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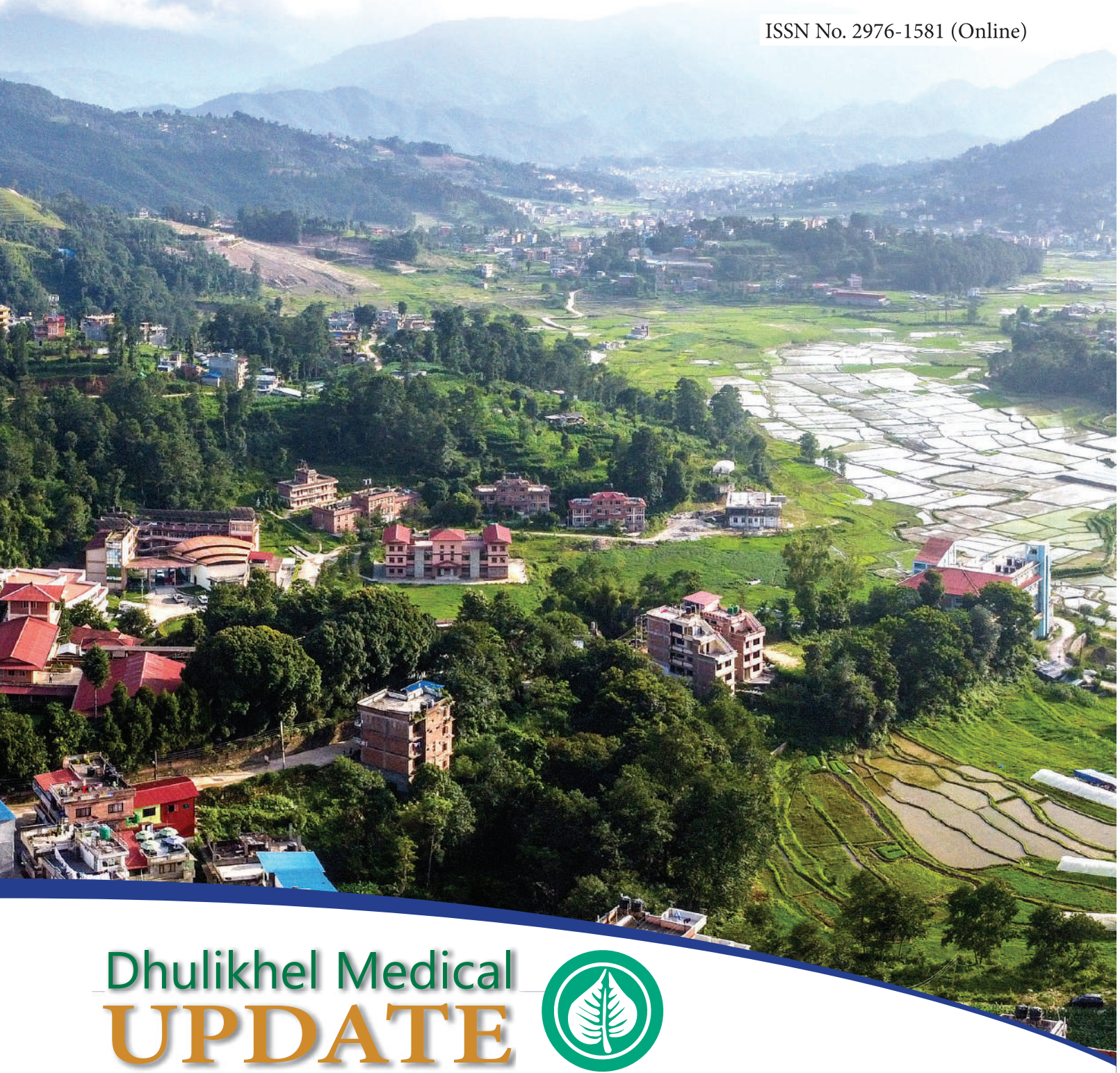

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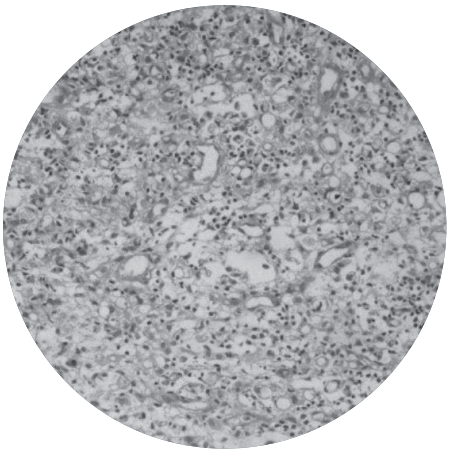

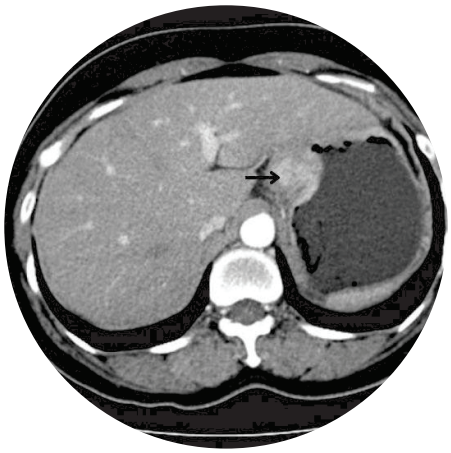

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Rosai-Dorfman Disease Presenting as Isolated Cervical Lymphadenopathy in a 12-Year-Old Female: A Case Report

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Abstract

Background

Rosai-Dorfman disease (RDD) is a rare, benign disorder characterized by painless, massive cervical lymphadenopathy. First described in 1965, it commonly presents with fever, leukocytosis, elevated ESR, and hypergammaglobulinemia. Diagnosis is confirmed by histopathology showing histiocyte proliferation with emperipolesis and positive S-100 and CD68 staining.

Case Presentation

A 12-year-old girl presented with a gradually enlarging left cervical lymph node. Fine-needle aspiration cytology and excisional biopsy confirmed RDD. After surgical excision, she was treated with steroids, leading to regression of lymphadenopathy.

Conclusions

RDD generally has a benign, self-limiting course. However, careful monitoring is essential due to possible relapse or extranodal involvement. Early diagnosis and appropriate management yield favorable outcomes.

Keywords: *Case Report, Histiocytosis, Juvenile, Lymph Nodes, Rosai-Dorfman Disease, Sinus Histiocytosis.*

Introduction

Rosai-Dorfman disease (RDD), or sinus histiocytosis with massive lymphadenopathy (SHML), is a benign, self-limiting disorder marked by painless cervical lymph node enlargement[1]. Classic symptoms include fever, leukocytosis, and hypergammaglobulinemia[2]. Notably, 40% of cases exhibit extranodal involvement in organs such as skin, eyes, nervous system, bone, gastrointestinal tract, and more, often mimicking other conditions[2-6]. Diagnosis of nodal RDD is typically straightforward on H&E staining with supportive immunohistochemistry, but extranodal presentations or those with fibrosis and minimal emperipolesis can be challenging[7]. Our case is notable for presenting as isolated unilateral cervical lymphadenopathy in a 12-year-old without systemic symptoms or extranodal involvement. FNAC followed by excisional biopsy confirmed RDD, highlighting the importance of histological evaluation even in clinically ambiguous cases. The rarity of such isolated pediatric presentations without systemic signs makes this case

unique and worthy of reporting.

Case Report

12-year-old female presented to the Otorhinolaryngology outpatient department of Dhulikhel Hospital with a one-year history of swelling over the left upper lateral aspect of the neck (Figure 1). The swelling was painless and gradually progressive, with no associated history of fever or cough.

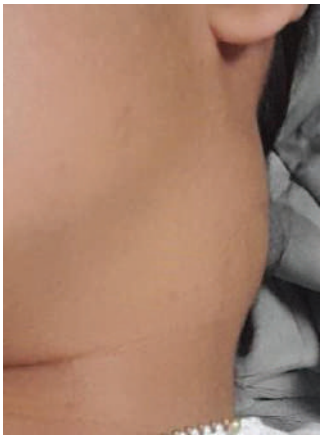

Figure 1. ***Rosai-Dorfman disease: A 12-year-old girl with left cervical lymphadenopathy***

Clinical examination revealed multiple enlarged left cervical lymph nodes, ranging in size from 1×2 cm to 2×2 cm. The overlying skin appeared normal, with no change in color or local rise in temperature. The lymph nodes were non-tender, firm to hard in consistency, mobile, with smooth and regular surfaces, and not adherent to the skin. No other lymphadenopathy was detected.

Ultrasonography of the neck showed multiple enlarged lymph nodes with loss of central fatty hilum at levels IB and V on the left side, the largest measuring 25×11 mm. CT scan of the neck revealed multiple homogeneously enhancing, discrete enlarged lymph nodes in the left cervical region (levels IB and V), the largest measuring 25×22 mm.

Fine-needle aspiration cytology (FNAC) of the left cervical lymph nodes showed numerous histiocytes with eccentrically placed round-to-oval nuclei, prominent nucleoli, and abundant pale cytoplasm, with evidence of lymphophagocytosis (emperipolesis). The cytological diagnosis was Rosai-Dorfman disease (RDD). The patient was initially managed with anti-inflammatory medication and kept under regular follow-up. However, after two months, the swelling did not regress, and an excisional biopsy was planned. Under general anaesthesia, a single soft, yellowish, well-circumscribed encapsulated globular lymph node mass measuring 3×3 cm was excised (Figure 2).

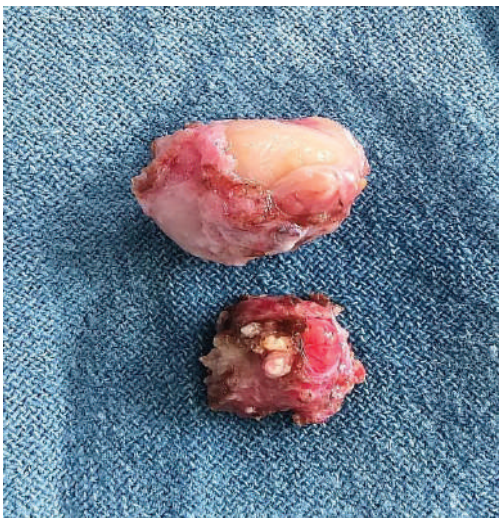

Figure 2. *Photograph of excised lymph node*

Histological examination revealed encapsulated lymphoid tissue with effaced architecture (Figure 3). The distended sinuses contained a prominent population of histiocytes characterized by large round-to-oval nuclei, prominent nucleoli, and abundant cytoplasm showing emperipolesis, engulfment of intact lymphocytes.

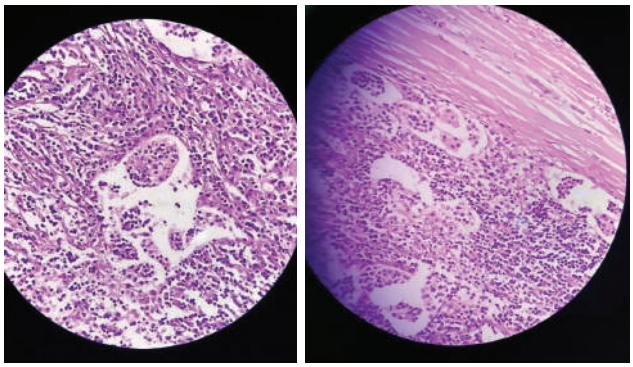

Figure 3. *Photomicrograph showing lymphoid tissue with effaced architecture and emperipolesis, suggestive of sinus histiocytosis*

The intervening stroma showed an increased number of plasma cells, blood vessels, and areas of haemorrhage. The final diagnosis was sinus histiocytosis with massive lymphadenopathy (Rosai-Dorfman disease). Postoperatively, the patient was treated with oral corticosteroids for two weeks, after which progressive regression of the lymph node swelling was observed (Figure 4).

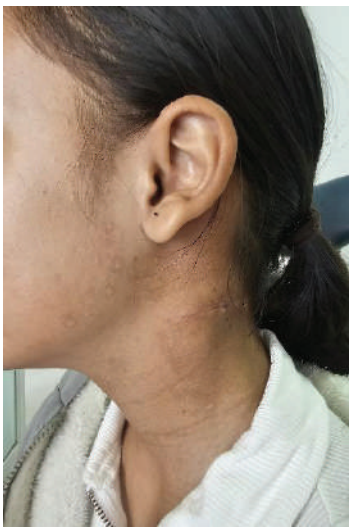

Figure 4. *Post-operative photograph after excision of the lymph nodes*

The patient remains under regular follow-up, with no evidence of recurrence to date.

Conclusion

Rosai-Dorfman disease (RDD) is a rare, benign histiocytic disorder that can mimic more serious pathologies, particularly when presenting as isolated lymphadenopathy without systemic symptoms. Early recognition based on characteristic cytological features especially emperipolesis, supported by histopathology and immunohistochemistry, is essential for accurate diagnosis and appropriate management. Our case highlights an uncommon presentation of RDD as unilateral cervical lymphadenopathy in a pediatric patient without systemic or extranodal involvement, which makes it clinically unique and diagnostically challenging. Surgical excision combined with a short course of corticosteroids led to complete resolution. This case reinforces the importance of considering RDD in the differential diagnosis of persistent cervical lymphadenopathy in children and the value of a multidisciplinary approach in ensuring effective management and follow-up.

Consent

Written informed consent was obtained ensuring patient's anonymity.

Declaration of competing interest

There are no conflicts of interest.

Acknowledgement

N/A

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Human-Centered AI in Medical Education: Between Hype, Hope, and Responsibility

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Abstract

Thyroid carcinoma, the most common endocrine malignancy, presents with diverse histological subtypes and distinct genetic alterations. Recent advances in molecular genetics have improved our understanding of the oncogenic drivers underlying thyroid carcinogenesis. This short communication highlights the key genetic alterations associated with different thyroid cancer subtypes, including papillary, follicular, medullary, anaplastic, and Hürthle cell carcinoma. We discuss their clinical implications in diagnosis, prognosis, and targeted therapy, with a special focus on mutations in BRAF, RAS, RET, TP53, and TERT. Additionally, inherited cancer syndromes such as Cowden syndrome and DICER1 syndrome are reviewed.

Keywords: *Genetic alteration, Targeted therapy, Thyroid cancer*

Introduction

Thyroid carcinoma is a heterogeneous disease arising from follicular or parafollicular thyroid cells. While most thyroid cancers are well-differentiated and have a favorable prognosis, certain subtypes are aggressive and associated with poor outcomes. Genetic mutations play a pivotal role in thyroid carcinogenesis, influencing tumor initiation, progression, and response to therapy. A deeper understanding of these molecular events not only aids in diagnosis but also opens avenues for targeted therapeutic strategies.

Main Text

Papillary Thyroid Carcinoma (PTC)

Papillary thyroid carcinoma (PTC) is the most prevalent subtype, accounting for approximately 80–85% of all thyroid cancers[1]. It is characterized by a relatively indolent clinical course but can metastasize to regional lymph nodes.

BRAF Mutations

The most common genetic alteration in PTC is the B-Raf proto-oncogene, serine/threonine kinase (BRAF)^V600E mutation, found in nearly 60% of cases. This mutation leads to constitutive activation of the mitogen-activated protein kinase (MAPK)/extracellular signal-regulated kinase (ERK) signaling pathway, promoting tumor cell proliferation and survival[2]. Clinically, BRAF^V600E is associated with more aggressive tumor behavior, including extrathyroidal invasion, lymph node metastasis, and higher recurrence rates[1].

RAS Mutations

Mutations in the Rat sarcoma viral oncogene homolog (RAS) gene family (NRAS, HRAS, KRAS), particularly NRAS codon 61, are more commonly seen in the follicular variant of PTC. These mutations activate both the MAPK and phosphoinositide 3-kinase (PI3K)-AKT pathways, contributing to tumorigenesis. RAS mutations are typically mutually exclusive with BRAF mutations[3].

RET/PTC Rearrangements

REarranged during Transfection (RET)/PTC rearrangements result from fusion between the RET tyrosine kinase domain and various partner genes such as Coiled-Coil Domain Containing 6 (CCDC6) and Nuclear Receptor Coactivator 4 (NCOA4). These rearrangements are predominantly found in radiation-associated PTC and younger patients. They cause constitutive activation of RET and downstream MAPK signaling[4].

Follicular Thyroid Carcinoma (FTC)

Follicular thyroid carcinoma (FTC) accounts for 10–15% of thyroid malignancies and is often associated with vascular invasion and distant metastasis.

RAS Mutations

Similar to PTC, NRAS and HRAS mutations promote activation of the MAPK and PI3K-AKT pathways in FTC[3]. The presence of RAS mutations is useful in differentiating FTC from benign follicular adenomas, as the latter typically lack these alterations.

PAX8-PPARG Fusion

A specific chromosomal translocation t(2;3)(q13;p25) results in the Paired Box 8 (PAX8)–Peroxisome Proliferator-Activated Receptor Gamma (PPARG) fusion gene, observed in 30–35% of FTCs. This fusion produces a chimeric protein that interferes with normal thyroid cell differentiation and promotes oncogenesis[5].

Medullary Thyroid Carcinoma (MTC)

MTC arises from parafollicular or C cells of the thyroid and comprises 3–5% of thyroid carcinomas. It may occur sporadically or as part of inherited syndromes.

RET Mutations

Activating mutations in the RET proto-oncogene are central to the pathogenesis of both sporadic and hereditary MTC. Inherited RET mutations cause multiple endocrine neoplasia type 2 (MEN2) syndromes, including MEN2A, MEN2B, and familial MTC. Mutations at codons 634 and 918 are linked to aggressive disease and early onset[6].

Anaplastic Thyroid Carcinoma (ATC)

Anaplastic thyroid carcinoma is a rare but highly aggressive form of thyroid cancer, often arising from dedifferentiation of pre-existing differentiated thyroid carcinoma.

TP53 Mutations

Tumor protein p53 (TP53), a tumor suppressor gene, is mutated in approximately 70% of ATC cases. This mutation results in loss of cell cycle control, increased genomic instability, and resistance to apoptosis[7].

TERT Promoter Mutations

Mutations in the promoter region of the Telomerase reverse transcriptase (TERT) gene (notably C228T and C250T) are frequently observed in ATC and aggressive PTC subtypes. These mutations enhance telomerase expression, contributing to cellular immortality and tumor progression[8].

Hürthle Cell Carcinoma (HCC)

Hürthle cell carcinoma, a rare and distinct variant, is characterized by oncocytic cells rich in mitochondria. It exhibits more aggressive behavior compared to conventional FTC.

Mitochondrial and Nuclear Mutations

HCCs often harbor mutations in mitochondrial DNA affecting genes involved in oxidative phosphorylation. They also display chromosomal instability and widespread loss of heterozygosity, suggesting a distinct genetic pathway from other follicular neoplasms[9].

Genetic Syndromes Associated with Thyroid Cancer

Cowden Syndrome

Cowden syndrome is an autosomal dominant disorder caused by germline mutations in the Phosphatase and Tensin Homolog (PTEN) tumor suppressor gene, leading to activation of the PI3K-AKT pathway. It is associated with increased risk of follicular thyroid carcinoma and multinodular goiter, requiring regular surveillance[10].

DICER1 Syndrome

DICER1 syndrome is a rare hereditary cancer predisposition syndrome caused by germline mutations in the DICER1 gene, which encodes an RNase III endoribonuclease critical for microRNA processing and gene regulation. Patients with DICER1 mutations have an increased risk of multinodular goiter and differentiated thyroid carcinoma, especially in pediatric populations[12]. Tumors in these patients often demonstrate unique molecular characteristics and a less aggressive clinical course.

Familial Non-Medullary Thyroid Cancer (FNMTC)

FNMTC accounts for 5–10% of non-medullary thyroid cancers. Susceptibility loci identified include Forkhead Box E1 (FOXE1), SLIT-ROBO Rho GTPase Activating Protein 1 (SRGAP1), and Hyaluronan Binding Protein 2 (HABP2), though the precise genetic basis is not fully elucidated[11].

Clinical Implications

Understanding the genetic basis of thyroid carcinoma has revolutionized clinical management. Molecular testing

is now integrated into diagnostic protocols, especially in indeterminate cytology. For instance, detection of BRAF^V600E supports PTC diagnosis and indicates potential resistance to radioiodine therapy. Conversely, RET mutations in MTC guide decisions about prophylactic thyroidectomy and familial screening. Targeted therapies, such as tyrosine kinase inhibitors (TKIs) against RET and BRAF, have shown promise in advanced thyroid cancers, marking a shift toward personalized oncology.

Conclusion

Genetic alterations are central to the pathogenesis, classification, and clinical behavior of thyroid carcinoma. Molecular profiling enhances diagnostic accuracy and facilitates tailored therapeutic interventions. As genomic technologies become more accessible, future research should focus on novel genetic drivers, resistance mechanisms, and combinatorial treatment strategies to improve patient outcomes.

Table 1. Genetic Alterations in Thyroid Carcinoma Subtypes

Thyroid Cancer Subtype	Key Genetic Alterations	Molecular Pathways Affected	Clinical Implications
Papillary Thyroid Carcinoma	BRAF^V600E, NRAS, HRAS, KRAS mutations, RET/PTC rearrangements	MAPK, PI3K-AKT	Aggressiveness (BRAF), radioiodine resistance, younger age (RET/PTC)
Follicular Thyroid Carcinoma	NRAS, HRAS mutations, PAX8-PPARG fusion	MAPK, PI3K-AKT	Differentiation from adenoma, metastatic potential
Medullary Thyroid Carcinoma	RET proto-oncogene mutations (codons 634, 918)	RET tyrosine kinase signaling	Early onset, familial syndromes, targeted TKIs
Anaplastic Thyroid Carcinoma	TP53 mutations, TERT promoter mutations	Loss of tumor suppressor, telomerase activation	Aggressive behavior, poor prognosis
Hürthle Cell Carcinoma	Mitochondrial DNA mutations, chromosomal instability	Oxidative phosphorylation, genomic instability	Distinct molecular pathogenesis, aggressive course
Genetic Syndromes	PTEN (Cowden), DICER1 (DICER1 syndrome), FOXE1, SRGAP1, HABP2 (FNMTC)	Tumor suppressors, miRNA processing, unknown	Increased hereditary risk, surveillance required

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Human-Centered AI in Medical Education: Between Hype, Hope, and Responsibility

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Abstract

Artificial Intelligence (AI) is rapidly transforming healthcare, yet its role in medical education is still evolving. As institutions explore curriculum reform, AI offers opportunities to personalize learning, simulate clinical practice, and streamline assessment. However, its integration raises ethical, pedagogical, and equity concerns. This article highlights the promise and pitfalls of AI in medical education and emphasizes a human-centered, ethically grounded approach to developing AI-literate healthcare professionals.

Keywords: *Artificial Intelligence, Digital Health, Education, Medical Ethics..*

Introduction

Artificial Intelligence (AI) is increasingly shaping modern healthcare, from diagnostics to treatment decisions. While clinical applications are well-established, its role in medical education is still under development. As medical schools pursue curriculum innovation, integrating AI tools and digital literacy into training programs is becoming both relevant and necessary.

Main Text

AI systems can significantly enhance medical education by personalizing learning experiences, adapting to individual performance, and providing timely feedback. AI-powered simulations and virtual patients offer risk-free environments to develop decision-making and procedural skills. These technologies also support educators by automating assessments, generating predictive insights, and reducing administrative workload, potentially increasing educational efficiency[1–2].

Despite its benefits, AI adoption in education presents serious risks. Algorithms trained on biased or non-representative data may reinforce existing inequities, misguide learners, or marginalize vulnerable populations[3]. Over-reliance on automation risks diminishing critical thinking, empathy, and reflective practice—core competencies of ethical healthcare providers. AI should support, not replace, human judgment in the learning process.

To prepare students for AI-integrated healthcare, medical curricula must emphasize competencies such as data literacy, algorithmic transparency, ethical reasoning, and digital professionalism. Learners should understand how AI systems operate, critically evaluate outputs, and effectively communicate these insights to patients. Importantly, educators must also be empowered with the tools, training, and institutional support to implement AI thoughtfully[4].

In low-resource environments, the potential of AI to supplement limited faculty and clinical exposure is compelling. However, indiscriminate adoption risks widening existing disparities. Effective AI deployment must be context-specific, culturally informed, and aligned with local health system capacities. Infrastructure investment, workforce training, and inclusive policy frameworks are essential to equitable implementation.

Conclusion

Medical education is not merely about knowledge transfer, it is about shaping compassionate, critical, and responsible professionals. AI should serve this vision, not distract from it. As institutions integrate AI, the emphasis must remain on cultivating AI-literate, humanistic clinicians. A technology-driven future must still be grounded in ethics, empathy, and equity.

Acknowledgments

N/A

Conflicts of Interest

The author declares no conflicts of interest.

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Aggressive Gastric Adenocarcinoma after Eradication of Helicobacter Pylori: A Rare Case Report

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Abstract

Background

Gastric adenocarcinoma is closely linked to Helicobacter pylori infection. While early gastric cancer has a slow doubling time (577–3,462 days), late-stage disease progresses more rapidly (69–305 days).

Case Presentation

A 39-year-old non-smoker female presented with loss of appetite, vomiting, dysphagia, generalized weakness, and significant weight loss. Initial endoscopy was normal, but biopsy revealed H. pylori-associated gastritis, treated with triple therapy. Symptoms persisted for six months post-eradication. Imaging later showed a hard, nodular mass in the gastric corpus and body, confirmed as invasive diffuse adenocarcinoma. She underwent palliative total gastrectomy with Roux-en-Y esophagojejunostomy.

Conclusion

This case highlights a rare occurrence of rapidly progressive gastric carcinoma developing shortly after H. pylori eradication, emphasizing the need for close follow-up in symptomatic patients.

Keywords: *Advanced gastric cancer, Case report, Roux-en-Y esophagojejunostomy, Signet ring cell carcinoma.*

Introduction

Gastric adenocarcinomas (GA) are malignant tumors of the gastric mucosal glands. GA is the fifth most common carcinoma and ranks fourth in global cancer-related mortality[1]. While environmental and dietary factors contribute to its risk, Helicobacter pylori infection is the major cause, associated with 75% of gastric cancers worldwide[2]. On average, GA takes seven years to progress from mucosa to submucosa and an additional 2–3 years to become advanced cancer[3]. Although GA progression after H. pylori eradication has been reported, it remains rare and poorly understood.

We report a case of unusually rapid GA progression within 6 months of H. pylori eradication.

Case Report

A 39-year-old non-smoker female with no known comorbidities presented to the outpatient department with a six-month history of loss of appetite and vomiting, generalized body weakness for four months, and dysphagia for three months. Vomiting occurred immediately after intake of solid food and was neither blood-stained nor bile-stained. These symptoms were associated with significant unintentional weight loss

(from 58 kg to 52 kg). She also reported progressive dysphagia with a sensation of a foreign body in the throat. Additionally, she experienced melena three to four times during the past week.

There was no significant family history of similar symptoms. Six months earlier, she had visited another clinic where an upper gastrointestinal (GI) endoscopy revealed no abnormalities (Figure 1). However, biopsy from that visit showed Helicobacter pylori-induced chronic active gastritis. She was treated with a triple therapy regimen, but her symptoms persisted despite eradication.

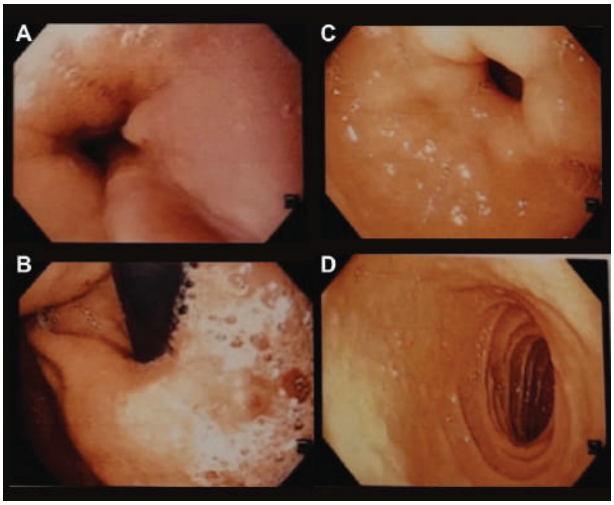

Figure 1. *Upper Gastrointestinal endoscopy showing (A) Gastroesophageal junction (B) Fundus (C) Antrum and (D) First part of duodenum with normal impressions of the GI tract six months ago.*

On her current presentation, the general examination revealed only pallor. Abdominal examination showed a palpable epigastric mass measuring 2 × 2 cm. Laboratory findings were within normal limits, except for microcytic anemia with a hemoglobin level of 8.0 g/dL. Helicobacter pylori testing from biopsy was negative. However, upper GI endoscopy revealed a hard, nodular mass over the corpus and body of the stomach, highly suggestive of malignancy (Figure 2). Biopsy and histopathology confirmed invasive diffuse adenocarcinoma, characterized by ill-defined glands, signet ring cells, and pleomorphic hyperchromatic nuclei (Figure 3). These findings were in stark contrast to the gastroduodenoscopy and biopsy results obtained six months earlier.

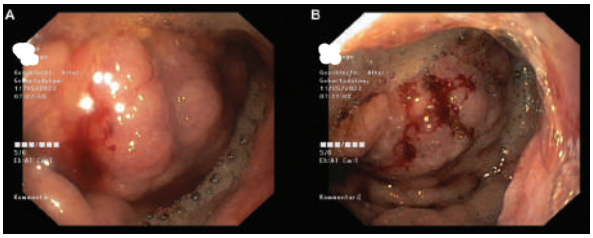

Figure 2. *Upper Gastrointestinal endoscopy showing hard nodular mass over (A) corpus and (B) body likely malignant.*

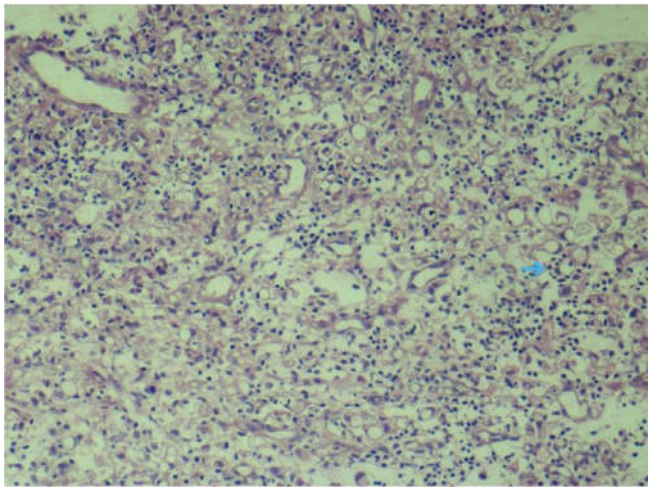

Figure 3. *Hematoxylin and eosin stain (20 X) showing invasive diffuse adenocarcinoma with ill defined glands, signet ring cells and pleomorphic hyperchromatic nuclei.*

An advanced endoscopic nasogastric tube was placed for enteral feeding purposes. Computed tomography (CT) scan found asymmetrical thickening of body and antrum of stomach with mass measuring 3.3 x 4.07 cm in lesser curvature at antrum. The mass was limited within gastric walls with ulceration and perigastric fats and locoregional lymphadenopathy with few homogeneously enhancing likely reactive left paraaortic lymph nodes from which metastasis can't be ruled out (Figure 4).

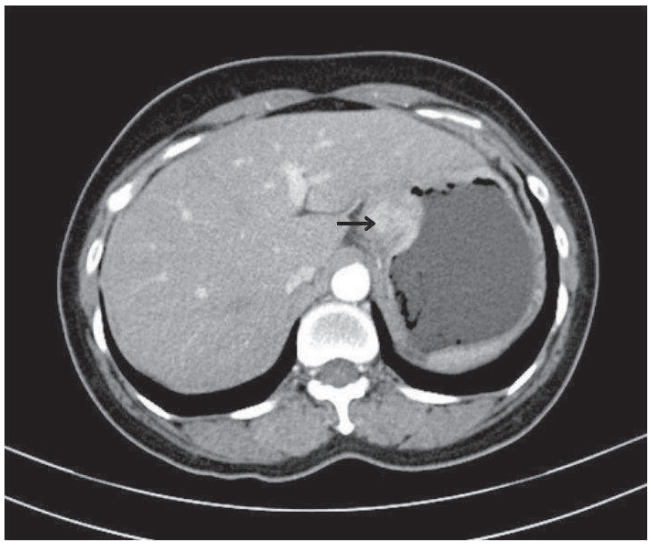

Figure 4. *Computed tomography (CT) scan showing asymmetrical thickening of body and antrum of stomach with locoregional lymphadenopathy.*

Diagnostic laparoscopy was performed to assess abdominal involvement and for surgical planning. It revealed omental deposits, but no parietal peritoneal deposits. Serosanguinous peritoneal fluid was collected and sent for cytological analysis. Intraoperatively, a hard mass was found at the lesser curvature of the stomach, extending proximally to the pylorus and 3 cm below the

gastroesophageal junction, involving the body of the stomach with serosal involvement. The posterior wall of the stomach with the mass was adherent to the superior border of the pancreas but was separable. No additional lesions were observed.

The patient underwent a palliative open total gastrectomy. The left gastric artery was ligated and divided, short gastric vessels were divided, and a Roux-en-Y esophagojejunostomy (end-to-side, antecolic) was performed. The patency of the anastomosis was confirmed intraoperatively. Pathological examination of the surgical specimen revealed a poorly differentiated, invasive diffuse adenocarcinoma. The patient remained stable postoperatively and during the one-month follow-up.

Discussion

The prevalence of gastric carcinoma among endoscopic patients in Nepal increased from 1.8% to 2.4% according to studies conducted in 2013 and 2021[4–5]. The antrum has been identified as the most common site, with chronic Helicobacter pylori infection being the principal cause of non-cardiac gastric cancer[6]. Epidemiological data suggest a male predominance, with a peak incidence in the 51–70 age group, accounting for more than 50% of cases, while patients below 40 years comprise only 6.2%[5].

Histopathologically, gastric cancers are classified into intestinal and diffuse types. The intestinal type is more commonly associated with chronic H. pylori infection, whereas the diffuse type is more prevalent in younger patients[7]. This case report presents a rare occurrence of diffuse-type adenocarcinoma in a young patient following H. pylori eradication.

In our case, the biopsy was negative for H. pylori following treatment with a triple regimen, and no abnormalities were seen on endoscopy. However, due to persistent symptoms, further investigations were performed after six months. CT imaging revealed a tumor involving the body and antrum, and histopathology confirmed invasive diffuse adenocarcinoma. A cohort study in Sweden reported a 0.2% prevalence of gastric carcinoma in patients post-H. pylori eradication[8]. Therefore, the development of the tumor in our patient, in the absence of family history and after eradication therapy, is extremely rare and of uncertain cause.

Time and tumor growth rate can help retrospectively estimate the timing of cancer onset and predict patient survival. However, growth rates vary significantly among individuals[9]. One study found that the risk of severe intestinal metaplasia and differentiated-type gastric cancer increases with age, suggesting it can take up to 10 years for cancer to develop through atrophic gastritis and intestinal metaplasia[3].

Several studies have also described the exponential growth nature of human cancers. Gastric cancer doubling times, assessed via X-ray imaging, ranged from 54 to 3,462 days. For early gastric cancer, doubling times are reported to be 577 to 3,462 days, while advanced gastric cancer ranges from 105 to 305 days[9]. In our case, the appearance of a large tumor within just 180 days after a normal endoscopy raises questions about the tumor's doubling rate and suggests unusually rapid progression.

Genetic testing and neoadjuvant chemotherapy were not performed in this case due to financial limitations, which could have contributed to improved diagnosis and prognosis. Further research is needed to explore the association between H. pylori and rapidly growing tumors in younger patients, along with more effective diagnostic approaches in such rare presentations.

CONCLUSION

Thus, unique growth of gastric adenocarcinoma after H. pylori eradication as presented in this case is a reminder of the ever evolving nature of cancer research and the need of clinicians to recommend long term surveillance for high risk patients and refine understanding for improvement of patient outcomes.

Consent

Written informed consent was obtained ensuring patient's anonymity.

Declaration of competing interest

There are no conflicts of interest.

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Co-occurrence of Marchiafava-Bignami disease and Wernicke's Encephalopathy in a Single Patient: A Rare Case Report

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Abstract

Background

Marchiafava-Bignami disease (MBD) and Wernicke's encephalopathy (WE) are rare alcohol-related neurological disorders caused by chronic malnutrition, particularly thiamine deficiency.

Case Presentation

We report a 56-year-old male with chronic alcoholism who presented with altered mental status, slurred speech, and limb weakness. MRI revealed T2/FLAIR hyperintensities in the genu of the corpus callosum and dorsomedial thalami, with features of both MBD and WE. Diffusion-weighted imaging showed restricted diffusion in the corpus callosum.

Conclusions

This case highlights the rare co-occurrence of MBD and WE in the same patient. Early recognition and vitamin supplementation are key, although prognosis remains guarded in alcohol-related encephalopathies.

Keywords: *Alcohol-Related Disorders, Case Report, Marchiafava-Bignami Disease, Magnetic Resonance Imaging, Thiamine Deficiency, Wernicke Encephalopathy.*

Introduction

Marchiafava-Bignami disease (MBD) and Wernicke encephalopathy (WE) are rare but serious complications of chronic alcoholism, primarily due to thiamine deficiency1. MBD is characterized by demyelination and necrosis of the corpus callosum, while WE typically affects regions around the third and fourth ventricles including the thalami, mammillary bodies, and periaqueductal gray matter2. Clinical features range from confusion and ataxia to coma. Accurate diagnosis requires high clinical suspicion and MRI, which reveals hallmark imaging patterns1,2. We report a unique case of a patient presenting with overlapping radiological features of both MBD and WE, emphasizing the role of MRI in timely diagnosis and treatment initiation.

Case Report

A 56-year-old male with a history of chronic alcohol use disorder arrived at the Emergency Department (ED) due to altered mental status, weakness of bilateral upper and lower limbs, and slurred speech, as reported by a family member. Upon examination in the ED, the patient exhibited hypertension (blood pressure: 170/100 mmHg) and tachycardia (123 beats per minute). Although the rest of his vital signs were otherwise normal, he appeared drowsy, non-verbal, and displayed gait instability. Laboratory results indicated elevated aspartate aminotransferase (205 U/L), while the remaining laboratory tests were within normal limits. Ultrasonography showed features of chronic liver disease with portal hypertension.

The patient is a chronic alcoholic, consuming alcohol daily for the past 30–40 years, typically drinking five glasses of homemade alcohol each day. He had previously been hospitalized for similar problems associated with altered mental status and ataxia in both legs; however, no documentation was available. For the past one and a half months, he experienced decreased appetite, weakness of limbs, reduced interaction, and altered sleep patterns. He visited the psychiatric outpatient department and was diagnosed with alcohol dependence syndrome (ADS)-induced psychosis and was prescribed Tab Fluoxetine, Tab Olanzapine, and Tab Lorazepam. His last intake of alcohol was recorded seven days prior to admission.

A computed tomography (CT) scan of the head showed no significant abnormalities. After receiving primary care, the patient was admitted for further workup. Contrast-enhanced MRI of the brain (Figure 1) revealed T1 hypointense and T2/FLAIR hyperintense lesions in the middle of the genu of the corpus callosum, sparing the dorsal and ventral aspects, showing mild diffusion restriction in DWI images with an ADC value of 0.5×10^{-3} mm^2/s. No areas of hypointense blooming were noted in SWI images. The lesion showed heterogeneous enhancement in the post-contrast study. T2/FLAIR hyperintensity was noted around the tip of the frontal horns of the bilateral lateral ventricles, dorsomedial thalami, basal ganglia, periaqueductal region, and around the fourth ventricle. These lesions showed no diffusion restriction in DWI images and no significant enhancement in the post-contrast study (Figure 1(d)). The combination of chronic alcoholism and specific imaging findings favored the diagnosis of Marchiafava-Bignami disease (MBD) and Wernicke's encephalopathy (WE).

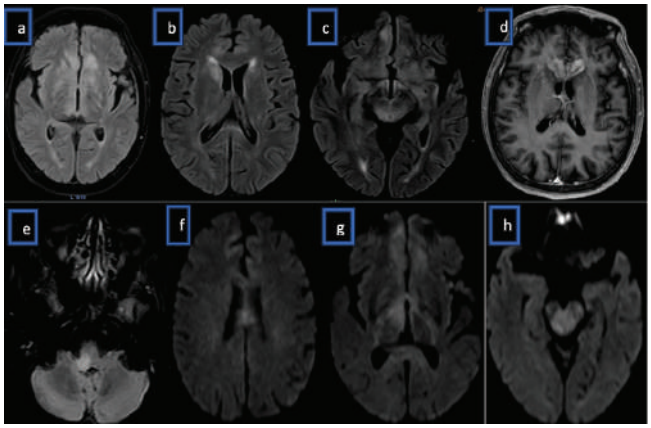

Figure 1. *Axial brain MRI images showing (a, b) FLAIR hyperintensities in the genu of the corpus callosum and dorsomedial thalami; (c) hyperintensities in the periaqueductal region; (d) post-contrast T1 image with heterogeneous enhancement in the genu of the corpus callosum. DWI images (e–h) show diffusion restriction in the genu of the corpus callosum, and (g) bilateral symmetrical hyperintensities in the frontal cortices.*

During hospital admission, neurology and psychiatry consultations were done. The patient was started on intravenous thiamine along with folic acid and multivitamin supplementation. The patient showed slight overall improvement; however, he remained drowsy (GCS E4V3M4). There was minimal progress in daily activities, particularly regarding sitting balance, and weakness in all limbs persisted. A week later, the patient was discharged at the request of family members, as they observed no significant changes in his condition, and a follow-up MRI could not be conducted due to missed appointments.

Discussion

MBD and WE are distinct neurological issues linked to chronic alcoholism. Italian pathologists first identified MBD through observations of corpus callosum degeneration in heavy red wine drinkers, leading to seizures and coma. This rare disorder primarily affects long-term alcohol users and is characterized by demyelination, necrosis, and sometimes hemorrhages, often due to thiamine and folate deficiencies[3]. While 92.8% of MBD cases occur in those with alcohol dependence, it can also affect non-drinkers with conditions like depression or post-bariatric surgery complications[4]. Alcohol disrupts protein expression in white matter, lipid metabolism, and blood vessel integrity, causing small vessel necrosis, blood-brain barrier disruption, and cytotoxic edema, resulting in symmetric demyelination and ischemic damage to the corpus callosum and other central nervous system structures[2,4].

Clinical manifestations vary significantly and are categorized into three types: acute (mental clarity, ataxia, delirium, dysarthria, coma, or death), subacute (confusion, memory issues, walking difficulties, behavioral changes, excessive sleepiness), and chronic (interhemispheric disconnection syndrome, progressive dementia, unusual behaviors)[3]. Heinrich et al. (2004) identified two main clinic-radiological subtypes: Type A MBD, which presents subacutely with hypertonia, pyramidal signs, altered consciousness, and a poor prognosis, and Type B MBD, characterized by gait issues, interhemispheric disconnection syndrome, and a generally positive prognosis with normal to mild cognitive impairment[5].

Historically, postmortem tissue examination was essential for diagnosing MBD. Now, diagnosis primarily relies on clinical assessment and radiological evaluations, with MRI as the "gold standard." CT scans typically show MBD as a hypodense corpus callosum, while hemorrhages may appear isodense or hyperdense. MRI reveals hypointense lesions in the corpus callosum on T1-weighted images and hyperintense lesions on T2/FLAIR, and DWI sequences, often affecting the cortex and white matter without mass effects. Early stages show reduced apparent diffusion coefficient (ADC) values

due to cytotoxic edema, while later stages may show increased ADC values, indicating demyelination without axonal damage[4]. Magnetic resonance spectroscopy (MRS) indicates elevated choline levels and a high choline/creatine (Cho/Cr) ratio during the acute phase, along with a lactate peak during acute or subacute demyelination. Single-photon emission computed tomography (SPECT) shows bilateral decreases in cerebral blood flow. In acute MBD, the "sandwich sign" appears as hyperintensity in the central corpus callosum on T2/FLAIR images, while chronic lesions may show well-defined cavitations[3,4].

In WE, the medial thalamic nuclei, tegmentum, periaqueductal gray matter, tectal plate, and mammillary bodies around the third ventricle are most affected due to their high oxidative metabolism and susceptibility to thiamine deficiency. MRI shows increased T2/FLAIR hyperintensity involving these regions along with contrast enhancement and restricted diffusion[6].

The enhancement in the corpus callosum likely indicated active inflammation or demyelination related to MBD, while the lack of enhancement in the mammillary bodies, periaqueductal region, and midbrain suggested a chronic condition of WE. Our patient's clinical features and the imaging (CT and MRI) findings suggested a patient presented with MBD rather than WE.

When assessing MBD and WE, differential diagnoses like Korsakoff syndrome, and combined Wernicke-Korsakoff syndrome, as well as demyelinating disorders (e.g., multiple sclerosis), recurrent artery of Heubner infarction, central pontine myelinolysis, and tumors such as astrocytoma and lymphoma should be considered[7].

There is no standardized treatment for MBD, but early thiamine and folic acid administration can aid recovery, while corticosteroids may reduce inflammation. The effectiveness of amantadine is unclear, as it has only been tested with vitamin B[3,8]. Significant clinical improvement was not seen following vitamin supplement treatment in our case. It has been suggested that the combination of MBD and WE influenced the prognosis. A limitation of this case report is the inability to obtain follow-up MRI images, as the family declined further treatment and follow-up imaging was unsuccessful.

MBD has a high mortality rate, particularly in its acute phase, and can lead to various outcomes, including recovery, persistent symptoms, vegetative state and death[9]. Over half of non-alcoholic MBD patients fully recover, compared to about 10% of those with alcoholic MBD. Survivors often experience significant neurological impairments, though some may recover partially or completely[8,9]. Survivors should focus on quitting alcohol, seeking rehabilitation, and obtaining nutritional support.

Conclusion

This case demonstrates the rare co-occurrence of Marchiafava-Bignami disease and Wernicke's encephalopathy in a patient with chronic alcoholism. MRI plays a pivotal role in diagnosis. While treatment with thiamine and nutritional support is essential, outcomes depend on early recognition and intervention. This report highlights the need for heightened clinical suspicion and the importance of addressing chronic alcohol use and malnutrition.

Consent

Written informed consent was obtained ensuring patient's anonymity.

Declaration of competing interest

There are no conflicts of interest.

Acknowledgement

N/A

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Reimagining Medical Education: Embracing the Future of Healthcare

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Introduction

In an era characterized by rapid technological advancements and evolving patient needs, the field of medical education stands at a pivotal crossroads. Traditional paradigms are increasingly being challenged, necessitating a reimagining of how we train the next generation of healthcare professionals. To effectively prepare medical students for the complexities of modern healthcare, it is imperative that we embrace innovative educational strategies, integrate interdisciplinary approaches, and prioritize patient-centered care.

Main Body

Medical education has long been rooted in a didactic approach, with a strong emphasis on memorization and rote learning. However, as the landscape of healthcare continues to evolve, there is a growing recognition of the need for educational models that foster critical thinking, problem-solving skills, and adaptability. The incorporation of technology, such as simulation-based training and digital health tools, offers promising avenues for enhancing learning outcomes and preparing students for real-world challenges[1].

Simulation-based training, for instance, allows students to practice clinical skills in a controlled environment, providing immediate feedback and opportunities for iterative learning. This approach has been shown to improve both technical skills and clinical decision-making abilities[2]. Furthermore, the integration of virtual reality and augmented reality into medical education is beginning to transform how complex procedures and anatomical concepts are taught, offering immersive and interactive learning experiences[3].

The complexities of modern healthcare demand a more holistic approach to education, one that transcends traditional disciplinary boundaries. Interdisciplinary education where medical students collaborate with peers from nursing, pharmacy, social work, and other fields can significantly enhance the quality of care delivered to patients[4]. This collaborative approach not only improves communication and teamwork skills but also fosters a deeper understanding of the roles and contributions of different healthcare professionals.

Recent studies have demonstrated that interdisciplinary education improves patient outcomes and enhances the overall effectiveness of healthcare teams[5]. By working together on case studies, simulations, and clinical rotations, students gain valuable insights into the complexities of patient care and develop skills that are critical for effective interprofessional collaboration.

Central to any reimagined medical education model is a focus on patient-centered care. This approach emphasizes understanding patients' needs, values, and preferences, and integrating them into the care process. Training programs that incorporate patient-centered principles such as empathy, communication skills, and shared decision-making prepare students to provide more compassionate and effective care.

Patient-centered care is increasingly recognized as a key component of high-quality healthcare. Studies have shown that patients who experience care that is attentive to their individual needs and preferences are more likely to have better health outcomes and higher satisfaction with their care[6]. Integrating these principles into medical education not only enhances the quality of care but also fosters a more empathetic and patient-focused healthcare workforce.

Conclusion

As we look to the future of medical education, it is clear that innovation, interdisciplinary collaboration, and a focus on patient-centered care are essential to preparing

students for the evolving demands of the healthcare landscape. By embracing these principles, medical schools can better equip future healthcare professionals with the skills and knowledge necessary to deliver high-quality, compassionate care.

The journey towards an evolved medical education system is ongoing, and it requires the collective efforts of educators, institutions, and policymakers. By prioritizing these forward-thinking approaches, we can ensure that the next generation of healthcare providers is not only well-prepared for the challenges ahead but also capable of driving positive change in the world of medicine.

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Case of Non Healing Chronic Venous Ulcer Healed after COVID-19

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Abstract

Background

Chronic venous insufficiency with edema, skin changes, and ulceration indicates advanced chronic venous disease. Active venous ulcers usually heal with proper wound care and surgical treatment of underlying venous abnormalities. However, ulcers associated with obstructive venous pathology, incompetent perforators, and varicose veins often heal poorly or recur frequently. The association between venous ulcer progression and COVID-19 is poorly understood.

Case Report

We report a case of a 55-year-old female with a chronic non-healing venous ulcer, venous hypertension, and varicose veins, whose ulcer healed following COVID-19 infection. This improvement is suspected to be related to COVID-19-associated coagulability (CAC).

Conclusion

This case suggests that COVID-19 infection may influence the healing of chronic venous ulcers, potentially through alterations in coagulation pathways, warranting further investigation.

Keywords: *Case report, COVID-19, Deep Vein Thrombosis, Sclerotherapy, Venous ulcer.*

Introduction

Venous leg ulcers are responsible for 60-80% of the leg ulcers and are prevalent among 0.18-1% of the general population.[1] Non healing venous ulcers are common in cases of deep vein thrombosis, varicose veins, chronic venous insufficiency and are associated with significant morbidity affecting quality of life.[2] Treatment for venous ulcers includes compression therapy, leg elevation, pharmacotherapy and surgical treatment[3] Here, we report a case of improved prevailing venous ulcer following COVID-19 infection. This manuscript has been reported according to SCARE 2020 criteria.[4]

Case Report

A 55-year-old female presented to the outpatient department with a history of improvement of prevailing venous ulcer in 2 weeks time following infection with COVID-19. She was diagnosed with symptomatic familial Deep Vein Thrombosis (DVT) fifteen years back with history of bilateral lower limb swelling with DVT involving common femoral vein, superficial femoral vein and popliteal veins on both the sides. On examination for the past thirteen years, she had bilaterally prominent dilated veins in lower leg along with a non healing ulcer in the inner aspect of right leg just above the ankle joint.

She has been taking warfarin regularly since the last fifteen years with dose adjusted to maintain International Normalized Ratio (INR) 2-3.

Radiological investigations were sent and the imaging revealed bilateral secondary varicose veins with multiple incompetent lower leg perforators. She had two surgical procedures involving ligation of incompetent perforators along with two sessions of targeted sclerotherapy. The ulcer is deep with irregular border and clear watery discharge. The skin around the ulcer shows purple pigmentation (figure 1).

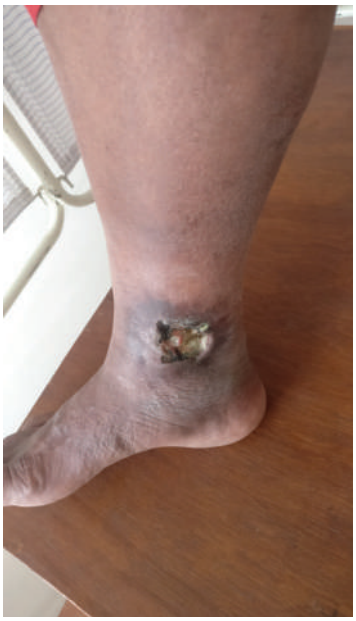

Figure 1. *Chronic venous ulcer on the medial aspect of the right ankle.*

However, the ulcer showed no signs of healing for 7 years despite daily dressing, multiple layered bandaging and sclerotherapy. These procedures helped for some symptomatic improvement in terms of pain and discharge from ulcer but the ulcer had never healed for seven years.

She had a mild COVID-19 infection six months back and was treated at home. The symptoms resolved in a week. However, in follow-up after a month of COVID-19 infection, her ulcer surprisingly started healing. The ulcer appeared shallow with irregular borders and the overlying wound bed has yellow fibrinous exudate formation with no discharge (Figure 2). Her other symptoms. due to ulceration like discharge and pain had also subsided. Doppler ultrasonography revealed thrombosis of all the tributaries under the ulcer.

Discussion

Venous ulcers usually present as a sequelae of Deep Vein Thrombosis (DVT), varicose veins, perforator or muscle pumping insufficiency of the lower limb.1,5,6 There are superficial and deep veins; drainage from deep vein is facilitated by perforator vein and pump action of calf muscles.5 The calf muscle pump with good dorsiflexion of the index leg and a higher percentage of volume of blood displaced are the potential protective factors of ulceration.[5]

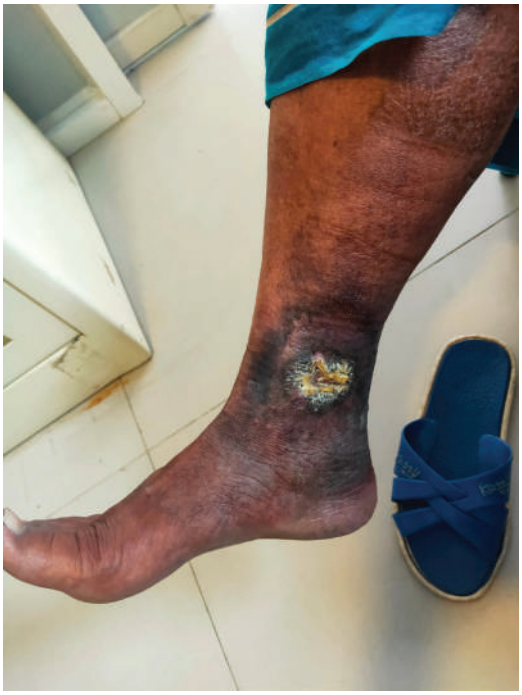

Figure 2. *Healing venous ulcer on the medial aspect of the right ankle.*

Damage to both calf muscle and perforator veins can lead to venous pooling of blood which leads to venous hypertension and ulceration later on.[5] Incompetent venous reflux or venous obstruction also causes increased permeability and leakage of hemosiderin in the skin changing its texture and elasticity.[6]

The risk factors for development of venous ulcer include advancing age, family history of venous diseases, physical inactivity and/or history of prolonged standing, obesity, previous thromboembolic events, DVT, high estrogen state, varicose veins and factor V Leiden mutation.[1,7] Larger wounds, ulcer duration of >1 year, fibrin in >50% of wound surface, history of venous ligation are the poor prognostic factors to this disease.1 While venous ulcers present as a sequelae of DVT and varicose veins, incidence of concomitant DVT and varicose veins is rare.[8] No cases of concomitant DVT and varicose veins have been reported in association with venous ulcers in Nepal to our knowledge. Furthermore, the role of COVID-19 in regard to the course of the venous ulcers has not been explored yet.

The patient in our case is an elderly female with DVT and varicose veins with a family history of venous disease (DVT), all of which are associated with risk of developing venous ulcer. Additionally, the venous ulcer had persisted for seven long years and had no signs of healing suggesting poor prognostic characteristics.

Venous ulcers predominantly occur in the lower third of the leg around the medial malleolus also known as gaiter's area.[2,9] This finding was consistent with our case, where the patient has venous ulcer on the right gaiter region.

Venous thromboemboli are the most frequently occurring thrombotic event in COVID-19. Endothelial inflammation, breakdown of intercellular junction and the production of microthrombi are all distinct microvascular anomalies in COVID-19.[10] A meta-analysis of 42 studies involving 8271 patients showed that overall venous thromboembolic events rate was 21%, and DVT rate was 20% among SARS-CoV-2 infected patients; severely infected ones being at higher risk.[11]

A case control study among 241 people aged 40 to 99 years estimated people who had a diagnosed thrombo-embolism were at almost three times higher risk of having a leg ulcer.[12] Robertson, et al. in a case control study of 240 people estimated that patients with varicose veins (most common in great saphenous vein followed by short saphenous vein), chronic venous insufficiency and deep vein incompetence are at greatly increased risk of ulceration.[5]

Treatment options for venous leg ulcers include compression therapy, leg elevation, medications(Pentoxifylline, aspirin) and surgical treatment(debridement, skin grafting) among which compression therapy is the standard of care.[3] General patients diagnosed with DVT with venous ulceration have ulcer healing in about 80% within 24 weeks following appropriate treatment.[13] Nelzen et al. concluded that the overall ulcer healing for patients with varicose veins treated conservatively was about 60% at 5 years in his prospective cohort study among 382 participants 14 However in case of venous ulceration due to concomitant DVT and varicose veins, ulcer healing is exceedingly rare despite adequate management.[8]

In our case, she had a history of symptomatic DVT (familial and recurrent), bilateral secondary varicose veins along with non healing ulcers for thirteen years. Despite intervention and adequate compression therapy, her ulcer hasn't healed for thirteen years. However, her ulcer started healing following COVID-19 infection. Venous ulceration was at stage C6 before she contracted COVID. After COVID, the ulcer started to heal and turned to stage C5.

Thrombosis as a result of SARS CoV2 infection in the veins underlying venous ulcer might have eliminated the processes responsible for recurrent discharge from the ulcer. The discharge usually is rich in proteolytic enzymes causing proteolysis of growth factors that was required for healing. The removal of discharge might have reduced the proteolytic activity enhancing the growth factors function and thereby, healing the venous ulcer.[15]

Conclusion

Venous ulcers associated with Deep Vein Thrombosis (DVT) and varicose veins, despite appropriate management, are linked with delayed healing. Moreover, literature suggests that the venous thromboembolic pathology of COVID-19 infection is associated with poor prognosis of venous diseases.11. Contrary to this, the patient in our case had a remarkable healing of the venous ulcer from stage C6 to C5 post COVID-19 infection.

Consent

Written informed consent was obtained ensuring patient's anonymity.

Conflicts of Interest

There are no conflicts of interest.

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Sources of Reflux in Patients Diagnosed with Varicose Veins of Great Saphenous Vein Subjected for Radiofrequency Ablation: A Cross Sectional Study

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Abstract

Background

Varicose veins result from defective valves in the venous system, particularly at the junctions between the superficial and deep veins. Understanding the reflux sites in the great saphenous vein (GSV) is crucial for planning surgical procedures, such as Radiofrequency Ablation (RFA), and preventing recurrence.

Methods

This cross-sectional study analyzed patients who underwent RFA for GSV varicosities from July 1 to June 30, 2023. GSV mapping was conducted to identify reflux in the saphenofemoral junction (SFJ) and perforators, classified by location (mid-thigh, above knee, below knee, mid-calf, above ankle). Reflux was defined by a reflux time >500 ms and/or peak velocity >30 cm/s.

Results

Among 211 patients, 27.5% had only right lower limb involvement, 39.8% had only left, and 32.7% had both limbs affected. SFJ incompetence was found in 100% of right limbs and 96.7% of left limbs. Perforator incompetence was observed in 63 sites on the right and 82 on the left, with the mid-calf perforator being most common.

Conclusion

Reflux in the SFJ is predominant in GSV varicosities, but perforator reflux is also significant. Identifying reflux sites is essential for effective surgical planning to prevent recurrence.

Varicose veins are caused due to defective valves between the superficial and deep venous systems. These defects can occur at the level of the main junction between superficial and deep systems or at the level of various perforators. Understanding of these systems help in the surgical procedures for varicose veins and will help prevent their recurrence. This study is aimed to identify the reflux sites in great saphenous veins (GSV) in patients diagnosed as varicose veins of GSV and subjected for RFA with or without adjunct procedure.

Keywords: *Lower Extremity Varicosities, Superficial veins, Perforating veins, Reflux, Sapheno femoral junction*

Introduction

Varicose veins are dilated superficial veins, commonly in the lower limbs, and are associated with pain, pigmentation, and ulceration[1]. They result from valve incompetence, particularly at junctions between superficial and deep veins, and affect 15–25% of the population[2–4]. These junctions include the saphenofemoral and saphenopopliteal junctions, as well as anatomically classified perforators (e.g., mid-thigh, mid-calf)[5]. Perforators may be direct (connecting superficial to deep veins) or indirect (connecting to muscular veins)[6]. Surgical treatment like RFA requires accurate identification of reflux sites, as missed incompetent perforators are a common cause of recurrence[7]. The CHIVA protocol emphasizes targeting escape points, making reflux mapping essential[1,8,9]. Persistent perforator incompetence increases the risk of recurrent ulcers[10]. While both GSV and SSV are involved, GSV has longer courses and more reflux points, necessitating detailed mapping. Intraoperative Doppler helps identify sites not reached by RFA, such as mid-calf perforators[11]. This study aims to identify GSV reflux sites in patients undergoing RFA.

Methods

This retrospective study included all patients who underwent radiofrequency ablation (RFA) for great saphenous vein (GSV) varicosities at Dhulikhel Hospital between July 1, 2023, and June 30, 2024. Cases with recurrent varicose veins were excluded. Ethical approval was obtained from the Institutional Review Committee of Kathmandu University School of Medical Sciences, and written informed consent was taken.

Bilateral procedures were analyzed as separate cases. Preoperative mapping of the GSV was performed by a consultant vascular surgeon with patients standing in Doppler stands. A portable ultrasound machine (Acuson P300, Siemens Medical Solutions USA, Inc.) with a 5–10 MHz linear probe was used[12,13]. Reflux at the saphenofemoral junction (SFJ) or perforators was defined as reflux time >500 ms and/or peak velocity >30 cm/s. Perforators were classified anatomically: mid-thigh, above-knee, below-knee, mid-calf, and above-ankle.

RFA cannulation was done at a site with GSV diameter >5 mm, straight segment ≥5 cm, and depth <5 mm[14], usually proximal to the distal insufficiency point. In cases of distal GSV dilation, cannulation was performed at the mid-calf to avoid saphenous nerve injury[15]. The VNUS ClosureFast RF generator was used, targeting 120°C within 3 seconds and maintaining it for 20 seconds at 10–20 W output. After ablation, distal GSV segments were scanned for residual reflux and managed as needed, often with perforator ligation.

Data was entered in Microsoft Excel and analyzed using SPSS v19. Scalar variables were expressed as mean ± standard deviation; categorical variables as percentages. Independent t-test was used for comparisons, with $p < 0.05$ considered significant.

Results

There were a total of 211 patients, 110 male patients (52.1%) and 101 female patients (47.9%). Mean age was 44.23 years (range 18-78, standard deviation 13.9). The mean age in male patient was 43.80 years and that in the female patient was 44.69 (p=0.643).

The right lower limb alone was involved in 58 patients (27.5%). The left lower limb alone was involved in 84 patients (39.8%). In 69 patients (32.7%) both lower limbs were involved and thus bilateral RFA was done.

In patients where the right lower limb was involved, all cases had SFJ incompetence. Table 1 shows the involved perforators. There was perforator incompetence in a total of 63 sites. The most common perforator involved in the right lower limb was the mid calf followed by the above ankle.

Table 1. Involved perforator in right lower limb varicose veins involving GSV and subjected for RFA

Involved perforator	Number	Percentage
Mid calf	27	21.3
Above ankle	19	15
Below knee	11	8.7
Above knee	6	4.7

In the left lower limb there were 148 cases with incompetent SFJ (96.7%) and five cases (3.3%) where SFJ was competent. In those five cases, they had multiple perforator incompetent causing long segment GSV dilatation requiring RFA.

Table 2 shows the number and percentage of different perforators which were incompetent in the cases. There was perforator incompetence in 82 sites. The most common incompetent perforator in the left side was also mid calf perforator followed by below knee and above knee.

Table 2. Involved perforator in left lower limb varicose veins involving GSV and subjected for RFA

Involved perforator	Number	Percentage
Mid calf	39	25.5
Below knee	21	13.7
Above knee	12	7.8
Above ankle	10	6.5

Out of 211 patients, presence of perforator incompetence was noted in 107 patients (50.7%). The number of perforator incompetence in a patient is shown in Table 3. Besides no perforator incompetence, the most common number of perforator incompetent was one (27.5%) followed by two (17%). There were a maximum of four incompetent perforators in a patient. Mean number of perforator incompetence in each patient was 0.80.

Table 3. Number of perforator incompetence

Number of perforator incompetence in patient	Number	Percentage
0	104	49.3
1	58	27.5
2	36	17.0
3	12	5.7
4	1	0.5

Discussion

Although varicose veins are generally more common in females, our study observed a higher prevalence among male patients[16]. This may reflect gender differences in health-seeking behavior in our region, where men often the primary earners, are more proactive about seeking medical care, whereas women tend to rely on traditional remedies due to limited financial autonomy[17].

Identifying the reflux site is critical for formulating an effective treatment strategy. In our study, all patients with right-sided GSV varicosities had saphenofemoral junction (SFJ) reflux. Among those with left-sided involvement, 96.7% showed SFJ reflux. A study by Carrison et al. analyzing 1027 cases of varicose veins found SFJ reflux in 82% of cases with full-length GSV involvement[18]. The higher proportion of SFJ involvement in our cohort likely reflects our inclusion criteria; patients selected for RFA typically have long-segment GSV involvement. Localized GSV varicosities, often associated with isolated perforator incompetence, were not included.

Perforator incompetence was identified in 50.7% of patients in our study. This is consistent with findings from a study by Pant et al. in Nepal, where at least one incompetent perforator was noted in 59.4% of cases[19]. Similarly, Tolu et al. reported perforator-related incompetence in 44.7% of patients[10]. The most commonly involved perforator in our study was the mid-calf perforator on both sides. On the right side, the second most common site was the above-ankle perforator, while on the left it was below the knee. Our earlier study also reported the mid-calf perforator as the most frequently involved site, followed by the above-ankle perforator[20]. This pattern was also noted in the study by Pant et al.[19] Given the frequent involvement of mid-calf perforators, they should be evaluated and treated during above-knee RFA when present.

The CHIVA (Conservative and Hemodynamic treatment of Venous Insufficiency in the Office) approach emphasizes treating only the refluxing segments while preserving normal venous anatomy[8]. For this, precise knowledge of reflux locations is essential. Our study provides valuable insight into the pattern of SFJ and perforator involvement, which may aid surgical planning.

In a study evaluating superficial reflux in patients with venous ulcers, perforator incompetence was found in 81.4% of patients with ulcers in the lateral region (44 of 54 cases), and in 91.3% of those with ulcers in the medial region (94 of 103 cases)[21]. Overall, 89% of patients in that study had perforator incompetence[21]. These findings further underscore the importance of thoroughly evaluating perforators when managing chronic venous insufficiency.

Conclusion

Identifying the site of reflux is essential for effective surgical planning and ensures that all incompetent points are addressed during intervention for varicose veins. While saphenofemoral junction (SFJ) reflux is commonly observed in GSV-related varicosities, our study highlights that a significant proportion of patients also exhibit reflux at the perforator level, underscoring the need for comprehensive preoperative evaluation.

Consent

Written informed consent was obtained from each patient ensuring anonymity.

Declaration of competing interest

There are no conflicts of interest.

Acknowledgement

N/A

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