Title: Complements never lie: Bacterial endocarditis caused by Bartonella henselae infection masquerading as an anti-neutrophil cytoplasmic antibody (ANCA)-associated small vessel vasculitis

Author: Stefanie Christina Robert

Co-Authors: Suzanne Forbes
Ravi Rajakaria
Sorush Soleimanian
Michael Millar
Julia S Hadley

Topic: Case Reports

AIM: We present a patient with culture negative bacterial endocarditis secondary to Bartonella henselae infection, masquerading as an anti-neutrophil cytoplasmic antibody (ANCA)-associated small vessel vasculitis (SVV).

OUTCOME: Our 74 year-old patient presented with several weeks of constitutional symptoms and acute kidney injury. He was known to have a prosthetic aortic valve. On examination he was febrile with palpable splenomegaly and vasculitic lesions on both hands. Nephritic screen revealed a strongly positive c-ANCA (PR3 > 100AU/ml) with consumed complements. Renal biopsy showed a proliferative glomerulopathy (picture 1). Initial differential diagnoses included SVV or infective endocarditis. Transoesophageal echocardiography, however, demonstrated no vegetations and serial blood cultures were negative. Immunosuppression with prednisolone and cyclophosphamide was therefore instituted. Shortly thereafter the patient developed septic shock requiring multiorgan support in intensive care. An atypical screen for culture negative endocarditis revealed a strongly positive IgG antibody titre (1:512) against Bartonella henselae. In view of this and the fact that all five minor modified Duke criteria were fulfilled, culture negative endocarditis was diagnosed. Immunosuppression was discontinued and treatment for chronic bartonellosis (gentamicin, clarithromycin) commenced. The patient made a remarkable recovery, was discharged and has regained renal function.

CONCLUSION: This case highlights the challenge of differentiating infective endocarditis from SVV in light of their similar presentation and overlapping symptomatology. The importance of clear history taking and performing tailored investigations is reinforced. Although c-ANCA is highly specific for vasculitis, it is vital to be aware of causes of false-positivity as the treatment options vary significantly. Here, without the correct diagnosis, the outcome may have differed dramatically.
Title: GHB withdrawal: Emergence of a new phenomena

Author: John Bright

Co-Authors: Shoneen Abbas
Tania Syed
Vengal Nagareddy
Jacob Wembri

Topic: Case Reports

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AIM

To highlight the emergence of a new phenomena of acute presentations of GHB (gamma-hydroxybutyrate) withdrawal.

CASE SERIES

Over a six month period November 2010 to May 2011 we noted prospectively three cases of GHB withdrawal.

On average the consumption was 5-10 bottles per month (up to 300mls per month).

All patients were ingesting on a daily basis up to every hour.

All patients were male 20-30 years old. One was a student, one in full time employment and the other unemployed.

Patient two presented to the Emergency Department with severe agitation, hallucinations and tachycardia. He was visiting the UK and had not ingested GHB for 48-72 hours. His mother reported at least 8 years chronic usage on a daily basis with no other medications/drugs.

He presented with delirium, auditory and visual hallucinations and disorientation in time and place. He was profusely sweating with mild tachycardia 110-120 beats per minute (ECG confirmed sinus tachycardia). Only laboratory abnormality was CK 750 U/L. His initial management was normal saline and PRN diazepam. However due to poor symptomatic response he quickly required high dose oral and intravenous benzodiazepines. High dose β-blockers were used to control autonomic features. By day 6 his symptoms were manageable and he was orientated in time and person. However total length of admission was 13 days (8 of which on acute medical unit).

All patients presented with delirium and autonomic instability. All required high dose benzodiazepines oral and IV. All required prolong inpatient stays on MAU as initially erroneously labelled as alcohol withdrawal “Delirium Tremens”.

CONCLUSIONS

We do not feel we have identified a new clinical entity, but we have educated ourselves to consider GHB withdrawal in all young patients presenting with delirium and presumed alcohol withdrawal.

In view of prolonged symptoms and autonomic features we recommend this patient group not be managed in acute medical units, but are transferred to general medical wards for expected prolonged admissions.

We therefore need to improve awareness of this growing clinical problem.
Title: The Case of the Weak Legs

Author: Rebecca Gray

Co-Authors: Alan Vallon

Topic: Case Reports

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Aim

To increase awareness of a rare condition presenting on an acute medical take.

Methods

A 30 year old gentleman was referred by his GP to the AMU with a two week history of progressive difficulty in walking and some right sided back ache. He had sustained a rugby tackle two weeks earlier but had no injuries at the time other than presumed bruised ribs. He had one episode of feeling feverish 48 hours prior to referral. He had no past medical history and was on no regular medications.

Positive examination findings were bilaterally weak legs – power 4/5. Sensation was decreased to a level of T7. Reflexes and anal tone were intact. Planters were flexor. His temperature was 38 C. His bloods tests showed a raised CRP.

Results

An urgent MRI (picture 1) showed an epidural abscess at T3-T6, soft tissue swelling from the abscess was also seen on the chest x ray (picture 2). He was discussed with the neurosurgeons and transferred for definitive treatment within 6 hours of presentation. He underwent surgical decompression of the abscess on the same day. The culture grew Staphlococcus aureus and he received i.v antibiotics and made a full recovery.

Conclusion

Epidural abscesses are rare (2.5-3 per 10 000 admissions\(^1\)) but potentially devastating, early diagnosis and appropriate management is essential for a good outcome. Blunt trauma is reported to precede symptoms in 15-35% of patients\(^2\). Epidural abscess should be considered in patients presenting with fevers, back ache and focal neurology.

References


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Title: Exploding Headache

Author: Shahideh Safavi

Co-Authors:

Topic: Case Reports

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Introduction

Headache is a common presentation on the acute medical take, and diagnoses range from tension headache to subarachnoid haemorrhage. This is the case of an uncommon cause of headache.

Case Report

A 26-year-old man presented to the hospital with a 3-day history of headache, describing it as “sudden onset exploding headache” with associated photophobia, not responding to paracetamol. He had also noticed trouble using the fork. The positive findings on examination were those of photophobia, neck stiffness, reduced power in left arm and leg, with up-going left plantar reflex.

At this stage, subarachnoid haemorrhage and right hemisphere stroke were the main differential diagnoses. The patient’s inflammatory markers, chest radiograph and ECG were all normal. He underwent CT brain, MRI brain and MRA brain, all of which were normal. Lumbar puncture showed lymphocytosis.

The diagnosis of HaNDL syndrome (headache, neurological deficit and CSF lymphocytosis) was made. The patient was reassured and discharged, and his symptoms resolved 2 days later.

Discussion

HaNDL syndrome is an uncommon cause of headache, with good prognosis, which can mimic acute stroke. Its diagnosis requires a high index of clinical suspicion, can reassure patients and avoid unnecessary, and at times, risky, invasive investigations.

However, when questioned, out of the 35 medical FY2 and CT1 doctors, none was willing to discharge a patient with background of HaNDL syndrome without lumbar puncture, if he presented with sudden onset headache. This is where sensible decision making during a post-take ward round can lead to patient reassurance and early discharge.

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Title: Spontaneous Pneumomediastinum: Not So Uncommon In Winter?

Author: You Yi Hong

Co-Authors: Ziaullah Khan
Marcus Simmgen

Topic: Case Reports

Background

Pneumomediastinum (mediastinal emphysema) is a consequence of air leakage from the bronchial tree or lungs into the mediastinal structures, with possible upward extension into the neck. While traumatic chest or oesophageal injuries are well-known causes of secondary pneumomediastinum, the first case of spontaneous pneumomediastinum was described only in 1939 by Hamman. It is considered rare, with approximate frequencies of 1 per 10,000 admissions (or fewer) reported.

Aim

Three cases of spontaneous pneumomediastinum presenting during the winter season 2010/11 serve to remind Acute Physicians of the causes and risk factors, common and unusual clinical presentations, key investigations, management principles and prognosis of spontaneous pneumomediastinum.

Case Reports

The first case contracted H1N1 influenza-A infection, suffered from underlying interstitial lung disease and had undergone CABG in the past. In the second case, the General Practitioner detected supraclavicular surgical emphysema when the patient presented with an acute respiratory illness and persistent dry cough. The third case developed in a 15 year-old boy with controlled asthma after a strenuous, but trauma-free football training session.

In all cases, a computed tomogram of the thorax was the definitive investigation for diagnosis. No associated pneumothorax was present. All patients were discussed with Cardiothoracic Surgery and no intervention was necessary. Chest radiographs were used to follow-up clinical resolution.

Key Points

Spontaneous pneumomediastinum usually occurs in lung disease, but exercise, recreational drug inhalation and various other circumstances need to be considered. Traumatic secondary causes must be excluded. The majority of cases can be managed conservatively. Intractable coughing hinders recovery. Antibiotic cover is only indicated in established infection.

Conclusion
Acute Physicians need to be mindful of spontaneous pneumomediastinum as it may present with both non-specific and unusual symptoms in a spectrum of pulmonary and non-pulmonary conditions.
Title: A hot bath to calm what ails you - the cannabis hyperemesis syndrome

Author: Vishal Luther

Co-Authors:

Topic: Case Reports

Aim:

Acute medical units are frequently exposed to the manifestations of drug abuse. A case is presented of a recently recognised complication of chronic cannabis use.

Method:

A 21 year old caucasian male was admitted with a 12 hour history of nausea, vomiting and epigastric pain. This was his seventh identical presentation in the last 18 months. He had no other previous medical history or significant family history. He rarely drank alcohol, but used cannabis heavily.

On examination, the patient was retching violently though appeared surprisingly clean. Unusually, he desperately wanted a hot bath. His haemo-dynamics were stable and his abdomen was soft. Admission blood tests, urinalysis and radiography all proved normal.

Following previous admissions, the patient typically self discharged after 24 hours of intravenous fluids, antiemetic and analgesia with a diagnosis of presumed gastritis. On each occasion, investigations proved normal. He had been seen and discharged by a gastroenterologist as an outpatient and had a normal endoscopy.

Outcome/results:

Cannabis is used synthetically to treat chemotherapy induced vomiting [1]. Paradoxically, chronic cannabis abuse is associated with a cyclical vomiting syndrome which characteristically eases with a hot bath. This effect may arise through thermoregulation of endogenous cannabinoid activity in the brain and enteric nervous system. Cannabis cessation results in complete symptomatic recovery [2].

Conclusion:

Cannabis use is common. Given its novelty, cannabis hyperemesis syndrome is likely to be highly under recognised (we have seen four more cases in the last three months), resulting in frequent unnecessary acute admissions.
References:


An 19 year old male was admitted to the Emergency Admissions Unit with a one week history of shortness of breath, productive cough, nausea and central pleuritic chest pain. On examination, he was febrile, tachycardic and hypoxic. Chest examination revealed few scattered expiratory wheeze but no crackles. Initial chest X-ray showed no focal lung consolidation.

He was started on intravenous co-amoxiclav and treatment dose dalteparin. CT pulmonary angiogram revealed bilateral diffuse, patchy ground glass opacification in keeping with early infection.

Next day, he developed multiple painful ulcers, with yellow exudates throughout his mouth. These spread onto the palate, lips and tongue and caused him to have difficulty swallowing. Later, we noted conjunctival involvement as his left conjunctiva was hyperaemic with purulent discharge. No lesions elsewhere were noted and, in particular, examination of the genitalia was normal. These lesions were considered to be characteristic of Stevens-Johnson syndrome.

He had a repeat chest x-ray which showed patchy air space changes in the left lower lobe in keeping with infection. Oral erythromycin was added for severe pneumonia.

PCR tests for herpes simplex and varicella zoster viruses, urinary antigen for legionella pneumophila and serum antibody for HIV were negative. Gram staining of material from the oral ulcers was also negative.

Mycoplasma serology tests were taken in the acute phase which showed mycoplasma pneumoniae CFT titre > 512 and the mycoplasma pneumoniae agglutination test was positive. Convalescent serum sample were taken 9 days post-acute phase was consistent with recent mycoplasma pneumoniae infection. Antibiotic therapy was continued and the patient made an excellent recovery.

Stevens-Johnson syndrome has been well documented as an extra pulmonary manifestation of mycoplasma pneumoniae infections. Stevens-Johnson syndrome is characterized by erythematous target lesions on the skin and involvement of the oral mucosa, genitals and conjunctivae. Several cases have reported incomplete presentation, primarily in children, which lack the typical rash, but retain oral, ocular and genital manifestations. Our case illustrates that incomplete presentation can occur with only oral mucosa and conjunctival lesions alone, without skin or genital involvement. Treatment of Stevens-Johnson syndrome remains supportive along with treating the underlying infection if recognised.
Title: SUDDEN PAINLESS VISUAL LOSS IN ACUTE MEDICINE- NOT GIANT CELL ARTERITIS, WHAT IS IT?

Author: Suneeta Teckchandani

Co-Authors: Ruby Roy

Topic: Case Reports

Introduction:

In Acute Medicine, Ischemic optic neuropathy is the commonest cause of sudden painless visual loss.

Ischemic optic neuropathy is categorized as anterior (affecting the optic disc, swollen disc) versus posterior (affecting retrobulbar optic nerve, no changes on fundoscopy) and as arteritic(GCA) versus nonarteritic (cardio-embolic).

Methods:

A 67 year old gentleman with a background of Asthma, presented with sudden visual loss in his left eye. He had been having a constellation of symptoms for the last 3 months: proximal muscle pains, paraesthesias in both hands, loss of manual dexterity, night sweats and weight loss.

Results:

On examination he was cachectic and pyrexial(39.3C). Temporal arteries were pulsatile and nontender. Mild weakness of hip flexors and small muscles of the hands were elicited.

Left eye examination revealed no perception of light, relative afferent pupillary defect and swollen optic disc (fig1). Bloods showed eosinophilia, raised inflammatory markers, positive p-ANCA. CT head demonstrated fluid in paranasal sinuses.

Churg Strauss Syndrome(CSS) is a systemic necrotising vasculitis characterized by peripheral neuropathy, pulmonary involvement, paranasal sinus abnormalities and eosinophilia.

It is associated with a number of ocular manifestations including granulomatous conjunctivitis, retinal vascular occlusions, cranial neuropathy and ischemic optic neuropathy.1

Our patient was commenced on methyl prednisolone and cyclophosphamide. At 3 months, his vision had minimally improved and he developed optic atrophy(fig2), but all his other symptoms had improved remarkably.

Conclusion:

In summary, our patient fulfilled the clinical criteria of CSS.2 Prompt diagnosis and initiation of the treatment is vital to avoid complications.
References


CASE REPORT

A 52 year old gentleman presented with 2 day history of headache followed by left facial droop, associated with slurred speech. He was discharged with prednisolone and acyclovir after diagnosing Bell’s palsy. He presented with right sided facial symptoms a week later with no other on-going symptoms. Rest of neurology was unremarkable apart from a bilateral LMN facial palsy.

Routine blood tests were all normal. Diagnosis centred investigations for Lyme disease, sarcoidosis, viral serology and vasculitis were negative. Imaging studies drew a blank as was CSF evaluation which only showed a mild nonspecific raise in protein level. He was diagnosed as idiopathic bilateral simultaneous facial palsy.

DISCUSSION

Bilateral facial paralysis is extremely rare with an incidence of only 0.3%–2%(1). The most common causes are Lyme disease, Guillain-Barre syndrome, idiopathic (Bell’s) palsy(2), leukaemia, sarcoidosis, bacterial meningitis, leprosy, infectious mononucleosis, skull fracture and viral infections.

Lyme disease is caused by Borrelia Burgdorferi. In our patient, the Lyme serology was negative. HIV, Herpes viruses and infectious mononucleosis may also affect the facial nerve but the viral screen and CSF- PCR for viruses were negative.

Guillain-Barre syndrome(4) was excluded by absence of associated neurology and non diagnostic CSF ana-lysis. Normal MRI brain imaging made CNS leukaemia, lymphoma and benign intracranial hypertension unlikely.

Sarcoidosis(3), SLE and PAN are also noted to cause facial diplegia, but with normal ESR, CXR, serum ACE and auto antibody screen, these possibilities were excluded.

Our patient was finally diagnosed to have idiopathic bilateral simultaneous facial palsy.

REFERENCES


Title: One hospital's experience of the 2011 London Marathon

Author: Matthew Mak

Co-Authors: Carl Reynolds

Topic: Case Reports

Aim

To describe a series of patients who were admitted to Chelsea and Westminster Hospital following the London Marathon

Methodology

The London Marathon is one of the biggest sporting events in the world. Over 36,000 participants ran the 31st London Marathon on 17th April 2011. Our hospital was not one of the designated receiving units for marathon related casualties. We analysed the case-notes and results of patients admitted to our unit after the marathon.

Outcomes and Results

Seven patients (5 male, 2 female) attended A&E after collapsing at the marathon, all of whom were subsequently admitted to the Acute Assessment Unit. The average patient age was 36 (range 28-49). Four patients had never run a marathon previously and all collapsed at the finish. All but one of the patients ran the marathon solo.

All patients experienced a significant rise in creatine kinase. Three patients had cardiac troponins tested, all of which were elevated and subsequently fell. None of these were felt to be significant after review by a cardiologist. The average length of stay was 1.57 days.

Conclusion

Marathon running places huge stresses on the human body. Exercise associated collapse can be associated with exertional heat illness/rhabdomyolysis or exertional hyponatraemia. Exercise induced troponin rises are most likely benign. Cessation of activity, rest during recovery and early aggressive fluid replacement are the mainstays of treatment. Emergency Departments and Acute Medical Units should be prepared for marathon related illnesses and educate staff to prevent unnecessary investigation and treatment of exercise related physiological changes.

References


