Post mortem examination diagnosed *Balamuthia mandrillaris* infection which was confirmed by PCR testing of brain tissue and cerebrospinal fluid.

This report details the neuropathological distribution of parenchymal cerebral necrosis, associated with trophozoite and encysted amoebic forms, which suggests a haematogenous portal of entry for the protozoa into the brain. We present detailed neuropathology findings on the third case of *Balamuthia mandrillaris* encephalitis originating in Western Australia to be reported in the literature.

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FETAL DYSPLASTIC MEGALENCEPHALY WITH ANGIOMATOSIS AND A PATHOGENIC MTOR MUTATION

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Dysplastic megalencephaly (DMEG) is a rare cerebral malformation defined by overgrowth and extensive cortical dysplasia of the cerebral hemispheres, usually due to MTOR signalling pathway gene mutations.

A 22 week gestation male fetus was diagnosed with macrocephaly on ultrasound. Subsequent brain MRI demonstrated features of DMEG, including macrocephaly, ventriculomegaly, polymicrogyria, enlarged corpus callosum, enlarged ganglionic eminences and cortical dysplasia. At 35 weeks gestation, the fetus was delivered by caesarean section with a birth weight of 3945 g (>99th percentile). The head circumference was 41cm (>99th percentile) and brain weight 674.5 g (expected 256 ± 70 g). The neonate died at day one of life.

Massive parallel sequencing on amniotic fluid identified a mosaic missense variant in the MTOR gene: LRG_734t1:c.6644=C>Tp.(Ser2215=Ser2215Phe), confirmed by Sanger sequencing. Neuropathology confirmed the neuroradiology findings. On histology dysmorphic neurones, balloon cells, multifocal nodular neuronal heterotopia and focal leptomeningeal glioneuronal heterotopia were identified. Widespread Sturge-Weber syndrome-like features of cerebral and brainstem leptomeningeal and parenchymal angiomas and cortical microcalcification were present.

We describe an unusual case of dysplastic megalencephaly with a pathogenic MTOR mutation, cortical microcalcification and additional Sturge-Weber syndrome-like features in the brain. The Sturge-Weber syndrome-like features have not been reported in DMEG, expanding the neuropathology phenotypic expression of this disease.

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DIFFUSE IDIOPATHIC PULMONARY NEUROENDOCRINE CELL HYPERPLASIA (DIPNECH) – A CASE REPORT

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Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH) as defined by the WHO is a generalised proliferation of pulmonary neuroendocrine cells that may be confined to airway mucosa or invade locally forming tumourlets or carcinoid tumours.

We report a case of a female aged 82 who presented with an incidentally detected right upper lobe lung lesion, on a surveillance scan following breast cancer. She had an associated chronic dry cough. Her medical history includes childhood asthma. Preoperative lung function tests were normal. Biopsies of this lesion showed a well differentiated neuroendocrine tumour (carcinoid). She underwent a lobectomy which showed one hilar nodule and one parenchymal nodule. Histopathology showed multiple foci of neuroendocrine cell hyperplasia; as well as a 6 mm carcinoid within the bronchial wall at the hilum, 6 mm carcinoid within lung parenchyma and multiple tumourlets. Although uncommon, this is an important entity to recognise as it is usually treated with steroids, and carcinoid tumours that develop are indolent.

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TALL CELL CARCINOMA WITH REVERSED POLARITY – A CASE REPORT

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Tall cell carcinoma with reversed polarity is a rare subtype of invasive breast carcinoma. Immunohistochemical and molecular research show that it is a distinct entity. It is negative for thyroid specific markers.¹ It also has its own unique molecular mutations

(IDH2 hotspot mutations and PI3K pathway mutations).² We present a case of tall cell carcinoma with reversed polarity in the breast of an 81-year-old woman. The lesion showed papillary morphology and columnar cells exhibiting reversed nuclear polarity. Immunohistochemistry showed the tumour cells were positive for CK5/6. They showed weak positivity for GATA3 and GCDFP and were negative for TTF-1. Stains for p63 and p40 showed an absence of myoepithelial cells around tumour nests. The core biopsy showed triple negative hormone receptor status. Further molecular testing was performed which showed a mutation in the PIK3CA gene. An activating mutation in the IDH2 gene was also detected. The combination of morphologic, immunohistochemical and molecular findings are diagnostic of tall cell carcinoma with reverse polarity. It can present a diagnostic challenge when encountered. IDH2 and PI3K mutation testing may provide more confidence with diagnosing these lesions and to help distinguish it from other breast neoplasms.

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TYPE AB THYMOMA WITH AN ATYPICAL TYPE A COMPONENT – A CASE REPORT

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Thymomas are rare tumours of the mediastinum, their cell of origin being thymic epithelial cells, with a variety of presentations and criteria for malignancy. It has always been challenging to classify them appropriately. Atypical type A thymoma has been recently added to the WHO classification (in 2015).¹ There are rare reports of atypical type A thymomas as well as AB thymomas with an atypical A component demonstrating cytologic atypia as well as foci of necrosis and increased mitoses.² We report a case of type AB thymoma with an atypical A component. Our case showed both type A and type B areas as well as an atypical type A component with areas of necrosis and increased mitoses. The case reports reported in the literature show a more aggressive course of atypical type A thymoma in comparison to type A or type AB.³ However, on extensive literature search there was a paucity of articles on atypical type A thymoma, their overall clinical course and prognosis. Further research is needed in this area.

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METASTATIC MEDIASTINAL ATYPICAL MENINGIOMA: A CASE REPORT

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Meningiomas are common neoplasms arising from the central nervous system meninges. Extracranial metastasis of meningiomas are rare and are usually found in less than 0.1–0.2% of cases, though isolated metastasis to the liver and lung have been reported. Because of its rarity and intriguing pathogenesis, we reported here a case of metastatic mediastinal atypical meningioma.

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A CASE OF STEROID RESPONSIVE NIVOLUMAB-INDUCED CHOLANGITIS

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Immune checkpoint inhibitors (ICIs) such as nivolumab are widely used agents in the treatment of a range of advanced malignancies given their demonstrated ability to achieve sustained clinical response and long-term survival in a significant subset of treated patients.¹ ICIs enhance the endogenous anti-tumour immune response and hence are associated with a distinct set of immunologically-mediated toxicities referred to as immune related adverse events (irAEs). ICI-related cholangitis is a rare irAE which currently lacks established diagnostic criteria or treatment guidelines.² Here we present a case of a 75-year-old female with non-small cell lung cancer treated with palliative nivolumab who subsequently developed nivolumab-induced small duct cholangitis which demonstrated an excellent response to corticosteroid therapy. We believe this is the first such reported Australian case. Given previous reports of poor corticosteroid response, more clinicopathological data is needed to characterise the potentially heterogeneous nature of ICI-cholangitis.³ Though a rare phenomenon, given the increasingly common use of these agents, it is an important reaction pattern of which pathologists and clinicians should be aware.

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