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Case report posters

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Ramadan and first presentations of diabetes

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Background

- Hyperglycaemic emergencies in patients with established diabetes fasting during Ramadan have been described.[1-4]
- An association between religious fasting and first presentation of diabetes has not previously been reported.
- We describe three previously well adults admitted with hyperglycaemia and ketosis during Ramadan 2023.

Case 1

- 19-year-old Pakistani man
- 3/7 coryza and dyspnoea
- 6/12 polyuria and 10kg weight loss
- Ramadan fasting for 2/52, stopping 7/7 pre-presentation

Case 2

- 20-year-old Somali man
- 2/7 abdominal pain
- 3/12 osmotic symptoms with 17kg weight loss
- Ramadan fasting for 10/7, stopping 2/52 pre-presentation

Case 3

- 29-year-old Pakistani female
- 6/12 polyuria and polydipsia
- Ramadan fasting at presentation
- History of autoimmune hypothyroidism on 125mcg LT4

No patients recognised their symptoms as related to diabetes

	Case 1		Case 2		Case 3		reference range
BMI (weight)	29.6kg/m2 (108kg)		30.09kg/m3 (103kg)		20.28kg/m2 (53.6kg)		18.5-23.5kg/m2
Family history of diabetes	Yes - Type 2 diabetes		Yes - Type 2 diabetes		No		
pH	7.056		7.196		7.409		7.25-7.45
Bicarbonate	8.6		12.4		23.7		22-29mmol/L
Ketones	4.2		6.7		2.3		<3
Glucose	32		16.8		18.7		3-7mmol/L
C-peptide (paired glucose) at presentation/ at 6 weeks	238 (15.5)	1128 (19.6)	531 (15.2)	Not tested	624 (14.3)	Not tested	370-1470pml/L
Hba1c	125		113		122		<42mmol/mol
Triple-antibodies (anti-GAD) at presentation/ at 6 weeks	49 (anti-GAD <5)	3	2	Not tested	> 1000 (1106kU/l)	Not tested	0-8 units/ml (GAD 0-5ku/L)

Case 1 diagnosis : Ketosis prone Type 2 diabetes presenting in diabetic ketoacidosis (DKA)

Case 2 diagnosis : Ketosis prone Type 2 diabetes presenting in DKA

Case 3 diagnosis : Type 1 diabetes presenting in hyperglycaemic ketosis

Discussion

- The stress of Ramadan fasting, particularly during long summer days, may provoke metabolic decompensation and hyperglycaemic emergencies in known, and undiagnosed diabetes.
- We reinforce the importance of symptom awareness education in at-risk groups, including:
  - those intending to fast
  - adolescents and young adults, in whom diabetes incidence is rising
  - those with a family history of diabetes and ethnic minority groups

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The New Great Mimicker: A Multi-speciality Condition

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Case Report

A 67-year-old man of Chinese descent was admitted to the hospital with painless jaundice, fever, and a weight loss of 6kg over four weeks. He denied using any new medications, herbal remedies or steroids. He had no significant medical or family history, except for well-controlled hypertension.

On Examination

Deeply Jaundiced with no signs of chronic liver disease. No hyper/hypopigmentation.

BP: 106/78 mmHg, HR: 78/min. No goitre or visual field defects on confrontation.

Initial Biochemistry

An obstructive jaundice picture with a normal inflammatory marker was present.

He was tested for hepatitis, HIV, Syphilis, and TB which were all negative.

Radiology

- Initially, abdominal ultrasound was performed, which showed cholelithiasis with intra and extrahepatic biliary duct dilatation and a complex liver cyst.
- He then had a staging CT-TAP, which revealed enlarged, haemorrhagic mediastinal and bilateral hilar lymph nodes, intrahepatic and CBD duct dilatation, pericardial and pleural thickening, as well as the presence of a 10mm left supraclavicular lymph node. Widespread metastasis from an unknown primary was suspected. Thus, a PET CT scan was recommended.
- PET CT confirmed CT findings of multi-system involvement, as shown in Figure 1.

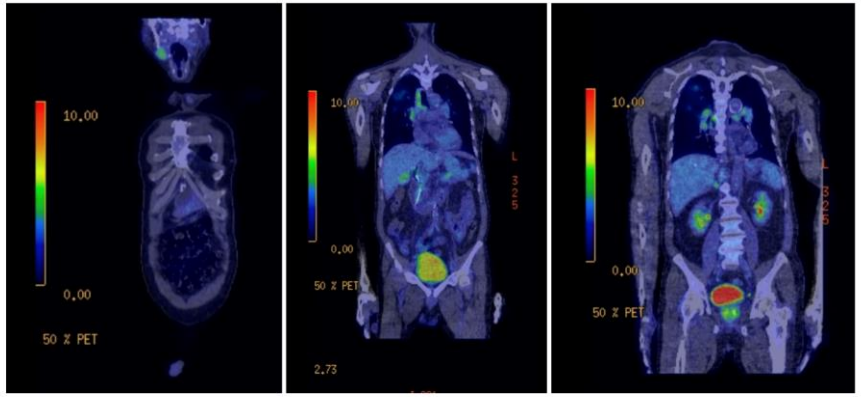

Fig.1: PET scan showing (a) enlarged right submandibular gland, (b) enlarged intrathoracic lymph node gallbladder (c) right adrenal and prostate

Case Progress

- The presence of Adrenal incidentaloma led to adrenal function testing, which showed a 9 am cortisol of 30nmol/L with raised potassium of 4.5mmol/L. He was immediately commenced on hydrocortisone replacement.
- A pituitary blood panel and an MRI were organised. His pituitary blood suggested panhypopituitarism (TSH:0.24mIU/L, fT4:<5.5 pmol/L, fT3: 1.9 pmol/L, LH: <0.2 IU/L, FSH: 0.3IU/L, Testosterone: <0.5nmol/L, Prolactin:587mIU/L, ACTH: 18ng/L, Urine Osmolaloty:378mosm/mol, Serum Osmolality: 305mosm/mol).
- The MRI (Figure 2) showed the presence of a thickened infundibulum and a bulky pituitary, suggestive of Hypophysitis.

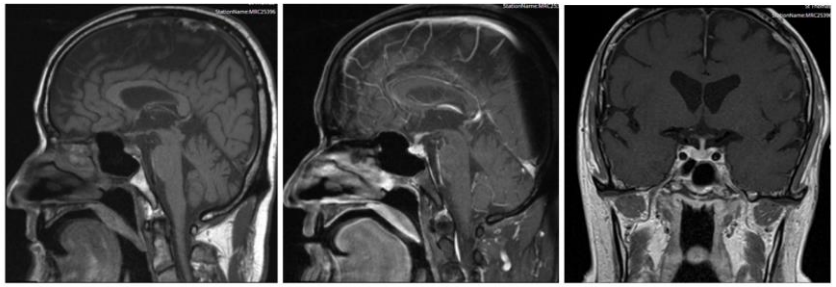

Fig.2: Pituitary MRI showing thickened infundibulum and a bulky pituitary (a) Sagittal section-T1WI, (b) Sagittal section-T2WI, (c) Coronal section

- He went on to have an EBUS for the supraclavicular Lymph node.
- Initial report suggested: Non-necrotising granulomatous lymphadenitis.
- Histopathology was reported as: Clumps of plasma cells show more than 90% of IgG-positive plasma cells also positive for IgG4, with a maximum count of approximately 60 IgG4-positive plasma cells per high-power field.
- His Immunoglobulin levels were checked (Table 1):

Name of the Test	Results	Reference range
Immunoglobulin G level	36.60	5.40-18.22g/l
Immunoglobulin A level	1.28	1.01-6.45g/l
Immunoglobulin M level	0.24	0.22-2.40g/l
Immunoglobulin G1 level	8.9	3.8-9.3g/l
Immunoglobulin G2 level	5.2	2.4-7.0g/l
Immunoglobulin G3 level	2.0	0.2-1.8g/l
Immunoglobulin G4 level	16.15	0.04-0.86g/l

Management

He was commenced on high-dose steroids and levothyroxine.

Follow-up under Rheumatology and Endocrinology was organised.

Discussion

This case reflects the diagnostic challenges faced while investigating a not-so-well-known or understood multi-system condition like IgG4-related disease, which can mimic disseminated malignancy.

A diagnostic criterion has been proposed by Umehara et al. for IgG4 RD, which predicts the probability of the presence of the disease based on the following criteria:

1. Characteristic symptoms of organ affection (in the form of diffuse or tumorous enlargement) or organ disorder.
2. Elevated IgG4 levels in serum (> 135 mg/dl)
3. Lesions observed in histopathological tests:
   - Lymphoplasmacytic infiltration and fibrosis.
   - Infiltration of IgG4-positive cells > 10 IgG4-positive cells in the field of vision in a high-definition microscope and with IgG4+/IgG ratio > 40%.

The diagnosis is inevitable if all three criteria are present, probable if criteria 1 and 3 are present, and likely if criteria 1 and 2 are present.

A varying degree of anterior and posterior pituitary involvement is noted in IgG4-related Hypophysitis. Early initiation of immunosuppression prior to setting in of fibrosis has been associated with the reversibility of hormonal insufficiencies. Focal adrenal lesions regarded as IgG4-related are uncommon.

Systemic glucocorticoids are currently the first-line approach for IgG4-RD.

For relapsed IgG4-RD, adding immunomodulatory drugs such as azathioprine and B-cell depletion with rituximab is effective.

Summary Points

- A high suspicion of a multi-system granulomatous condition like IgG4 is essential while investigating patients with disseminated lesions.
- Commoner conditions like malignancy lymphoma and systemic infections like Tuberculosis and Syphilis need to be ruled out before a diagnosis of IgG4 RD.
- The 1st line of treatment is steroids. However, increasingly, steroid-sparing agents are being used, such as rituximab.

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A rare case of slowly progressive fatal pneumonia following oral dexamethasone therapy

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INTRODUCTION

Corticosteroids are routinely used in the management of cerebral oedema in patients with intracranial lesions. Side effects with corticosteroids are common including infections due to immunosuppression. [1]

Nocardia is a gram-positive, partially-acid fast, bacillus that forms branching filaments. Nocardiosis is a rare yet life-threatening, opportunistic infection that can have devastating effects. We report a case of slowly progressive pneumonia following 3 weeks of oral dexamethasone treatment.

CASE SUMMARY

- A 86-year-old Caucasian man presented to the emergency department with collapse. He had history of multiple cancers including prostate, urinary bladder and most recently lung cancer which was managed by lobectomy.
- CT scan showed a lytic lesion on the skull involving the brain suspicious for metastasis (Figure 1).
- Patient improved quickly with conservative management and was sent home with oral dexamethasone to treat any possibility of cerebral oedema pending MRI report.
- Patient represented to emergency 6 weeks later with a 3-week history of insomnia, loss of appetite, oral thrush and cough with productive sputum.
- After reviewing his previous MRI from a different hospital record, his skull lesion was diagnosed as a congenital defect and a decision to wean off dexamethasone was taken.
- Patient's hospital course was complicated by a community acquired pneumonia treated with intravenous co-amoxiclav for a week with no significant clinical response.
- Antibiotics were changed to piperacillin-tazobactam and fluconazole for further 5 days untill the blood culture grew a branching gram-positive filamentous bacillus, identified subsequently as Nocardia farcinica, a rare cause of nocardiosis.
- Unfortunately, patient died of respiratory complications within 6 hours of positive blood culture before appropriate treatment for nocardiosis could be initiated.

FIGURES

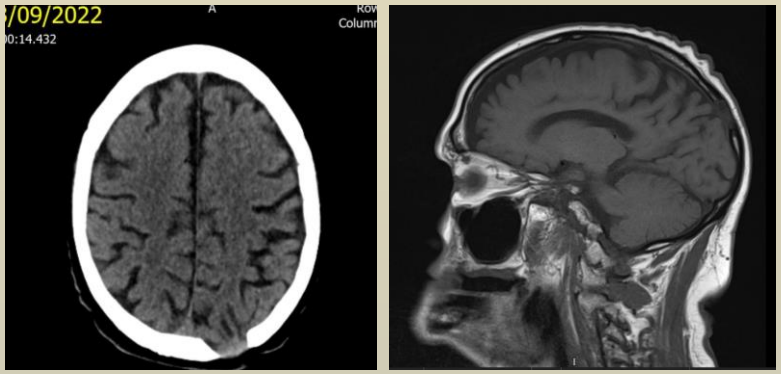

Figure 1. Brain MRI showing bony defect in the left paramedian occipital skull vault.

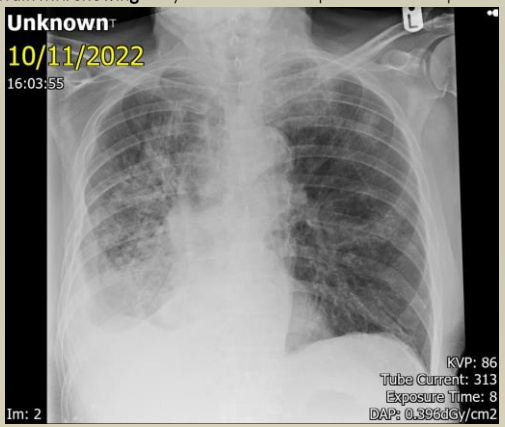

Figure 2. Chest radiograph showing consolidation in the right hemithorax.

Figure 3.

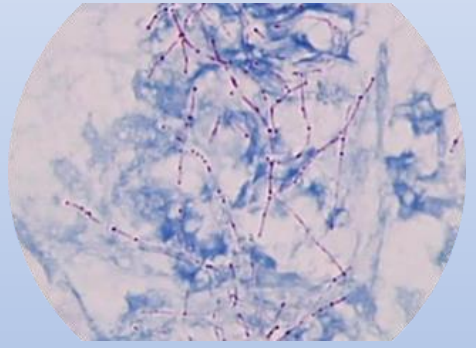

Figure 4.

DISCUSSION

- Thorough history taking is essential to make the correct diagnosis.
- Pulmonary nocardiosis lacks specificity and may remain cryptic but progressive in its course, which makes early diagnosis difficult.
- Nocardiosis should be considered in cases with slowly progressive pneumonia which remain clinically unresponsive to standard antibiotics.
- Microbiology team should be involved early for diagnostic and therapeutic intervention of unresolving pneumonia in immunocompromised patients.

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Head-scratching and Headlines:

A Rare Case of Bilateral Anterior Uveitis with Multi-Systemic Involvement

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History

42 year-old caucasian presents to eye casualty with an acute history of redness, pain and photophobia in both eyes

Systems review:

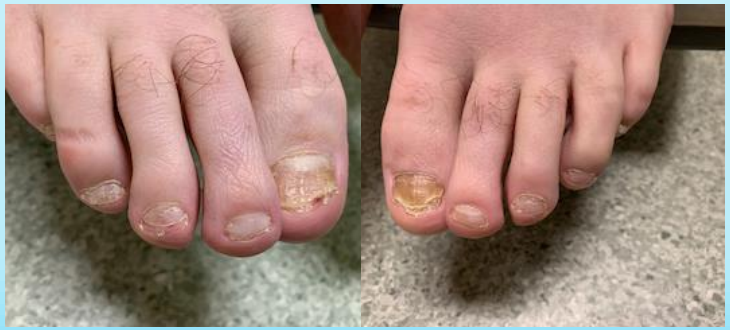
- Malaise
- Fever
- Pharyngitis and cough
- Tender neck lumps
- Polyarthralgia
- Truncal rash

He has already by seen by GP and medical take

Examination

Slit lamp examination confirms bilateral anterior uveitis (keratiic precipitates and anterior chamber cells)

Systemic examination is unremarkable apart from…



TYPE TO ENTER A CAPTION.

TYPE TO ENTER A CAPTION.

Investigations

Chest x-ray is normal however blood tests are not…

Blood test	2 Dec (GP)	4 Dec (GP)	8 Dec (GP)	12 Dec (GP)	5 Jan (me)
Hb	150	142	131	135	142
WCC	13.2	11	11.7	9.8	8.5
Plt	243	330	400	501	299
Neut	7.7	6.0	7.7	6.5	5.9
CRP	203	177	127	77	3
Cr	114	100	144	116	86
Alb			27	29	
IgG				21.1 (6-16)	
IgA				6.0 (0.8-2.8)	

Diagnosis

- Differentials include TB, sarcoidosis, Behcet's, ankylosing spondylitis, reactive arthritis, enteropathic arthritis and psoriatic arthritis etc.
- Psoriatic arthritis was my preferred diagnosis because of onychodystrophy and rash
- However AKI is surprising ?intrinsic renal pathology ?post-Streptococcal glomerulonephritis
- **Anti-Streptolysin O antibody 800 (<200)**
- Post-Streptococcal uveitis syndrome (PSUS) is a rare but recognised phenomenon[1-5]

Reflections

The eye is connected to the rest of the body

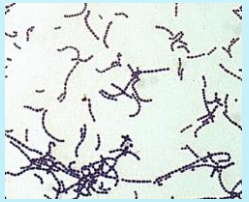
- An important reminder for both ophthalmologists and physicians

Beware of positive bias and red herrings

- Desire to diagnose psoriatic arthritis and fixation on toenails initially blinded me from considering other diagnoses
- Taking a step back to think laterally (e.g. remembering headlines about 'Strep A') can be helpful

Medicine is not a game, don't forget the patient

- Diagnostic uncertainty is usually not fun for the patient
- Importance of recognising limits of clinical expertise (?rheumatic fever ?rheumatic heart disease)



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Neuropsychiatric Lupus: Report Of An Interesting Case With Challenges In Diagnosis And Management

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INTRODUCTION

- Neuropsychiatric Systemic Lupus Erythematosus (NPSLE) is characterized by neurological and psychiatric manifestations.
- Its early identification, even for skilled physicians, can be challenging because it can present with negative serology, absence of systemic signs and symptoms, and is usually a diagnosis of exclusion.[2]
- This case was fascinating because attribution of a broad range of systemic signs and symptoms and altered behavior to SLE was not made, leading to delayed initiation of treatment and poor outcomes until she presented to our hospital.

MATERIALS AND METHODS

- Antinuclear antibody, SS-B autoantibody, anti-Smith antibody, and anti-double stranded DNA antibody tested positive. Complement C3 levels were low. Radiological scans indicated mild pleural effusion on both lung fields. [Figure 1].
- Vitals and Laboratory

Heart rate	**High**
Hb	Low
Platelets	Low
Creatinine and Urea	High
ESR and CRP	High
Proteinuria	≥3.5g/day

RESULTS AND DISCUSSION

- Given a multisystem involvement and altered sensorium, in the context of negative tests for infections and, metabolic causes, an Electroencephalogram (EEG) was performed, which did not reveal any epileptiform discharges.
- The aetiologies like CNS vasculitis, systemic diseases like granulomatosis with polyangiitis, Sjogren Syndrome, Neuro-Behcet's disease, viral infections, Hashimoto encephalopathy, Autoimmune encephalitis, and disseminated TB were excluded.

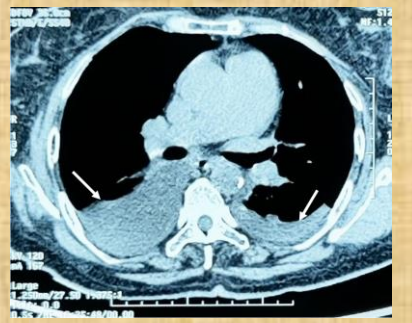


Figure 1

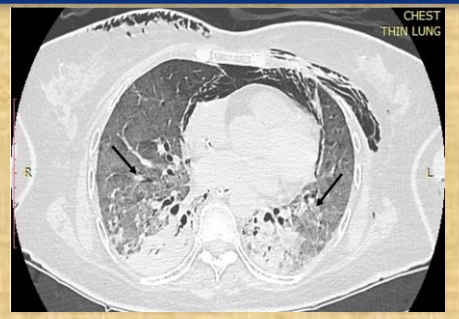
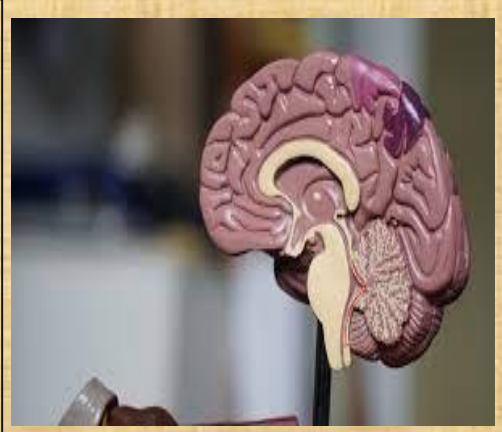


Figure 2

- NPSLE was diagnosed based on clinical features and labs.
- She was given **intravenous methylprednisolone (1000 mg daily for five days) and oral mycophenolate mofetil (1g per day)**
- After consequent doses of methylprednisolone, serum creatinine improved, and a decline in urine proteins was noted, but her psychosis persisted.
- She was started on oral prednisolone, and a renal biopsy was planned but had to be deferred as the patient sadly succumbed to COVID-19 pneumonitis during hospital illness (Figure 2)

CONCLUSION

- Thus, in a patient presenting with altered mental status and **psychosis of unexplained etiology**, high clinical suspicion for Lupus, along with a detailed and competent history and explicit workup, can aid solving this mystery.



Subclavian Steal Syndrome -- An Underappreciated Cause Of Neurological Symptoms

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SUMMARY

Subclavian steal syndrome (SSS) refers to the association of symptoms related to vertebrobasilar insufficiency and the phenomenon of subclavian steal. We report a challenging case of a 61-year-old female that presented with Posterior Circulation ischaemic stroke secondary to subclavian steal syndrome.

BACKGROUND

Subclavian Steal Syndrome is a vascular disorder characterized by a significant stenosis or occlusion of the subclavian artery proximal to the vertebral origin, resulting in retrograde flow in the ipsilateral vertebral artery. Most subclavian artery stenoses are asymptomatic because of the abundant collateral blood supply to the arm that will usually ensure adequate circulation with demand. The symptoms arise due to two types of mechanisms by which the arm "steals" blood flow from the vertebrobasilar territory; 1) a lack of blood supply because of subclavian artery stenosis or 2) rarely malformation disease, that may include an arteriovenous distal arm shunt.

The incidence or prevalence of subclavian steal syndrome is between 0.6% to 6.4%. The Joint Study of Extracranial Arterial Occlusion by Fields et al., showed a 2.5% incidence (168/6534), with only 5.3% of these patients experiencing neurological symptoms. Males are more affected compared to females, due to atherosclerotic causes, by a ratio of about 2 to 1.

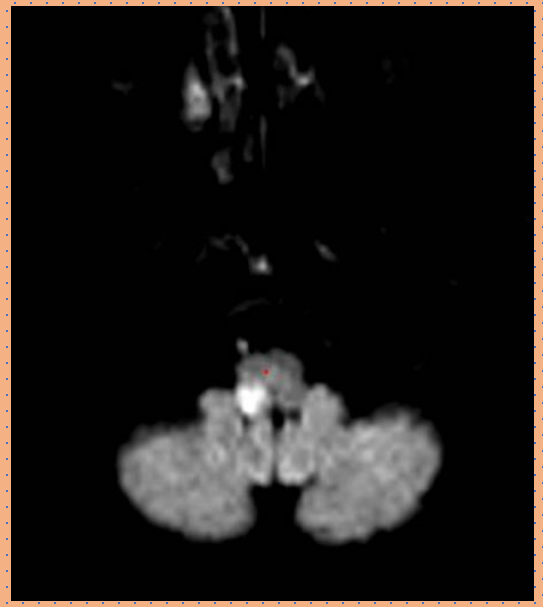
CASE PRESENTATION

A 61-year-old female was admitted initially with sudden onset of dizziness, light headiness with nausea and vomiting as a possible ischaemic stroke. Her medical history included CKD stage 3 and longstanding hypercholesterolemia and was known to have asymptomatic stenosis of her subclavian artery . The patient denied any history of chest pain, palpitations, visual changes, nausea, fever or aura. On further discussion patient reported progressive symptoms of left arm numbness and intermittent episodes of syncope over last few months

On physical examination, her blood pressure was 177/76 mmHg (right arm), left radial pulse was palpable but feeble, following which BP was recorded in the left arm which was found to be markedly low at 123/83 mmHg. Other clinical findings, laboratory investigations, ECG and chest X-ray were unremarkable. Given her presentation, the patient was alerted as stroke with NIHSS 16 on admission and underwent a Computed tomography (CT) of her Head and a CT angiogram of her carotid vessels. CTA showed an acute complete occlusion of V3 and V4 and right vertebral artery; partial occlusion of V4 of left vertebral artery and mid basilar artery. Foetal origin of the right PCA. Poor flow of P3 segment and beyond of right PCA. CTA also showed near-complete occlusion of the left subclavian artery origin, proximal to the vertebral artery origin which was likely chronic. MRI co-related right PICA territory in the right lateral medulla and inferior cerebellum in keeping with hypo perfusion.

TREATMENT

She had increasing frequency of syncopal episodes with longer durations as an inpatient after being treated for her stroke. She was discussed in neuroradiology and vascular multidisciplinary meetings. It was identified that the left vertebral artery branches after the subclavian stenosis which was likely responsible for these events as was the sole supply for the posterior circulation. This corresponded with all events on vomiting or on moving left arm. These findings were discussed and a diagnosis of subclavian steal syndrome was made. The patient underwent stenting of subclavian artery to improve cerebral blood flow with angioplasty. The procedure was well tolerated and immediately afterwards, there was reduction of the symptoms. We propose that percutaneous transluminal angioplasty with stenting placement is a good therapeutic option for subclavian steal syndrome.

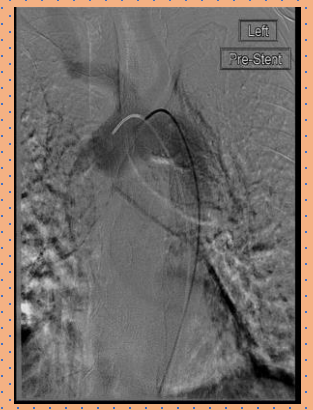


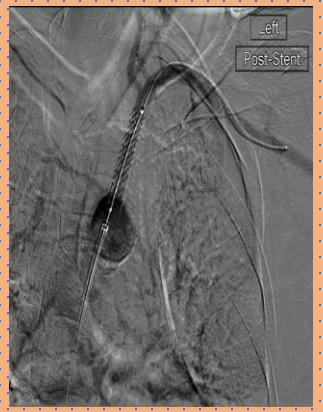
DISCUSSION

SSS is relatively rare condition. Atherosclerosis is the most common cause (95%).

We used a combination of duplex scan, CTA, and angiography to assess the proximal subclavian stenosis. Duplex scan allows the classification on the degree of hemodynamic disturbances of the vertebral artery: stage I (Occult steal, decreased blood flow), stage II (partial steal, transient or partial reversal of flow), and stage III (complete steal, permanent reversal of flow). Invasive treatment is indicated in symptomatic patients. Options include axillo-axillary bypass, carotid-subclavian bypass, and percutaneous transluminal angioplasty (PTA) of the stenotic proximal subclavian artery with stent placement. Bates *et al.* reported a 76% 5-year survival in a large case series of patients who underwent subclavian artery stenting.

We propose that percutaneous transluminal angioplasty with stenting placement is a good therapeutic option for subclavian steal syndrome. Repeat CTA showed further recanalization of the right vertebral segments compared with the previous scan. There was a significant improvement in symptoms and substantial decrease in the frequency and length of syncopal events.





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A Case Of ANCA-associated Vasculitis With Probable IgG4-related Disease

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Frimley Health NHS Foundation Trust

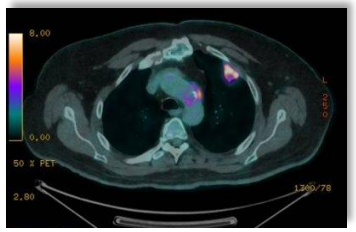
Background: IgG4-related disease (IgG4-RD) and granulomatous polyangiitis (GPA) have different pathology, although may respond to the same treatment. There are infrequent reports of the two conditions occurring simultaneously.

Case: 65-year-old male
PMH: T2DM, CABG and ablation for AF.
Presentation in October 2022: low back pain radiating to the perineum.

Investigations:

CRP	102 mg/ml
eGFR	>90 ml/hr
cANCA	1/40 Positive
PR3	32 U/ml (N 0-1.9 U/ml)
IgG4	2.2 g/L (N 0-1.3g/L)
Other Immunoglobulins	normal
IGRA	negative
Lung nodule biopsy – December 2022	necrotising granulomatous inflammation and surrounding palisading histocytes, with no vasculitic element

Radiology:

CT urogram	possible aortitis
CT aortogram	a cuff of added tissue around the infrarenal aorta and iliac arteries
CT whole aorta	• increase in the periaortic abnormal changes • a left upper lobe cavitating lesion
PET CT November 2022	• Increased FDG activity in the infrarenal aortic tissue, with focal uptake in the aortic arch and right superficial femoral artery. • Several pleural based and parenchymal lesions showing moderate increased FDG activity were seen  
CT sinuses	multifocal sinonasal mucosal thickening without bony erosions

Treatment And Progress:

December 2022
Prednisolone 40mg/daily Methotrexate 15mg/weekly

Quick symptomatic improvement

February 2023
CRP 14 mg/ml

The national IgG4 MDT: He had features of both GPA and IgG4-RD

March 2023
follow-up CT scan: reduction in the volume of para-aortic tissue, with regression of some lesions in the chest, while others increased

remains well with normal CRP, stable renal function and normal urinalysis

September 2023
IgG4 0.67 g/L

Discussion: The ANCA positivity and lung biopsy findings satisfy GPA classification criteria. While aortic involvement is not commonly seen in GPA; it is typical of IgG4-RD; however, its diagnostic criteria require a confirmatory biopsy. This can be challenging due to the risk of sampling around the aorta and ureters. Our case would fulfil criteria for 'probable' IgG4-RD. In retrospect, an earlier PET CT scan may have spared the patient several CT scans.

Conclusion: 1. The co-existence of IgG4-RD and ANCA-associated vasculitis may be more common than might be expected by chance. **2.** The usefulness of early PET CT scanning in rheumatic conditions. **3.** Checking ANCA is important in suspected IgG4-RD. **4.** Future AI programmes may help clinicians better judge the timing of investigations that may be more expensive, invasive or have increased radiation exposure in such complex cases. **5.** The usefulness of a national MDT for IgG4-RD. **6.** Treatment modalities are similar for both conditions, with rituximab being used as a second line agent.

References

SCAN ME

The Conundrum Of Low To High Sodium- Unmasking Diabetes Insipidus In A Small Cell Lung Cancer Patient.

Hira Yousuf ; Nick Brown; Ersan Alzamel; Jenny Jones; Ashishkumar Ihare

Introduction :

Association of Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) with small cell lung carcinoma is not uncommon, however, pituitary metastases causing diabetes insipidus concomitantly is extremely rare. There are only few cases reported in the literature [1,2]. We report an extremely rare case of Diabetes insipidus secondary to synchronous pituitary metastasis, at the time of new presentation of metastatic small-cell lung carcinoma.

Presentation:

A 59-year-old Slovakian male was admitted with a one-week history of facial swelling. Chest X-ray showed mediastinal lymphadenopathy in the right suprahilar region, and subsequent CT TAP (computerised tomography: Thorax-abdomen and pelvis) revealed enlarged lymph nodes in mediastinum and right hilar region causing compression of the superior vena cava, enlarged adrenals, inguinal nodes, and a head of pancreas lesion. Left groin biopsy confirmed small cell carcinoma of lung origin. Pre-treatment magnetic resonance imaging (MRI) of his head (figure- 1 A) showed a suprasellar tumour with embolic lacunar infarcts.

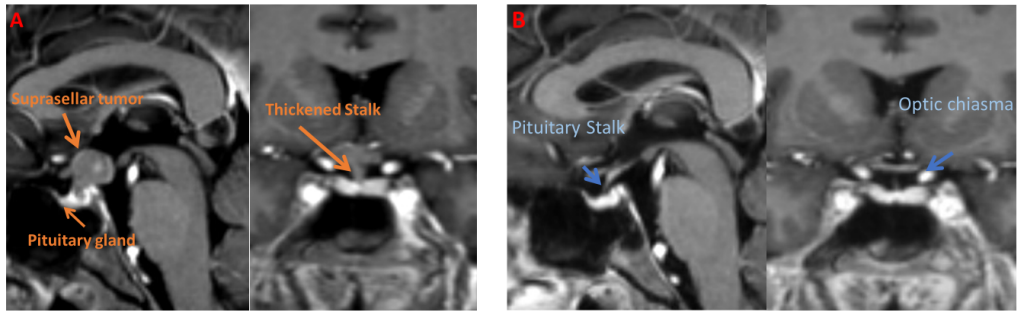


Figure 1 A Pretreatment T1 sagittal and coronal postcontrast images Suprasellar solid tumor engulfing pituitary stalk and causing mass effect on optic chiasma. B Post-treatment T1 sagittal and coronal postcontrast images shows complete resolution of suprasellar tumor with normal optic chiasma and midline pituitary stalk

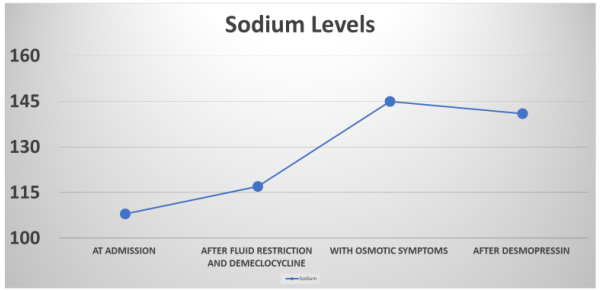
On admission, he was found to have a low Sodium of 108 mmol/L; further work-up for hyponatremia (as shown in Table-1) confirmed he had SIADH.

Test	Patient	Normal Range
Sodium	108	133-146 mmol/L
Urea	3.8	2.5-7.8 mmol/L
Creatinine	53	48-128 µmol/L
TSH	3.4	0.2-4.0 mU/L
Cortisol	411	184-623 nmol/L
Urinary Sodium	135	No measured vaules
Urinary Osmolality	652	50-1200mosmol/kg
Plasma Osmolality	233	275-295 mosmol/kg
ECHO: Normal Left ventricular function. No wall motion abnormalities		

Table-1: Workup of Hyponatremia

He was fluid restricted and commenced demeclocycline as advised by Endocrinology. The skull base multidisciplinary team (MDT) ruled out surgery to the pituitary lesion. He was offered the first cycle of carboplatin and etoposide whilst an inpatient, and his Sodium improved to 117mmol/L (graph 1) on discharge.

He was seen in the clinic after discharge and immunotherapy with atezolizumab was added as his performance status improved. Four weeks later, there were new symptoms of excessive thirst, dry mouth, polyuria, polydipsia, and his Sodium level was 144mmol/L. Work-up(table 2) by the endocrinology team confirmed diabetes insipidus, and the patient was commenced on desmopressin.



Graph-1 :Shows sodium levels during the course of treatment.

Test	Patient	Normal Range
Sodium	144	133-146 mmol/L
Urinary Osmolality	101	
Plasma Osmolality	297	275-295 mosmol/kg

Table -2 Workup of Diabetes insipidus

After treatment with desmopressin, there was an improvement in osmotic symptoms and normalization of sodium (Graph 1). His cancer showed an excellent clinical and radiological response after completion of chemotherapy and immunotherapy (figure- 1 B) and he remains well with normal sodium levels.

Learning Point:

This patient developed diabetes insipidus, most likely due a loss of normal ADH production caused by the effect of metastatic disease upon the hypothalamic-pituitary axis. This was masked on presentation, however, by inappropriate secretion of ADH by his small cell lung cancer. Once the small cell lung cancer was controlled by chemotherapy, inappropriate secretion of ADH was reduced, and the symptoms of diabetes insipidus became apparent. Clinicians should be aware of the potential for this to occur during treatment in patients with SIADH on presentation, and metastatic disease (or other conditions) that potentially affect the hypothalamic-pituitary axis. Our case presentation adds value to the existing literature that this possibility, though rare, should be considered especially when diabetes insipidus could be masked by the presence of SIADH and it can manifest as hypernatremia once the primary tumour responds to the treatment.

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Case report: Close Eye Hallucinations and Acute Occipital Infarct

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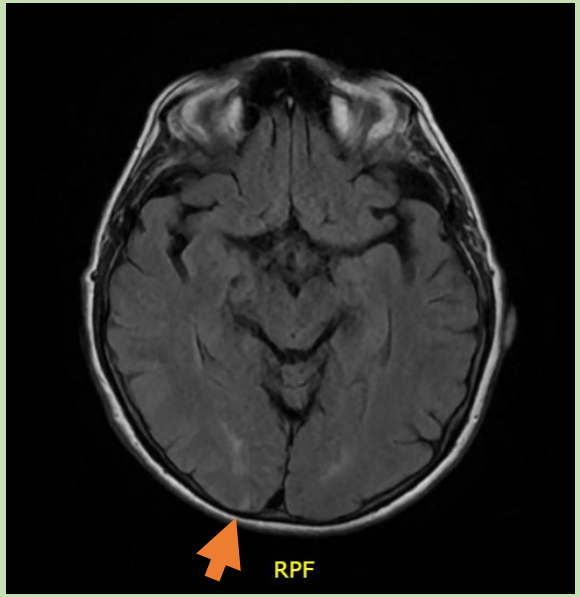
Introduction and Background

- Release visual hallucinations are associated with cerebrovascular events affecting the visual cortex which causes disinhibition of earlier visual areas thereby resulting in spontaneous firing. Most hallucinations in such setting are usually associated with concomitant visual impairment or visual field defects. They can present as simple (phosphene) or complex hallucinations. [1,2]
- These phenomena are sometimes not recognised by the clinicians and may be misdiagnosed as psychosis or early dementia.[1,3]
- We would like to highlight the following case with visual hallucinations without any other associated visual impairments, which made the diagnosis challenging.

Case summary

- 75-year-old lady with history of arterial hypertension and anxiety disorder
- Three day history of sudden onset of complex visual hallucinations (shapes, patterns, letters), immediately on closing her eyes. No focal neurological features or visual field defect.
- Initially seen by ophthalmology. No localised cause was found, hence referred for medical review.
- Patient's concerns:
  - Insomnia due to closed eye hallucinations
  - Distress regarding stigma of hallucinations and associated psychosis
- MRI brain: a tiny focus of restricted diffusion in the right occipital lobe in keeping with acute ischaemia
- Secondary prevention for stroke was commenced. Explanation and reassurances were provided to address her concerns.
- She was referred to mental health liaison team on a subsequent follow up visit in view of her anxiety after the event.



MRI T2 flair sequence showing a tiny ischaemic focus in the right occipital lobe (arrow)

Discussion and Learning points

This case highlighted

- the importance of clinicians to recognise that ***isolated visual hallucinations*** can represent ***systemic*** conditions like stroke, however rare it could be. The absence of typical features of a stroke can make the diagnosis challenging and a ***delay in reaching a definitive diagnosis*** can compound the ***patient's anxiety.***
- that subtle symptoms like hallucinations could be distressing for the patient and can exert negative impact on their quality of life.[1,4] It is essential that clinicians appreciate this and address the ***mental health*** *of their patients.*

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Acute Adrenal Insufficiency Secondary to Bilateral Adrenal Infarction in a Postpartum Woman with Antiphospholipid Syndrome

JE Manning[1,2] & DJ Arachchillage[1,2]

Introduction

Antiphospholipid syndrome (APS)[1] is an acquired, autoimmune prothrombotic disorder characterised by:

Thrombosis

AND/OR

Obstetric Morbidity

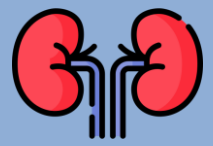
aPL Antibodies

≥2 occasions
≥12 weeks apart

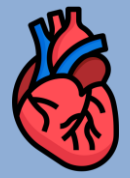
In association with multi-system manifestations:

Seizures, white matter Lesions

Nephropathy



Valvulopathy Vegetations



Livedo Ulcers

Not so 'rare' since antiphospholipid antibodies present in up to:[2]

~14% ischaemic strokes
~10% deep vein thrombosis & myocardial infarctions
~6% of women with recurrent pregnancy morbidity

Case Presentation

Severe back pain + confusion
1/52 post emergency CS
For PET + HELLP syndrome

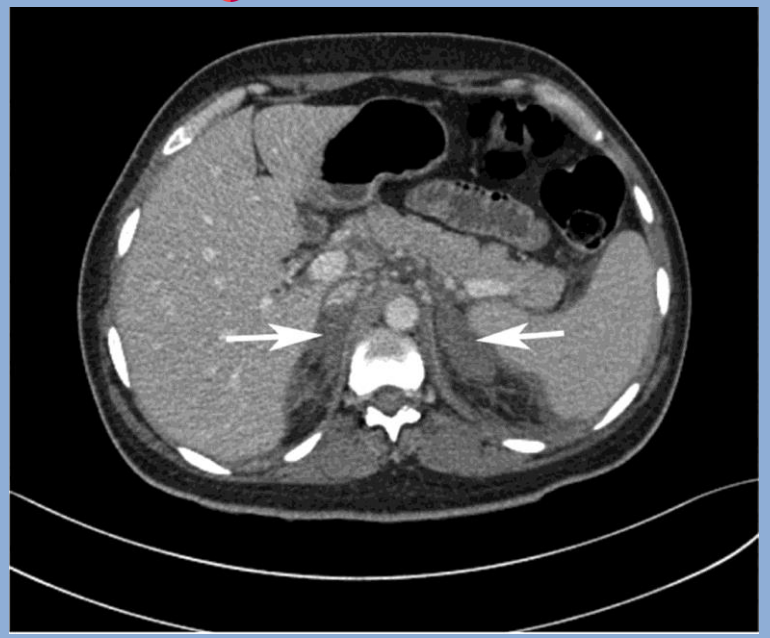
PMHx

'triple positive' APS

DHx

Px LMWH
(Tx dose during pregnancy)

Na 116 mmol/L



Case Outcome

Transferred to 3º haem/obstetric medicine centre

IV Steroid replacement -> oral switch
Tx LMWH
Hydroxychloroquine (immunomodulation)

Biochemistry normalised. Confusion resolved

Home with plan for Warfarin bridging & endo F/U

Learning Points

Patients with APS present to the general medical take – a working knowledge of the condition is vital

Patients with APS are very high risk for the development of thrombotic complications – care when prescribing anticoagulation

DOACs are inferior to warfarin in many APS patients

A multidisciplinary approach to care is often needed

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A Deceitful Case of Acute Coronary Syndrome

Jayden Patel[1], Tha Nyi[1], May Ohn[2], Rupert Williams[1,2]

[1] Cardiology department, Kingston Hospital NHS Foundation Trust [2]Cardiology department, St George's University Hospitals NHS Foundation Trust

Introduction

- Coronary artery embolism is an acknowledged nonatherosclerotic cause of acute coronary syndrome (ACS). Although the diagnosis is challenging in the acute setting, it is important to consider it in the context of relevant risk factors and clinical presentation.

Case presentation

- A 63 year-old man presented with a 6-week history of fevers, night sweats and confusion on the background of a recent tissue aortic valve replacement.
- He was treated for sepsis of unknown origin however he developed septic shock and complicated with ACS.
- He developed new ST elevation in the inferior leads and first-degree heart block on his Electrocardiogram (ECG).
- He was transferred urgently to a tertiary centre for management of possible aortic root abscess.
- Trans-esophageal echocardiography (TOE) confirmed a large aortic valve vegetation, and aortic root abscess (Figure 1).
- Angiogram demonstrated occluded proximal diagonal branch of left anterior descending artery (LAD) ,severe proximal obtuse marginal (OM) disease and occluded left posterior descending artery (LPDA) (Figure 2).
- He underwent emergency salvage valve replacement but despite cardiopulmonary bypass and intra-aortic balloon pump, he passed away in theatre.

Discussion

- Coronary artery embolism is a rare cause of type 2 myocardial infarction.
- In a case series by Roux V et al, 2.2% of 1210 patients with IE developed ACS and 88% of ACS were caused by coronary embolism. Those complicated by ACS had higher risk of developing heart failure (2.5 times) and the mortality rate was twice higher compared to their counterparts (1).
- The mechanism of infarction can also be due to external compression of the coronary artery ostia by an aortic root abscess (2).
- Prosthetic valve endocarditis (PVE), accounts for approximately 20% of all endocarditis cases with only <1% developing septic emboli (3).
- Development of an aortic root abscess is both rare and a poor prognostic marker with the mainstay of treatment being via emergency surgery.

Conclusion

- This case highlights the importance of having a high suspicion of PVE in patients with previous valve surgery.
- PVE complicated by ACS is frequently due to direct coronary embolism of a vegetation.

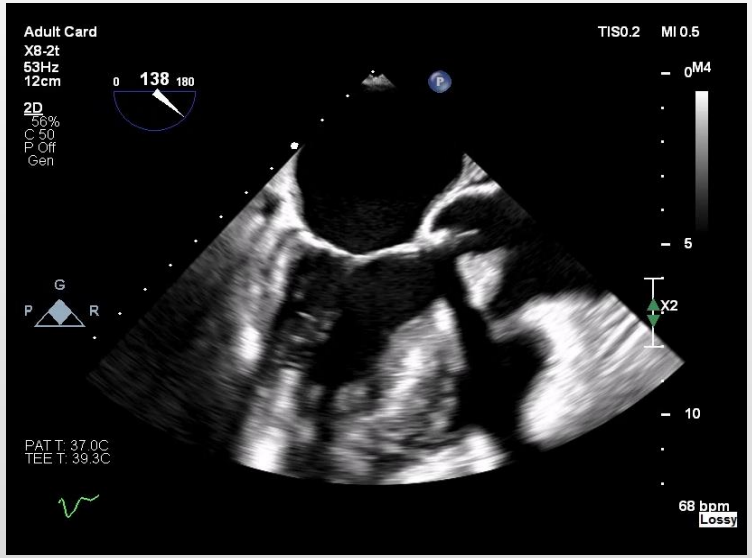

Figure 1. TOE showing presence of large aortic valve vegetation

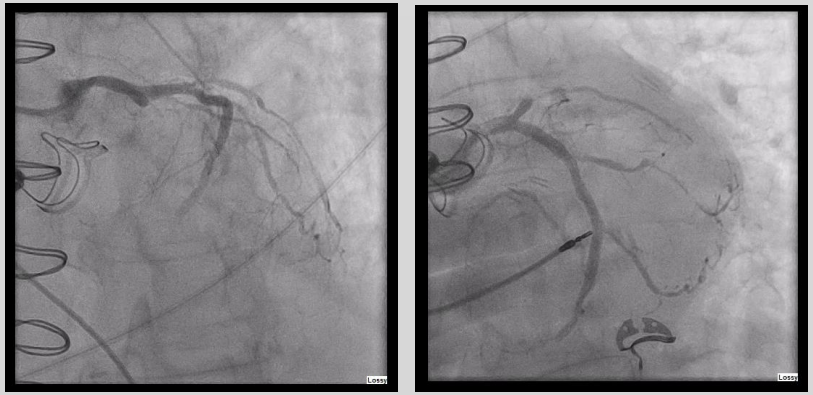

Figure 2. Coronary Angiogram demonstrating occluded proximal D1 of LAD, severe proximal OM disease and occluded LPDA

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A Complex Presentation Of Melioidosis: Uncovering Acute Osteomyelitis

Presenting Author: Dr Joshlin Maria Shaji, Internal Medicine Trainee
Co-Authors: Dr Anil N.X, Dr Anup R Warrier, Dr Gladson C.J, Aster Medcity, India

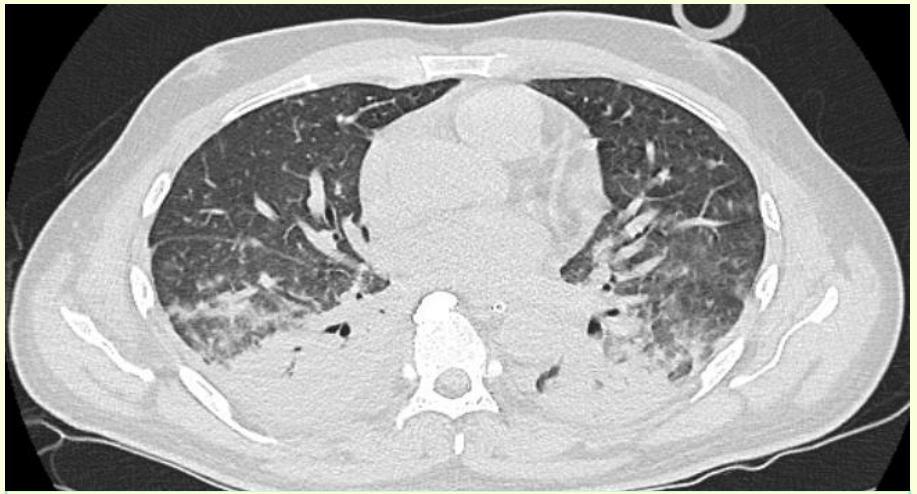
INTRODUCTION

- **Burkholderia pseudomallei** is a gram-negative bacterium that causes melioidosis, a serious disease with a wide range of clinical manifestations ranging from localised subacute infections to septicaemia with abscesses in multiple organs, pneumonia, sepsis or death.
- It is found in soil and water.
- Humans are infected by inoculation, inhalation or ingestion.
- Delayed diagnosis contributes to increased mortality and morbidity.

CASE REPORT

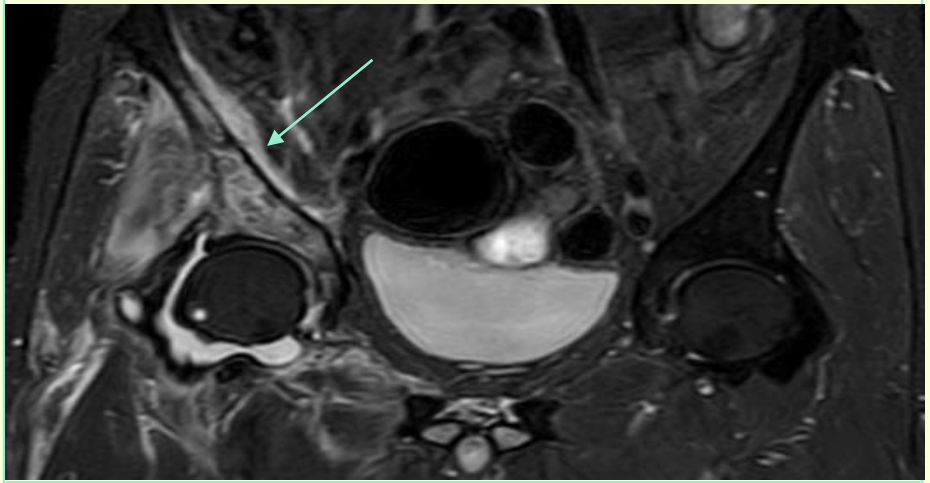
- A 51-year-old **non-diabetic** male presented with a 2-week history of fever with chills.
- O/E: Temp: 101°F, HR: 126/min, BP: 90/70 mm Hg, RR: 48/min.
- Auscultation: Bilateral basal crepitations.
- Labs: Total count: 2.4 K/uL, Hb: 16.3 g/dl, Platelet: 95 K/uL, **CRP: 490 mg/L**
- Chest X- ray: Bilateral pleural effusion with basal consolidation.
- In view of respiratory distress, he was intubated.

- **CT Chest: Dense consolidation with areas of ground glassing in the posterior and superior segments of both lower lobes.** Bilateral mild pleural effusion.



- **Blood culture: Burkholderia pseudomallei**, initiated treatment with IV Meropenem.
- He developed **right hip joint pain** with restriction of movement on day 18.
- **MRI Right Hip: Acute osteomyelitis with an intramedullary abscess in the right acetabulum.**
- A hip arthrotomy and joint washout were performed.
- **Intensive phase of treatment**: **IV Ceftazidime 2 g Q6H for 6 weeks**, in combination with **oral Co-trimoxazole**.
- Due to drug-induced haemolytic anaemia, IV antibiotics were discontinued.
- The patient is currently in **the eradication phase of treatment,** receiving **oral Co-trimoxazole**.

- He is undergoing regular follow-up and is showing good progress.



CONCLUSION

- **Musculoskeletal melioidosis** is a well-recognized manifestation of the disease, which can manifest as soft tissue abscesses, osteomyelitis, septic arthritis, spondylitis and sacroiliitis.
- This case underscores the importance of raising awareness about this infection and its potential complications, particularly among clinicians who may not be familiar with the condition.

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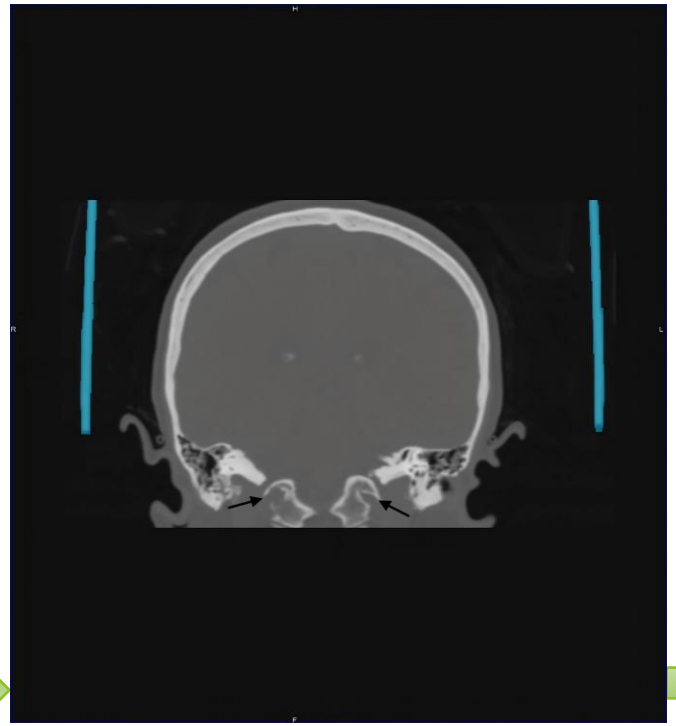
A Case of Bilateral Hypoglossal Nerve Paralysis Secondary to Base of Skull Metastasis in a Patient with Adenocarcinoma Of Lung

Dr. Muhammad Khursheed Ullah Khan Marwat *MBBS*
Queen's Centre for Oncology and Haematology, Cottingham

Case Presentation

A 59-year-old female was admitted with 2-day history of not being able to articulate and dysphagia (to both liquids and solids). She had recently been diagnosed with advanced metastatic adenocarcinoma lung (PDL1 80-90%, EGFR -ve, ALK -ve, KRAS mutation +ve) with mediastinal, spinal and cutaneous metastasis. On examination she had dysarthria with poor swallow. Tongue paralysed with no obvious fasciculation or atrophy. Rest of the neurological examination was normal.

MRI Head with contrast showed Skull Base metastasis(involvement of occiput along with bilateral hypoglossal canals and C1, C2 vertebrae)



Discussion

Skull-base metastasis from distant tumors occur in 4% of cancer patients. The three most common cancer types to metastasize to base of skull are breast, lung, and prostate cancers, accounting respectively for 40, 14 and 12% of cases [1]. Metastatic deposits in the skull can lead to various presentations depending upon the site of the metastasis. In a retrospective study of 43 patients treated for base-of-skull metastasis, Greenberg et al. identified five clinical syndromes of different frequencies: the orbital (7%), parasellar (16%), middle-fossa (35%), jugular foramen (16%) and occipital condyle syndromes (21%) [2].
While occipital condyle syndrome is characterized by severe, unilateral, occipital headache and ipsilateral twelfth-nerve palsy [8], the patient in discussion did not have the typical features of occipital condyle syndrome. Definitive treatment of base of skull metastasis may include one or a combination of radiotherapy, surgical intervention and systemic anticancer treatment [3].

Conclusion:

Isolated bilateral hypoglossal nerve paralysis can be a rare presentation of base of skull metastasis. It is important to include a differential diagnosis of metastatic disease on background of known/suspected cancer along with other causes of neurological deficit. Management can be challenging and should involve a multidisciplinary team including but not limited to Oncologists, neurosurgeons, radiologists, dietitians, speech and language therapists.

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A rare cause of septic shock: Capnocytophaga Canimorsus bacteremia

INTRODUCTION

Capnocytophaga is a gram-negative, nonspore-forming bacilli found mainly in the oral cavities of dogs and to a lesser extent cats. The bacterium cultures may not be visible for up to 48hours due to slow growth. Because of this, the first 5 days incubation normally show no bacterial growth.

Capnocytophaga canimorsus does not elicit a strong inflammatory response which results in excessive replication before detection by the host immune system. As a result, most patients have bacteremia before significant symptoms develop. Infections in humans is often associated with immunosuppression, often results in sepsis and can be fatal. The mortality is around 30% and doubles with septic shock presentation .

CASE PRESENTATION

Middle aged gentleman presented with a 5-day-history of high fever, productive cough, vomiting and diarrhea. Past Medical History includes Ischemic heart disease, Hypertension as well as history of intravenous drug use. Examination was unremarkable except widespread papular rashes over the body including chest, tummy.

INVESTIGATIONS

Blood tests showed metabolic acidosis, raised inflammatory markers, deranged coagulation profile and acute kidney injury. He was admitted to the intensive care unit (ICU) with a diagnosis of septicemia and multiorgan failure. Leptospira spira 16S DNA test, Vasculitis and Screening for TTP, atypical infection including fungal infections were all negative. CT Chest Abdomen Pelvis showed consolidation in both lungs without any other specific source of infection.

TREATMENT

His condition deteriorated rapidly with increase oxygen requirement and respiratory failure needing intubation and mechanical ventilation. Although repeat blood culture showed no growth within 48 hours, **Capnocytophaga** species was later detected in the first blood culture sample taken at admission. Further testing on subsequent blood culture identified **Capnocytophaga Canimorsus** using 16sRNA test 12 days after admission for which broad spectrum antibiotics have been prescribed. A detailed social history was then revisited and this elicited dog ownership but no history of bites.

OUTCOME AND FOLLOW UP

After one month in intensive care and a difficult tracheostomy wean, he was stepped down to a medical ward. Gangrenous toes were noticed whilst on ICU, but this was attributed to complications from septicaemia. This was later suspected as possible entry site for **Capnocytophaga Canimorsus** bacteria. The patient did not recollect any incident of dog bite but stated that the dog might have licked an existing toe wound. Vascular and orthopaedic team review of the gangrenous toes suggested a likely ischaemic cause from possible vasoconstriction from use of inotropes.

LEARNING POINTS

The fulminant sepsis caused by **Capnocytophaga canimorsus** has high mortality rate and timely diagnosis is critical to prevent death. In order to get the correct diagnosis, physicians should have high suspicion and make a comprehensive assessment including social history as well as full body thorough examination especially for the patients presenting with sepsis of unclear source. In addition, the NHS trust should have an assessment proforma for sepsis patient. In this way, zoonotic sepsis would be diagnosed and treated efficiently.

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The elusivity of SLE

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INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that is characterised by multi-organ inflammation. SLE affects predominantly women (female to male ratio 10:1).[1] Cardiac and renal involvement are common in lupus patients, affecting ~50% respectively, and contribute significantly to morbidity and mortality from the disease.[1,2]

CASE

We present the case of a 42yo female with a background of anti-phospholipid syndrome (APLS) who attended the acute medical unit in the setting of postural symptoms and chest tightness. She had repeated prior presentations in the setting of abdominal pain and fevers. Investigations revealed significantly raised inflammatory markers, raised serial high sensitivity troponin and ischaemic electrocardiogram changes. She was managed initially for acute coronary syndrome with dual anti-platelet therapy and a heparin infusion. She was commenced on antibiotics for possible underlying infection. Nonetheless, her condition deteriorated with subsequent upper gastrointestinal bleeding, acute renal failure, arrhythmia and acute heart failure with myopericarditis demonstrated on imaging. She was admitted to the intensive care unit for multiple organ support. Further investigations revealed an underlying diagnosis of SLE with both cardiac and renal involvement. She was treated with pulse methylprednisolone and rituximab with a significant improvement in her condition.

LEARNING POINTS:

- Identification of SLE can be elusive and is often significantly delayed
- 30-40% of systemic lupus erythematosus (SLE) patients also have anti-phospholipid syndrome [3]
- The most frequently recorded symptoms and signs prior to diagnosis of SLE are musculoskeletal, mucocutaneous and neurological symptoms however renal and cardiac involvement is seen in ~50% of patients with the diagnosis [1,2,4]
- Not all chest pain on the Acute Medical Unit is ACS; however SLE patients are at significantly increased risk of premature atherosclerosis and/or thrombosis [1-4]
- Do not treat SLE or APLS patients with heparin infusions; cannot interpret APTT /APTT ratios in patients with SLE or APLS due to presence of lupus anticoagulant which causes in vitro prolonged APTT [5]

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Atrial Myxoma: A Cause For Concern In Multiple Cerebral Infarctions In Absence Of Cardiac Symptoms

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SUMMARY

Ischaemic cerebral infarct is the most common neurological manifestation of myxomas.[1] A mid-70's male presented to the stroke resuscitation unit with ongoing sudden onset right hemiparesis and dysarthria. Although he had risk factors for developing stroke like hypertension, hypercholesterolemia, history of prostate cancer, and a new paroxysmal atrial flutter on admission; clinical suspicion, and detailed investigations in absence of cardiac symptoms revealed a 9 cm myxoma causing severe mitral stenosis. He underwent successful surgical resection with good functional recovery. This also highlights the need for thorough clinical examination especially in the absence of cardiac symptoms.

INTRODUCTION

About 75% of cardiac myxoma is found in the left atrium and it accounts for 50% of all benign cardiac tumour. The incidence peaks in the 40-60th year of age with female preponderance. 10% of myxoma are said to be of autosomal dominant disorder known as "Carneys complex" while the rest of them are sporadic. It can vary in size from 1 to 15 cm. Pathologically, they can either have a smooth, villous or friable surface with latter two associating more with embolic events while the former with obstructive. Surgical excision remains the only definitive treatment.

Cardioembolic strokes accounts for approximately 20% of all ischemic stroke.[2] It acts as an embolic source to the central nervous system, and other vascular regions.[3] It is a rare cause of stroke especially in young people.

CASE PRESENTATION

A mid 70's elderly male with background history of hypertension, hypercholesterolemia, localised prostate cancer, and well controlled asthma presented to the stroke resuscitation unit with right hemiparesis and dysarthria ongoing for 2 days.

He was left-handed, non-smoker, and independent with all activities of daily living (ADL).

His initial NIHSS score was 3, scoring 1 each for right upper limb drift, ataxia, and dysarthria. On examination, he exhibited moderate expressive dysarthria, and a diastolic murmur was noted during cardiovascular examination. Rest of the clinical examinations were unremarkable.

Routine stroke panel bloods were normal. Multiple 12 lead electrocardiogram showed that he was in paroxysmal atrial flutter with an average rate of 90 bpm.Initial non-contrast CT Head was unremarkable. CT angiography of head and neck did not reveal any significant atherosclerotic changes or stenosis. However, MRI Head showed multiple acute bilateral middle cerebral artery, and right posterior cerebral artery territory (vermis and right cerebellar peduncles) infarctions (Fig 1,2,3). A contrast enhanced CT TAP was also undertaken given the embolic nature of the stroke, and his history of cancer to rule out the possibility of metastatic disease and progression of cancer but was unremarkable.

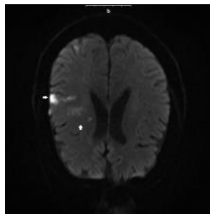

Fig 1: MRI DWI: Right MCA infarct

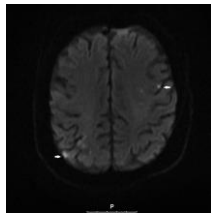

Fig 2: MRI DWI: Bilateral MCA infarct

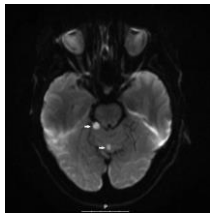

Fig 3: MRI DWI: Right Cerebellar infarct

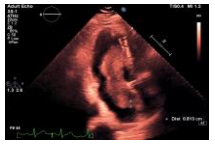

Fig 4: Apical 3 Chamber View of ECHO showing myxoma with stalk

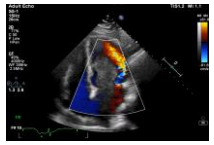

Fig 5: Mitral Valve Colour Doppler View of ECHO

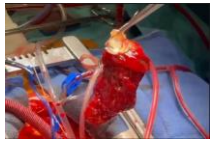

Fig 6: Intraoperative Gross Appearance of atrial myxoma after excision

Transthoracic echocardiogram was performed in this case due to the presence of a murmur and MRI brain scans suggestive of an embolic stroke. Mitral valve was broadly normal, thin, and mobile with good excursion of the leaflets. There was a significant functional mitral stenosis resulting from a large left atrial myxoma (9cm) resting between left atrium and left ventricle during diastole (Fig 4,5). There was a notable flow acceleration of around the body of the myxoma resulting mean pressure gradient of 10 mm of Hg. The mitral valve area (MVA) was 0.83 cm^2.

He was treated with high dose Aspirin (300mg) in accordance with the 2016 National Stroke Guideline initially with a view to start anticoagulation later. Given the degree of mitral stenosis, presence, and size of the myxoma he also underwent successful excision (Fig 6). Histopathological examination of the excised tumour revealed macroscopic appearance of 9x5x3 cm brown friable lobulated spongy mucoid mass. Microscopic appearance of nests of lepidic cells in abundant myxoid stroma admixed with clusters pigmented macrophages, thrombosis, and haemorrhages were noted.

DISCUSSION

Cases of ischemic stroke caused by myxomas have been well documented with 0.5% of all ischaemic strokes are being attributed to myxomatous embolization. [4]

If large, myxomas can cause obstructive symptoms, heart failure and pulmonary hypertension, and arrhythmias when they penetrate the myocardium. Fragments or complete tumour detachment can occur giving embolic complications like stroke which accounts for 80% of neurological symptoms caused by a myxoma or pulmonary embolism.[5] Clinicians may suspect atrial myxoma with auscultatory finding of a crescendo mid diastolic murmur.[6] Auscultatory abnormalities are found in 64% of patients and some also exhibiting "tumour plop".[7]

In case of uncertainty and high degree of clinic suspicion a transoesophageal echocardiography is preferred as it has been reported to show 100% sensitivity for cardiac myxoma.[8] Surgical excision remains the definitive treatment, and once diagnosed should be performed urgently to prevent future tumour growth, risk of embolization or acute cardiogenic shock and sudden cardiac death.[9] Patients may require annual follow-up with echocardiography for 3-4 years after surgery when the risk of recurrence is the highest.[10]

Asymptomatic cases are rare, and this important fact may have an impact on screening methods for such patients. Screening for myxoma is important in those cases of familial occurrence, in complex myxoma or Carney disease in particular.

CONCLUSION

Cardiac myxoma presenting as ischemic stroke is rare, and however this case highlights the importance of thorough physical examination, clinical suspicion, and use of imaging modalities as the myxoma could have been easily missed due to the presence of number of risk factors to explain the stroke.

LEARNING POINTS

- Aetiology of stroke is usually multifactorial. Efforts should be made to identify them.
- Rarer causes can be missed if not for high degree of clinical suspicion.
- Multiple territorial infarcts should raise suspicion of a cardioembolic stroke.
- Echocardiography should be considered in embolic strokes.
- CT brain is usually normal in acute strokes and MRI is required for confirmation.

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A unique case of co-trimoxazole induced hypoglycaemia

James Paget University Hospitals NHS Foundation Trust

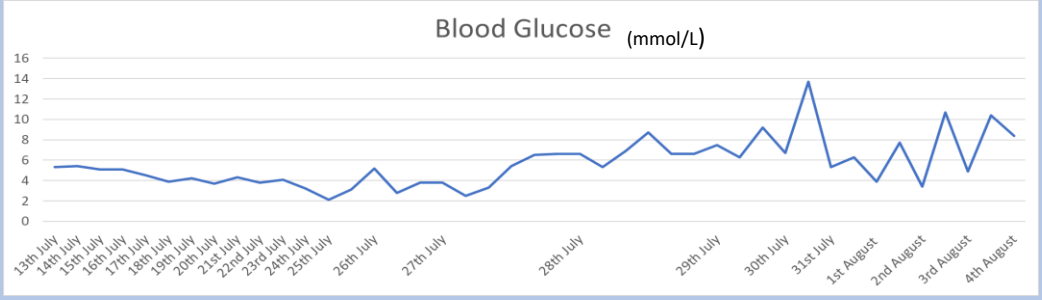
Dr Saad Naveed (SHO), Dr Wajiha Amjad (SpR), Dr Damodar Makkuni (Cons), Dr Jo Randall (Cons) James Paget University Hospital, Gorleston, United Kingdom

Background

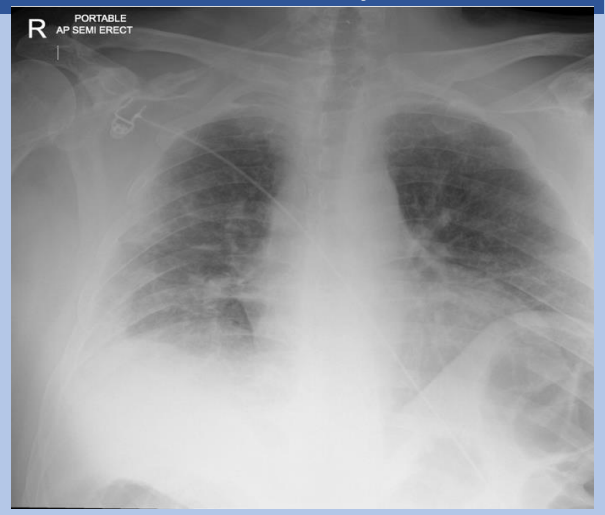
Co-trimoxazole is a commonly used antibiotic conventionally indicated in the management of Pneumocystis pneumonia(PCP) and atypical pneumonia in immunocompromised patients. Hypoglycaemia is a rare but life-threatening side effect of high dose co-trimoxazole in patients who are otherwise non-diabetic (1,2). It is believed that high dose co-trimoxazole stimulates pancreatic β-cells leading to hyperinsulinaemia and subsequently hypoglycaemia. Other factors which may predispose patients to this rare side effects include impaired kidney function, advanced age and malnutrition (3). There have been at least 28 reported cases of co-trimoxazole induced hypoglycaemia in the literature (4).

Case History

- A 60-year-old male presented with progressively worsening 3-week history of SOBOE and a dry cough with a background of GPA and renal vasculitis, for which he was on prednisolone, methotrexate and rituximab.
- He was treated with co-trimoxazole to cover for pneumocystis pneumonia, along with high dose oral prednisolone.
- Sputum cultures confirmed PCP and microbiology recommended an increased dose of 4320mg oral co-trimoxazole TDS for 2 weeks, whilst oral prednisolone was switched to IV hydrocortisone.
- He seemed to recover well and was deemed medically fit for discharge.
- Over the next few days, he gradually become more lethargic, hypotensive, constipated, nauseous with occasional bouts of post-prandial vomiting.
- He developed AKI 2 with hyponatraemia and hyperkalaemia . He was treated with IV fluids with potassium. It was initially suspected this was due to vomiting whilst SIADH was queried. USS abdomen was normal.
- Following an unwitnessed inpatient fall, his BMs were noted to be 2.1mmol/L. He was immediately treated with IV dextrose and glucagon.
- His BMs kept fluctuating, with frequent symptomatic hypoglycaemic episodes (tachypnoea, disorientation).

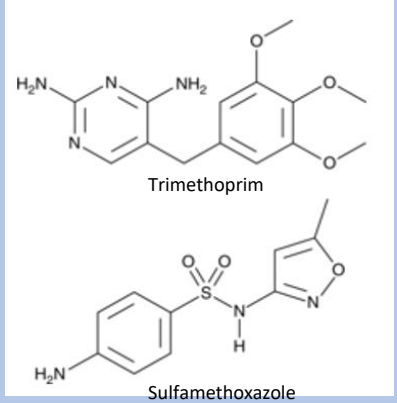


Chest X ray

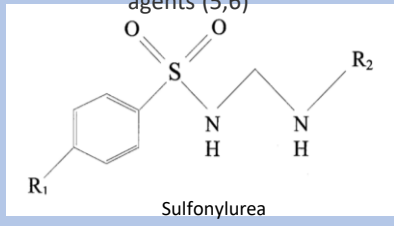


Lab tests

Investigation	Result
Sodium	108 mmol/L ↓
Potassium	6.2 mmol/L ↑
eGFR	41 ml/min/1.73m^2
Creatinine	155 micromol/L ↑
Serum osmolality	225
Urine osmolality	462
Urine Sodium	116 mmol/L
9am Cortisol	327 nmol/L



The sulfamethoxazole part of co-trimoxazole has the same sulfanilamide chemical structure as sulfonylureas commonly used as antidiabetic agents (5,6)



Other Scans

CT head	No acute intracranial pathology seen. No intra or extra-axial haemorrhage seen.
CTPA	Negative for PE. Worsening ground glass opacifications in lung.
USS abdomen	No acute abnormality appreciated.

Management

- He was commenced on hypertonic saline, 50% IV glucose and desmopressin with an aim to gradually restore the sodium levels.
- The case was discussed with the infectious diseases team at the nearest tertiary center
- Co-trimoxazole was stopped and the patient was subsequently started on clindamycin and primaquine.
- He improved gradually over a week's time with the abovementioned antibiotics and a reducing regime of prednisolone. He was discharged and followed up in rheumatology clinic.

Learning Points

- This unusual case presents a rare but potentially lethal side effect of co-trimoxazole
- Currently, BMs are not routinely checked in non-diabetic patients when being administered co-trimoxazole
- Blood glucose levels should be regularly monitored even in a non-diabetic patient receiving co-trimoxazole in addition to close monitoring of renal function, electrolytes and fluid balance

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An interesting case of Cannabinoid hyperemesis syndrome precipitating Wernicke's Encephalopathy: A case report.

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Background

Wernicke's encephalopathy (WE) is a severe and potentially life-threatening condition caused by a deficiency of thiamine (vitamin B1). It is considered a rare condition, with estimated prevalence ranging between 0.8% and 2.8%. The incidence of WE is higher in individuals with a history of alcohol dependence, AIDS, bariatric bypass surgery, and malnutrition. Diagnosing WE relies on maintaining a high level of *clinical suspicion* in patients at risk of vitamin B1 deficiency, conducting a focused clinical examination, and performing appropriate targeted laboratory investigations. Imaging studies and cerebrospinal fluid analysis are crucial in excluding other potential differentials, such as encephalitis, Miller-Fisher syndrome, Bickerstaff brain stem encephalitis, and toxic/metabolic encephalopathies.

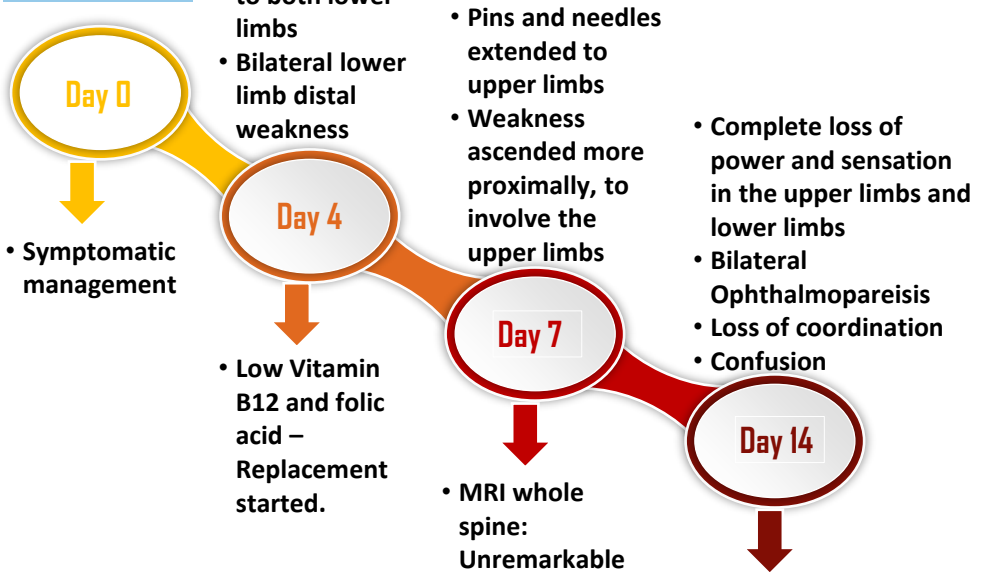
Case reported:

A 24-year-old female patient presented to the emergency department with abdominal pain and vomiting with frequency of 7-10 times per day, ongoing over the last 2-3 months.

Past medical history: anxiety, depression, polycystic ovary syndrome

Social history: lives alone, cannabis abuse, a current smoker and minimal alcohol consumption.

Timeline



Learning points

1- Wernicke's encephalopathy is a clinical diagnosis, a triad of change in mental status, ophthalmoplegia and gait dysfunction is usually highly diagnostic. However, it is only present in 10% of cases so a high suspicion index is required and treatment should be started promptly.

2- Non-alcoholic Wernicke's encephalopathy can present differently to alcoholic WE. However, treatment should not be delayed, while waiting MRI brain or blood or CSF results.

3- Raised CSF protein can occur in WE

4- Thorough history-taking is crucial to analyse the presenting symptoms especially when symptoms do not fir in one diagnosis as in the presented case.

Diagnosis of Wernicke's encephalopathy secondary to cannabinoid hyperemesis.

- Treatment initiated with IV Thiamine
- Patient was also given IVIg due to initial suspicion AMSAN.
- Confusion and ophthalmoparesis resolved
- Patient started to regain her motor and sensory function
- And was discharged to a rehab unit for further

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Pacemaker Lead Migration Presenting As Shoulder Pain and Pulsating Abdomen: A Case Report

Dr Sophia Khattak, Dr Basit Kakroo, Dr Saib Khogali

The Royal Wolverhampton NHS Trust

Introduction and Background

Lead displacement and migration remains a rare but potentially serious complication of pacemaker implantation.[1,2]

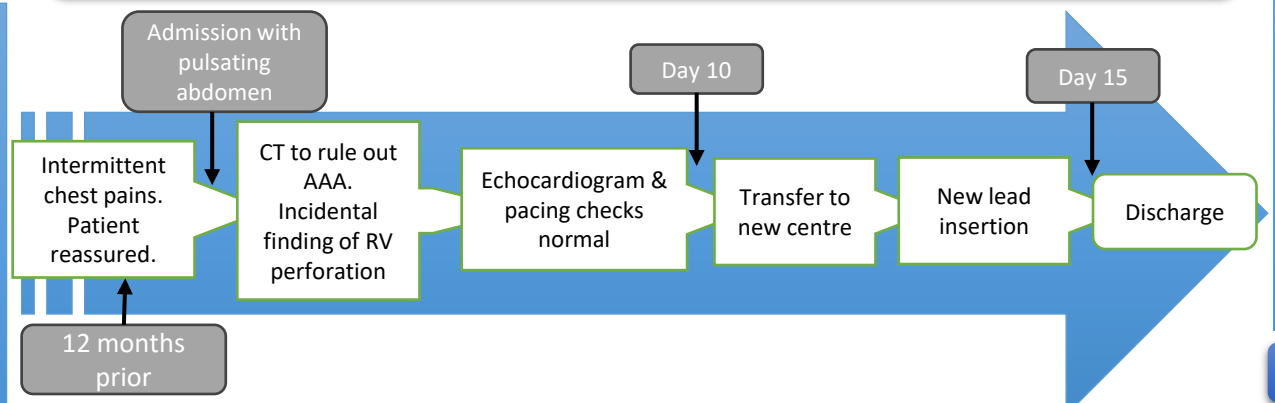
One of these complications is right ventricular (RV) perforation, which is rare but life-threatening .[3,4]

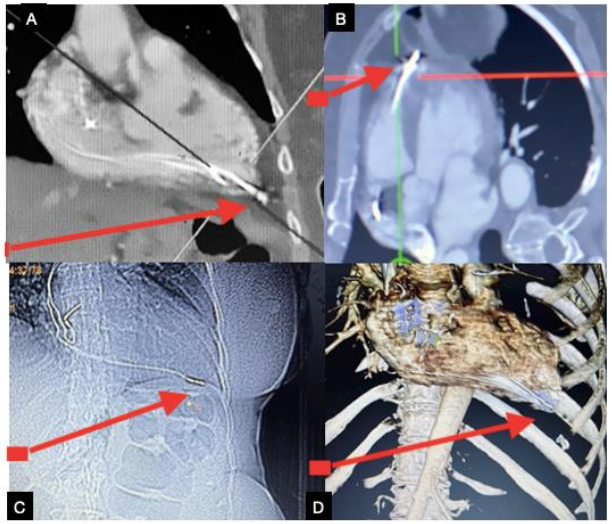
Presentation with RV perforation is usually acute or subacute as pericardial effusion, tamponade or rhythm disturbance from a malfunctioning pacemaker.

Case Description

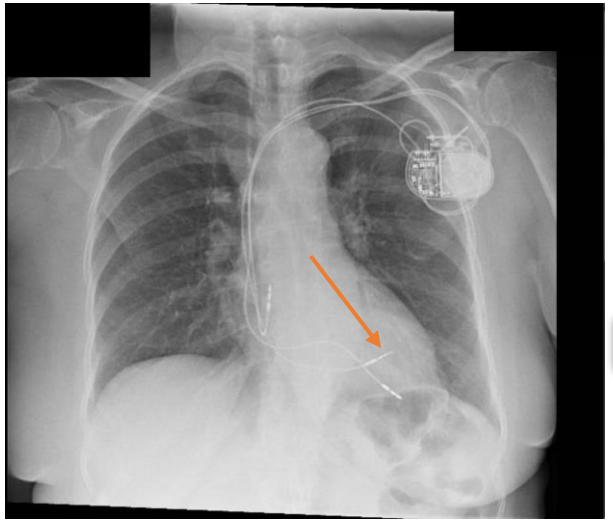
A 65 year old female was admitted with presenting complaint of a pulsating abdomen that felt like a 'jumping sensation in the chest.' The patient had symptoms of left shoulder pains intermittently for around 12 months.
PMH: Hypertension and permanent pacemaker inserted 6 years ago for complete heart block.

Timeline





CT images showing RV lead perforating the RV apex into the pericardium.
A and C are coronal planes, B = Axial plane, D = 3D coronal plane



CXR after new lead insertion showing a total of 3 pacemaker leads.
Second RV lead on the right ventricular septum (arrowed)

Outcome

The patient had placement of a new active RV lead to high septal position via axillary puncture which was connected to the existing device.
The migrated RV lead was capped and left due to risk of causing further perforation and haemodynamic instability.

Discussion & Conclusion

- RV lead perforation may present unusually years after implantation.
- Normal pacing check and echocardiogram does not rule out a perforation.
- There is no real consensus on the management in stable patients. MDT should always be consulted.

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