–2.00)
	Paraprotein	Not detected

She drank 7 units of alcohol per week and there was no family history of malignancy.

She was conscious and orientated in time, place and person and physical examination showed normal cardio-respiratory system. Neurological examination revealed positive cerebellar signs with wide-based gait, past pointing and positive heel shin test.

Investigations

The patient underwent comprehensive array of laboratory investigations as summarised in Table 1. The chest radiograph was reported as normal, but the contrast-enhanced computed tomography (CT) of chest, abdomen and pelvis demonstrated multiple scattered lesions in bilateral lung fields, the largest lesion measuring 2.8 × 1.3 cm in apical segment of right lower lobe with spiculated margins strongly suggestive of primary lung malignancy. There was no significantly enlarged mediastinal or hilar lymphadenopathy. Below the diaphragm there was evidence of 2.5 × 2.2 cm hypo-attenuating lesion in the posterior spleen.

Subsequently, she had magnetic resonance imaging (MRI) of the head with gadolinium that showed heterogeneous peripherally enhancing lesions on T1-weighted sequence in right cerebellar hemisphere and left occipital and parietal lobes with corresponding increase in T2WI signal reflecting marked surrounding peri-lesional oedema and localised mass effect on the occipital horn and body of left lateral ventricle (Figs. 1 and 2).

Treatment, outcome and follow-up

The case was discussed in Lung and Neurosurgical Multidisciplinary Team (MDT) meetings and there was overwhelming consensus on probable diagnosis of lung malignancy with brain metastasis. In view of patient's frailty and co-morbidities and reservations about her ability to tolerate active oncological therapy it was decided not to pursue histopathological confirmation as it was unlikely to change overall management and prognosis. The patient was commenced on dexamethasone 4 mg once daily to reduce the cerebral oedema and

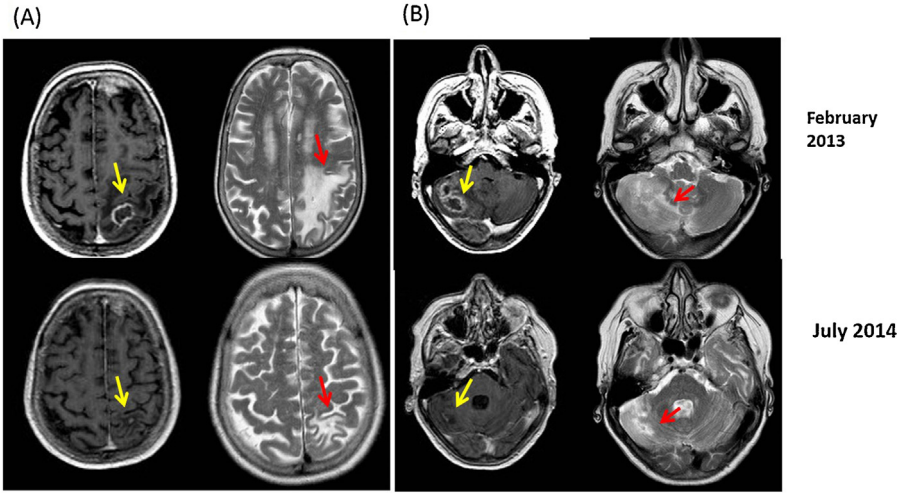

Fig. 1 – Contrast-enhanced (gadolinium) MRI showing SR of cerebral lesions. MRI in February 2013 demonstrated T1-weighted contrast-enhancing lesion (yellow arrow) in left occipital region (A) and right cerebellar hemisphere (B) with corresponding high signal on T2WI (red arrow). Both lesions had regressed on the MRI scan performed from July 2014.

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