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3.3.1 Number of research papers per teachers in the journal notified on UGC website during the year 2018-2019

S.No	Title of paper	Name of the author/s	Department of the teacher	Name of journal	Year of publication	ISSN number	Link to recognition in UGC enlistment of the journal	Page No
1	The Complex Management of 55- Year old Male with Long Standing Type 2 Diabetes, Non -Proliferative Diabetic Retinopathy, and Diabetic Nephropathy: A Multi- disciplinary Approach	B.Ramavathi	Pharmacology	Journal Of Current Innovations In Advanced Research	Sep-18	2636-6282	https://www.ijciar.com/index.php/journal/article/view/160	3
2	Pancytopenia : A Case Study with Uncommon Neurological Manifestations	D.Purnima Yadav	Pharmaceutics	Journal Of Integral Sciences	Dec-18	2581-5679	https://www.jisciences.com/index.php/journal/article/view/68	4
3	A Case Study On Ondine's Curse : A Challenging Case of Congenital Central Hypoventilation Syndrome	B.Yerni Kumar	Pharmacology	Journal Of Current Innovations In Advanced Research	Oct-18	2636-6282	https://www.ijciar.com/index.php/journal/article/view/161	5
4	Chronic Kidney Disease Stage 5 and severe complications in a 60- year - old Male with hypertesion	M. Divya	Pharmacology	Journal Of Current Innovations In Advanced Research	Nov-18	2636-6282	https://www.ijciar.com/index.php/journal/article/view/162	6



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5	The Complexities of Postpartum Hemorrhage in Antiphospholipid Antibody Syndrome (ALAP) : A Complex Case Study Analysis	A. Nanaji	Pharmacognosy	Journal Of Integral Sciences	Dec-18	2581-5679	https://www.jisciencess.com/index.php/journal/article/view/69	7
6	Case Study on X- Linked Adrenoleukodystrophy	M.Madhavi Kumari	Pharmacology	Journal Of Integral Sciences	Mar-19	2581-5679	https://www.jisciencess.com/index.php/journal/article/view/71	8
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"The complex Management of 55-Year-Old Male with Long-Standing Type 2 Diabetes, Non-Proliferative Diabetic Retinopathy, and Diabetic Nephropathy: A Multi-disciplinary Approach"

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Abstract:

A 55-year-old male with diabetes type 2 mellitus, non-proliferative diabetic retinopathy, and recently diagnosed chronic renal failure related to diabetic nephropathy is the subject of this case. The patient approached with complaints of general weakness, tiredness, and a 15-day midnight fever. Laboratory tests revealed that the patient had high blood glucose levels, anemia, increased liver enzymes, and compromised renal function. Tight control of glucose, Managing of blood pressure, anemia correction, and treatment of etiologies are all part of the multidisciplinary care strategy.

Keywords: diabetes type 2 mellitus, etiologies.

Introduction:

Diabetes nephropathy is the primary cause of chronic kidney disease in individuals commencing renal replacement treatment (1) and is linked to an increased risk of cardiovascular death (2). Proteinuria more than 0.5 g/24 h has traditionally been used to diagnose diabetic nephropathy. Overt nephropathy, clinical nephropathy, proteinuria, or macroalbuminuria have all been used to describe this stage. Seminal European research from the early 1980s demonstrated that modest levels of albumin in the urine, which were not normally detectable by traditional procedures, were predictive of the eventual occurrence of proteinuria in type 1 (3-5) and type 2 (6) patients with diabetes. Microalbuminuria or initial nephropathy were terms used to describe this stage of renal dysfunction. A variety of risk factors have been linked to the onset and growth of Diabetic retinopathy. Diabetes duration, control of glycemic levels, age, type of diabetes, hypertension, renal illness, dyslipidemia, pregnancy, anaemia, smoking, and alcohol are all systemic risk factors. Ophthalmic risks include posterior retinal detachment, cataract surgery, and pre-existing chorioretinopathy, among others. The duration of diabetes and the level of control of glucose are the most powerful indicators of the possibility of retinopathy [7].

Case study:

A 55-year-old male patient was admitted to the hospital with the major complaint of loss of appetite over the past month, as well as general weakness and fatigue. Patient had a midnight fever for 15 days. Since 13 years, the patient suffers from type 2 diabetes mellitus (DM) and





Pancytopenia: A Case Study with Uncommon Neurological Manifestations

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Abstract

This case study investigates a unique presentation of pancytopenia in a patient featuring an unusual neurological symptom. The exploration covers the clinical features, examination findings, clinical hypothesis, intervention, outcomes and engages in a comprehensive discussion on the diagnostic challenges and management strategies associated with pancytopenia.

Keywords: Pancytopenia, Unusual neurological symptom.

Introduction

Pancytopenia, a hematological disorder, characterized by a simultaneous reduction in red blood cells, white blood cells, and platelets, occasionally presents with atypical symptoms [1]. This case study highlights a rare neurological manifestation, progressive peripheral neuropathy, in a patient with pancytopenia, emphasizing the need for a sophisticated approach to diagnosis and treatment. Progressive peripheral neuropathy is a neurological condition that results from injury to the peripheral nerves, which are found outside of the brain and spinal cord. The hands and feet are typically affected by this ailment, which can also produce weakness, numbness, and pain. Other bodily systems and processes, such as digestion and urination, may also be impacted [2].

All blood cells will originate from the hematopoietic stem cells (HSC). HSC's differentiate into multipotent progenitors such as myeloid progenitor and common lymphoid progenitor. The Myeloid lineage produces red blood cells (erythrocytes), platelets (thrombocytes), and various types of white blood cells (granulocytes, monocytes) and the lymphoid lineage produces lymphocytes, including T cells, B cells, and natural killer (NK) cells. The disturbances in the hierarchical tree of human hematopoiesis leads to cytopenias [5].

Cytopenia is not specific, can be caused by various underlying health conditions such as infections, aplastic anemia, cancer in bone marrow, nutritional deficiencies and autoimmune issues [3,4].

Clinical Features

A 38-year-old female presented with classical signs of fatigue, tingling sensations and diminished reflexes which are worsening at night in General Medicine Department. The patient's medical history, lifestyle factors, and family background were thoroughly examined to understand the potential underlying causes.

Examination Findings

Clinical assessments revealed the typical signs of pancytopenia, including pallor, petechiae, and anemia-related fatigue. The levels Hb, red blood cells, white blood cells, and platelets were significantly decreased. Notably, the patient exhibited progressive peripheral neuropathy, characterized by tingling sensations, weakness, and diminished reflexes. Hematological investigations and nerve conduction studies were conducted to explore the correlation.

Diagnosis

The integration of hematological and neurological findings led to a clinical hypothesis suggesting an underlying

A Case Study On “Ondine's Curse: A Challenging Case of Congenital Central Hypoventilation Syndrome

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Abstract:

Congenital Central Hypoventilation Syndrome (CCHS) is also known as Ondine's Curse. A rare hereditary condition known as congenital central hypoventilation syndrome (CCHS) affects babies and is evidenced by breathing difficulties during sleep without apparent respiratory warning manifestations. It is a globally underreported illness caused by a mutation in the PHOX2B gene. Affected people are increasingly able to endure life to adulthood due to increased survival rates that were achieved by early detection and care. The clinical characteristics, diagnostic difficulties, and management strategies for CCHS are covered in the current study.

Keywords: Ondine's Curse, PHOX2B gene, Hypoventilation, Genetic disorder, Management strategies.

Introduction:

A mutation in the PHOX2B gene develops congenital central hypoventilation syndrome (CCHS), a rare inheritable condition impacting the autonomic nervous system (ANS)¹. Typically, during nonrapid eye movement sleep, neonates with CCHS exhibit symptoms such as apnoea, oxygen deprivation, along with elevated carbon dioxide levels, but they do not exhibit overt signs of respiratory distress². Mellins et al first described the syndrome in 1970, describing a case in which a child experienced inadequate breathing due to central nervous system causes despite normal respiratory triggers, such as increased carbon dioxide levels, and after ruling out primary conditions in the lungs, heart, chest, and neuromuscular system³. Despite the fact that more than 1,000 cases have been documented worldwide, the true incidence probably exceeds estimated rates of one in 200,000 live births in France and one in 148,000 in Japan^{4,5}. Some cases have been documented in older children and young adults, with severe respiratory difficulties or respiratory infections following general anaesthesia^{6,7}. Better detection of CCHS and early attention have culminated in higher rates of survival, allowing numerous impacted individuals to mature into adulthood. This discussion aims to explore the clinical characteristics, diagnostic complexities, and management hurdles encountered in CCHS patients. This investigation examines the clinical features of CCHS, along with the diagnostic challenges and medicinal techniques.

Case Presentation:

A 4 years old male patient was brought to the pediatric department with a chief complaint of recurrent episode of cyanosis, especially during the sleep. Parents reported the child appearing breathless and occasionally turning blue, less of concerns of potential respiratory distress.



Chronic kidney disease stage 5 and severe complications in a 60-year-old Male with hypertension

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Abstract:

The case study illustrates the complex interaction between hypertension with severe CKD, emphasizing the importance of integrated treatment to address chronic kidney disease, imbalances in electrolytes, and associated consequences.

Keywords: Hypertension, CKD, imbalances in electrolytes.

Introduction

Stages 5, with an ageing population contributing to this rise [1,2]. Diabetes is the single most prevalent cause of renal failure, accounting for 24 percent of patients with CKD in the UK [3]. The National Institute of Clinical Excellence (NICE) recommends that persons with CKD stages 4 and 5 be sent to a nephrologist for a professional assessment. General Practitioners (GPs) are usually able to manage stages 1-3 [4]. Other medical disorders, such as diabetes and coronary artery disease are frequently connected with CKD. Patients with advanced CKD are at a higher risk of death [5]. Whereas renal replacement therapy (RRT) has improved the prognosis of kidney disease, the disease continues to impact quality of life, and there is a large treatment cost associated with dialysis[6]

Case study

A 60-year-old male patient was admitted to the nephrology ward with the major complaints of acute dyspnea for one week, decreased urine production for seven days, loss of appetite for eight days, pedal edema for eight days, and general weakness. Normal sleep and aberrant bladder function are seen over use of analgesics. Known case of hypertension since 5years. On general examination, all vital signs are normal except blood pressure (140/90) hypertension along with GFR level less than 15ml/min/1.73m². Laboratory tests reveal hematuria, albuminuria, proteinuria, ketonuria, increased blood creatinine levels, hyperkalemia, anemia, hypocalcemia, and uremia.

Diagnosis

The patient was diagnosed with Chronic Kidney Disease Stage 5 as an indication of hypertension.

Management

Lasix 20 mg intravenous injection is given twice a day. Lasix is a furosemide that is used to treat edema. Pantoprazole 40 mg is administered intravenously once daily. Amlodipine 5mg in conjunction with atenolol 50 mg, taken orally, is used to treat hypertension. Once a day, a





The Complexities of Postpartum Hemorrhage in Antiphospholipid Antibody Syndrome (APLA): A Complex Case Study Analysis

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Abstract

In the presence of antiphospholipid antibodies, antiphospholipid antibody syndrome (APLA) is an autoimmune thrombophilic condition characterised by recurrent venous, arterial, or small artery thrombosis and pregnancy morbidity. Secondary APS can develop in conjunction with other autoimmune disorders, or it can be primary in the absence of any underlying illness. It's a significant obstetric emergency and one of the leading causes of maternal morbidity and mortality worldwide. Managing postpartum haemorrhage in women with APS can be complex. The use of anticoagulants to manage APS needs to be balanced against the risk of bleeding. Specialists will need to balance the risk of clotting with the risk of bleeding, aiming to provide effective management for both conditions without exacerbating either. This case study elucidates the challenges encountered in the management of postpartum haemorrhage (PPH) associated with Antiphospholipid Antibody Syndrome (APLA). The case of a 24-year-old postnatal woman experiencing excessive bleeding following delivery due to APLA and recurrent miscarriages emphasizes the complexities and considerations essential for effective management in such scenarios.

Keywords: autoimmune disorder, postpartum haemorrhage, phospholipid-binding proteins.

Introduction

APLA syndrome, also known as Hughes syndrome, was first described in the 1980s by Dr. Graham Hughes. Antiphospholipid antibodies cause antiphospholipid syndrome, also known as antiphospholipid antibody syndrome (APS or APLS), an autoimmune hypercoagulable condition. [1] Antiphospholipid syndrome (APLS) is a multisystemic autoimmune disorder. The presence of persistent antiphospholipid antibodies (APLA) in the context of arterial and venous thrombosis and/or pregnancy loss is the hallmark of APLS. [2] Some estimates indicate that the incidence of the APS is around 5 new cases per 100,000 persons per year and the prevalence around 40–50 cases per 100,000 persons. [3] APS is a medical condition with a high prevalence rate, as 15 to 20% of women who experience recurrent miscarriage exhibit APS. [4] One of the main diagnostic criteria for APLA syndrome is the existence of aPL, which includes

antibodies such as lupus anticoagulant, anticardiolipin, and anti-beta-2-glycoprotein I antibodies. However not every individual with aPL experiences clinical symptoms, which complicates the process of diagnosis as well as treatment. [5] Uncertainty surrounds the precise processes by which aPL induce thrombosis and problems associated to pregnancy. It has been hypothesized that these antibodies cause a prothrombotic condition by interacting with different cells and proteins in the immune system, endothelium, and coagulation cascade. [6]



Case Study on X-Linked Adrenoleukodystrophy

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Abstract

X-Linked Adrenoleukodystrophy (X-ALD) is a rare and devastating genetic disorder characterized by progressive demyelination of the central nervous system and adrenal insufficiency. This X-linked disorder is caused by mutations in the ABCD1 gene, leading to the accumulation of very-long-chain fatty acids (VLCFAs). The patient in question is a 10-year-old male, presenting with a history of progressive neurological symptoms such as vision and hearing loss, difficulty swallowing, and motor dysfunction. Diagnosis poses challenges due to the diverse clinical presentation, requiring a multidisciplinary approach encompassing neurological assessments, imaging studies, and genetic testing. Understanding the genetic basis has facilitated genetic counseling and family screening, allowing for early identification of at-risk individuals. Current treatment strategies focus on symptom management and disease modification. Hormone replacement therapy addresses adrenal insufficiency, while dietary interventions and experimental treatments like hematopoietic stem cell transplantation aim to reduce VLCFA levels and slow disease progression. Ongoing research explores genetic therapies and targeted interventions, offering hope for improved outcomes. Long-term management involves regular monitoring, rehabilitation services, and palliative care as symptoms evolve. The comprehensive care of individuals with X-ALD requires collaboration between healthcare providers, researchers, and patient advocacy groups.

Keywords: X-ALD, Adrenoleukodystrophy, Very-Long-Chain Fatty Acids, rehabilitation services, motor dysfunction, Peroxisome, progressive demyelination.

Introduction

Adrenoleukodystrophy (ALD) is a genetic disorder that follows X linked inheritance pattern in most cases (X-ALD). A unique neonatal form classified as one form of Zellweger syndrome has an autosomal recessive inheritance pattern (N-ALD). Although, the earlier report in the 1900s described the clinical presentations suggestive of this disease.; the terminology and pathophysiology were first reported in the 1970s [1].

The brain, spinal cord, adrenal glands, and testes are the most commonly affected organs. Given the multiple organs, involvement multidisciplinary team approach is highly recommended in the management plan [2]. Adrenoleukodystrophy (ALD) is a genetic disorder that follows X linked inheritance pattern in most cases (X-

ALD). A unique neonatal form classified as one form of Zellweger syndrome has an autosomal recessive inheritance pattern (N-ALD) [3]. Although, the earlier report in the 1900s described the clinical presentations suggestive of this disease.; the terminology and pathophysiology were first reported in the 1970s [4]. The brain, spinal cord, adrenal glands, and testes are the most commonly affected organs. Given the multiple organs, involvement multidisciplinary team approach is highly recommended in the management plan [5].

Case Presentation

The patient in question is a 10-year-old male, presenting with a history of progressive neurological symptoms such as vision and hearing loss, difficulty swallowing, and motor dysfunction. A detailed family history revealed that the patient's maternal uncle had a similar clinical presentation, leading to suspicions of a genetic



Case study on recurrent meningitis with otitis media in hiv positive child.

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Abstract

Bacterial meningitis is a neurologic crisis. Immunization against normal microbes has diminished the weight of sickness. Early conclusion and fast commencement of empiric antimicrobial and adjunctive treatment are fundamental. Treatment ought to be started when blood cultures have been acquired, going before any imaging studies. Clinical signs of bacterial meningitis incorporate fever, migraine, meningismus, and a modified degree of cognizance yet signs might be scant in youngsters, in the old, and in meningococcal sickness. In this case study the child with HIV positive had a recurrent headaches and ear pain where the lab investigations shows the child has a streptococcal infection with middle ear inflammation.

Keywords: Meningococcal, Meningitis, Immunization.

Introduction

Streptococcal meningitis is an intense, purulent irritation of the layers encompassing the brain and spinal cord caused by microbes from the Streptococci species.

Meningitis is a disease and aggravation of the liquid and layers encompassing the cerebrum and spinal cord. These films are called meninges.

The inflammation from meningitis regularly sets off side effects like cerebral pain, fever and a stiff neck

Most instances of meningitis in the US are brought about by a viral disease. Be that as it may, microscopic organisms, parasites and growths additionally can cause it. A few instances of meningitis improve without treatment in

half a month. Others can cause death and require immediate antibiotic treatment.

Symptoms

- Sudden high fever.
- Stiff neck.
- Severe headache.
- Nausea or vomiting.
- Confusion or trouble concentrating.
- Seizures.
- Sleepiness or trouble waking.
- Sensitivity to light.
- No appetite or thirst.
- Skin rash in some cases, such as in meningococcal meningitis.

Various causes of meningitis

Bacterial meningitis

Microbes that enter the circulatory system and travel to the brain and spinal cord cause bacterial meningitis. Yet, bacterial meningitis likewise can happen when microscopic organisms straightforwardly attack the meninges. This might be brought about by an ear or sinus contamination, a skull fracture, or — once in a while — a few medical procedures.

