published reports on CAHA are from patients who have undergone cardiothoracic surgery with hypothermia.

**Aims:** To describe perioperative care challenges in a patient with hip fracture and CAHA, and review the literature on the topic. This is the second published case report regarding CAHA in an orthopaedic patient.

**Methods:** Case report and literature search using terms “cold agglutinins” AND “surgery” or “perioperative” or “orthopaedic” or “hip fracture” or “drug interactions” or “anticoagulation”.

**Results:** Case report: An 86 year old female with a 4-year history of recurrent episodes of CA-related haemolysis presented with hip fracture. Her past medical history also included atrial fibrillation (anticoagulated with Dabigatran), hypertension, strokes (and secondary epilepsy), vascular dementia, type 2 diabetes mellitus (complicated by diabetic retinopathy), osteoporosis (previous vertebral fractures), urinary incontinence, recurrent urinary tract infections, multinodular goitre with subclinical hypothyroidism. The presence of CAs was identified in 2016 and confirmed thereafter several times; over the following years she developed CAHA and was commenced on Prednisolone (62.5 mg daily) a month prior to the hip fracture. On admission, her haemoglobin was 69 g/L, the blood film showed red cell agglutination at room temperature, which dissociated at 37°C and acute haemolysis. She required transfusion of 5 units of packed red blood cells during her admission (unfortunately 2 units were not warmed in the emergency department). Her management included intensive temperature monitoring, use of body thermal blanket, warming intravenous and irrigation fluids and medications before administration. Surgery was performed under general anaesthesia on day 4 of admission. Dabigatran was withheld pre-operatively and restarted when haemoglobin was >90 g/L. She received Enoxaparin (40 mg daily) for venous thromboembolism prophylaxis, antibiotics (Cefazolin) for wound infection prophylaxis, and continued Prednisolone (62.5 mg, following haematologist’s advice). The patient was discharged to the rehabilitation ward on the 7th postoperative day. Bone marrow biopsy was not organised due to patient’s preferences.

**Discussion and Literature Review:** CAs are circulating autoantibodies (commonly IgM) directed against polysaccharide antigens located on the erythrocytes surface; low temperature activates CAs and the classical complement cascade resulting in haemagglutination, lysis, increased blood viscosity and microvascular thrombosis. CAHA accounts for 15%-32% of all autoimmune haemolytic anaemia. It occurs as a primary CA disease (CAD, being a clonal low grade lympho-proliferative disorder) or as a secondary CA syndrome (CAD, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma). CAD is rare (13-16 per million) with secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma) or as a secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma). CAD is rare (13-16 per million) with secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma) or as a secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma). CAD is rare (13-16 per million) with secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma) or as a secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma). CAD is rare (13-16 per million) with secondary CA syndrome (CAS, provoked by infections, e.g. Epstein-Barr virus, Mycoplasma, or lymphoma).

**Conclusion:** Patients with CAHA require careful perioperative examination and prompt initiation of effective therapy. A management algorithm is presented.

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**Abstracts**

**APLASTIC ANAEMIA SECONDARY TO SARS-COV-2 INFECTION**

**Dr Maya Wernick**1,2, Dr Stacy Lewis3, Dr Christie Moore3

1Christchurch Hospital, CDHB, Christchurch, New Zealand, 2Providence St Vincent Internal Medicine Residency, Portland, Oregon, USA, 3Providence Cancer Center Oncology and Hematology Care Clinic, Portland, Oregon, USA

**Introduction:** Aplastic anaemia (AA), a rare haematological disease with an estimated incidence of two per million in Western countries, is characterised by pancytopenia and a profoundly hypocellular bone marrow. While the exact pathogenesis of AA is unknown, activation of cytotoxic T-cells which inappropriately target antigens on haematopoietic stem cells are thought to play a large role. This immune phenomenon is known to be induced by viral infections, in particular HIV and the hepatitis viruses. We describe what we believe to be the first case of aplastic anaemia following infection with the SARS-CoV-2 virus.

**Case Description:** A 40-year-old Hispanic female presented with lower limb petchae ten days following resolution of symptoms from SARS-CoV-2 infection. Initial laboratory work-up showed pancytopenia with haemoglobin 103 g/L, white cell count 2 × 10^9/L, neutrophils 0.7 × 10^9/L, and platelets 5 × 10^9/L. Viral serology studies for HIV, Hepatitis B and Hepatitis C were negative. Coagulation studies were normal and there was no evidence of haemolysis. The reticulocyte count was low at 2.7 × 10^9/L. A peripheral smear confirmed pancytopenia without blasts.

Initially immune thrombocytopenia (ITP) was suspected, and treatment with dexamethasone and IVIG was given. No improvement in the pancytopenia was seen, and a subsequent bone marrow biopsy exhibited a markedly hypocellular marrow. Immunostaining for parovirus-19 was negative, as was subsequent flow cytometry for parovascular nocturnal haemoglobinuria (PNH). Due to the absolute neutrophil count at this time being less than 0.2 × 10^9/L a diagnosis of severe aplastic anaemia was made.

The patient’s age precluded her from an allogeneic haematopoietic stem cell transplant, and she was started on immunosuppressive therapy with anti-thymocyte globulin (ATG), cyclosporine (CSA), and prednisone. Initial therapy with the thrombopoietin agonist, eltrombopag, was not possible due to the patient’s lack of insurance and immigration status.

**Discussion:** This case highlights and adds to the growing literature surrounding the associated effects of SARS-CoV-2 infection. While the exact pathogenesis of aplastic anaemia is unknown, one theory proposes abnormal antigen stimulation and inappropriate cell activation due to increased levels of IL-17 as part of a cytokine storm leading to apoptosis, which in aplastic anaemia involves the haematopoietic stem cells. And while we cannot definitively say that the effect is causal, the close temporal relationship of this case should draw attention to the possible development of this rare haematological condition following infection with the SARS-CoV-2 virus, and to consideration of the virus being added to the array of viral infections that are known to be implicated in the pathogenesis of aplastic anaemia. Lastly, this case highlights the complexities of providing evidence-based care for patients without health insurance and legal documentation which during a pandemic with a novel virus adds to the growing inequities in outcomes following infection with SARS-CoV-2.

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3. Figlerowicz M, Mania A, Lubarski K, et al. First case of convalescent plasma transfusion in a child with COVID-19-associated severe aplastic anemia. Transfus Apheresis Sci. 2020;102866.