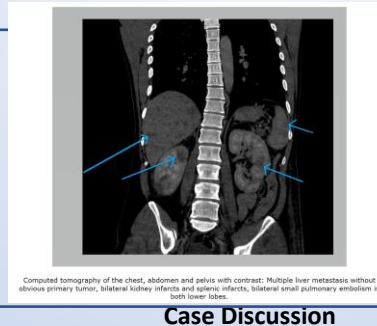
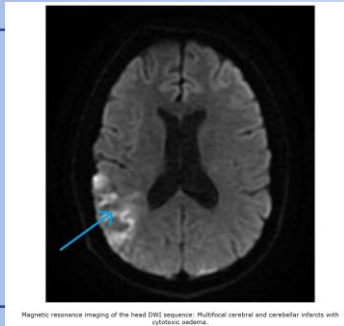


Background

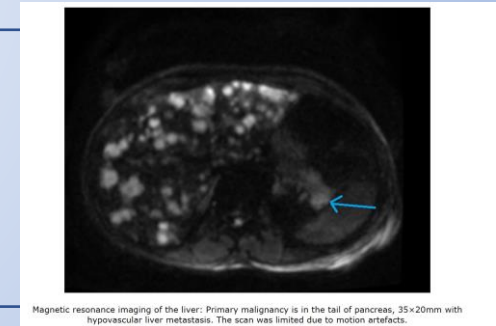
Marantic endocarditis and Trousseau's syndrome are historically linked with pancreatic cancer. The patient had catastrophic embolic events which caused multi organ infarct due to underlying advanced pancreatic cancer. Doctors should be aware of the early signs and symptoms of pancreatic cancer and conduct necessary clinical assessment and investigations which can prevent any severe complications.

Case Presentation

A middle-aged healthy and fit gentleman presented with left sided weakness, slurred speech, left sided sensory neglect, mild headache and fever. He didn't have any other systemic symptoms. He smokes 5-10 cigarettes since adolescence and drinks occasionally 1-2 beers every week. He was vaccinated against COVID-19 and COVID-19 PCR was negative. On examination he only had left sided hemiparesis and with mild sensory neglect and UMN type of facial nerve palsy and GCS 14/15. Other systemic examination was unremarkable. Vitals were stable except mild rise of temperature. CT head at admission showed acute right sided temporal ischaemic changes. He was not thrombolysed as onset of symptoms was more than 4.5 hours. Blood tests showed high CRP 202 with normal haemoglobin, clotting and renal function. Blood and urine culture were negative twice. ECG showed sinus tachycardia with normal CXR. Echocardiogram showed low normal EF with mild MR. He had TOE due to continued spike of temperature whilst on broad spectrum IV antibiotics to rule out IE and it showed myxomatous MV and MR. He later had MRI head and MRA carotids which showed multiple cerebral and cerebellar infarcts with normal carotid arteries. He mentioned later that he was having left leg pain for two months. USS doppler of left leg showed large left leg DVT involving the femoral vein. On the 9th day of admission he had liver function tests and it came back deranged with Bilirubin 13.7, ALT 212, ALP 188, GGT 415, albumin 29 and normal platelets and INR. Previous LFTs were normal. CT CAP was arranged and it showed bilateral PEs, kidneys and spleen infarction and multiple liver metastasis with unknown primary. Treatment dose of LMWH was started. Anti-phospholipid antibodies, HIV, Hepatitis and vasculitis screening was negative and non-invasive liver test: autoimmune liver screen, iron studies, A1AT, serum ceruloplasmin were negative. Gastro team was involved and MRI liver was planned. MRI liver showed multiple liver metastasis with primary mass in the tail of pancreas. Cancer screening bloods showed high CA 19-9 >10000.0 with normal AFP, CEA and PSA. We took collateral history from his mom who mentioned that he went to the GP for right leg pain three months ago and he was treated for right leg sprain. He then developed left leg pain one month later and attended hospital where he was treated for left leg cellulitis. D-Dimer at that time was 6501. The clinical events explained that most likely he developed Trousseau's syndrome three months ago and the embolic phenomena was due to Marantic endocarditis (Non-bacterial thrombotic endocarditis) secondary to pancreatic cancer. MDT advised for USS guided liver biopsy, but sadly he passed away the next day due to deteriorating clinical condition.



Case Discussion



Marantic Endocarditis, currently named as Non-bacterial thrombotic endocarditis (NBTE) is a consequence of hypercoagulable state due to chronic inflammatory conditions, autoimmune diseases and malignancy with complex pathogenesis. It is characterised by deposition of fibrin thrombi in previously undamaged heart valves with the absence of bloodstream bacterial infection. It has rarely been reported as antemortem. As the vegetations are more friable, the NBTE can cause systemic thrombo-embolism more rapidly and extensively than infective endocarditis. Sometimes the patients' neurological manifestations precede the diagnosis of advanced pancreatic cancer.

Trousseau syndrome, also commonly referred to as Trousseau's sign of malignancy to avoid confusion with Trousseau's sign of latent tetany is a type of paraneoplastic syndrome caused by adenocarcinomas, predominantly gastric, pancreatic and pulmonary, presenting as recurrent and migrating episodes of thrombophlebitis. The pathophysiology of the phenomena is not well established, but the mucin compounds produced by adenocarcinomas have been found to interact with the selectin family of adhesion molecules, leading to thrombocyte activation and aggregation. It can cause arterial and venous thrombosis and NBTE. The treatment involves therapeutic anti-coagulation and management of underlying malignancy.