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Case Study *Open Access*

A Cheek Mass Masquerade: Kimura Disease Mimicking a Slow-Flow Vascular Malformation with Radiological and Pathological Correlation

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Abstract

Kimura disease is a rare chronic inflammatory disorder predominantly affecting the head and neck region, often presenting as painless subcutaneous masses with associated lymphadenopathy, eosinophilia and raised serum IgE counts. It shows male preponderance, particularly affecting the Asian males of 18-40 yrs age groups. Due to overlapping imaging features, it can mimic vascular malformations and neoplastic conditions. We report a case of a 20 years old male presenting with a gradually enlarging left cheek swelling, initially suspected to be a slow-flow vascular malformation on Ultrasound and referred to tertiary care centre for Digital Subtraction Angiography (DSA). Subsequently patient underwent MRI and retrospectively repeat USG revealed findings suspicious for infective/inflammatory pathology. Cytological examination suggested features in favour of Kimura's disease and further warranted need of HPE. Excisional biopsy was performed and post-operative histopathological examination confirmed Kimura disease. This case highlights the importance of considering this in differential diagnoses of vascular-appearing soft tissue lesions and underscores the role of histopathology in definitive diagnosis.

Keywords: *Kimura disease, Vascular malformation, Cheek Swelling, Eosinophilia, Lymphadenopathy.*

INTRODUCTION

Kimura disease is a rare benign chronic inflammatory condition of unknown aetiology, which typically presents as painless subcutaneous masses in the head and neck region [1], often associated with regional lymphadenopathy and elevated serum IgE levels.

Radiologically, Kimura disease can mimic various conditions including vascular malformations, lymphoma, and salivary gland tumours [7]. Due to its rarity and nonspecific imaging features, misdiagnosis is common, making histopathological confirmation essential [2]

Our patient's presentation of Kimura's disease is quite unusual due to its early misdiagnosis. There are quite a significant number of research papers on Kimura disease and slow flow venous malformation separately [8]. Few research papers are dedicated on mimickers including Angio lymphoid hyperplasia with Eosinophilia (ALHE) [9], however no significant research articles dedicated to slow flow venous malformation mimicking Kimura disease exist at present time. Hence this unique case will be of significant interest particularly in the field of radiological diagnosis and clinical correlation.

CASE PRESENTATION

A 20 years old male presented with a history of painless swelling over the left cheek (as shown in Fig.1) for 3 months, gradually increasing in size. On further examination, subtle swelling was also noted over the left superolateral orbital region. No antecedent history of trauma present. Prior outside USG revealed heterogeneously hypoechoic lesion in the subcutaneous plane of left cheek region with multiple dilated anechoic channels. Serum IgE levels were not available at the time of evaluation. However peripheral eosinophilia was noted.

Fig.1. Case representation highlighting left cheek swelling.

Imaging Findings

MRI Findings

MRI of the face and neck (Plain and Contrast Angiography) revealed:

Ill-defined heterogeneous enhancing altered signal intensity lesion in the subcutaneous and intermuscular plane of the left parotid region, which appears isointense on T1-weighted images and heterogeneously hyperintense on T2/STIR sequences, with patchy areas of diffusion restriction. Distinct fat planes are maintained with the left parotid gland which showed normal signal intensity. On TWIST angiographic sequences, there is no evidence of an obvious arterial feeder or draining vein. The lesion produces a contoural bulge involving the subcutaneous tissues and overlying skin, while the fat planes with adjacent structures being preserved. Additional findings include multiple enlarged cervical lymph nodes involving bilateral levels I–IV. There is also an incidentally enlarged left lacrimal gland demonstrating diffusion restriction, consistent with dacryoadenitis. Furthermore, small lesions with similar morphology are noted in the sublingual space. Based on these findings, possibility of infective/inflammatory aetiology with phlegmon formation and lymphadenitis was raised.

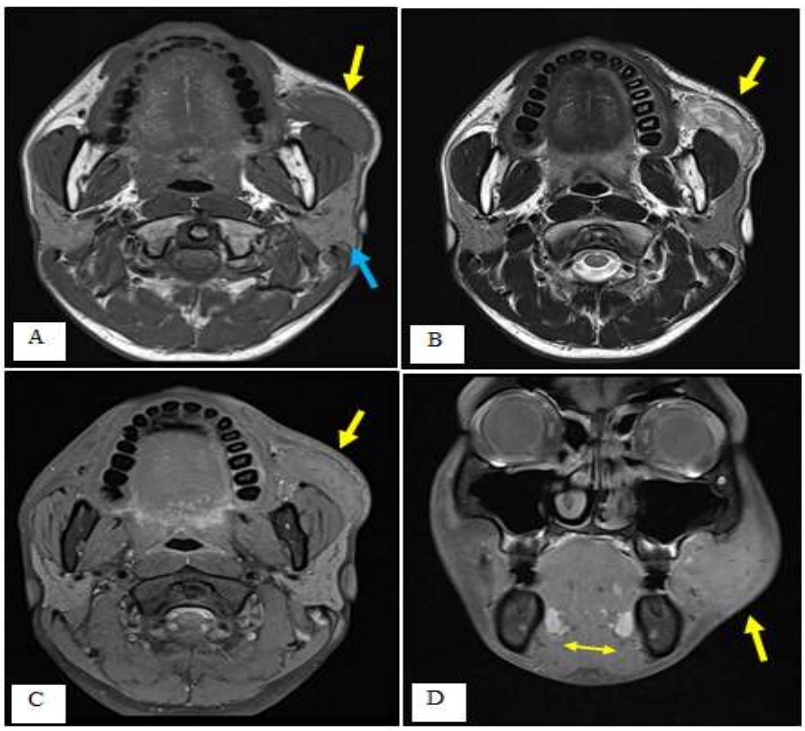

Fig. 2. A. T1 WI in axial view shows a relatively well-defined, oval lesion in subcutaneous plane of left cheek (yellow arrow) which is appearing mildly hypointense to adjacent muscles. No extension into the surrounding muscles of

mastication and left parotid gland (blue arrow). 2.B. T2 WI in axial view shows moderately hyperintense signal of the lesion, however not as bright as cystic area. 2.C. T1 Fat saturated weighted axial image shows isointense to mildly hypointense lesion. No obvious areas of signal drop to suggest fat suppression. 2.D. T1 Fat saturated weighted image in coronal view shows bilaterally enlarged lymph nodes in the floor of mouth (double headed yellow arrows).

Fig.3. T2 STIR coronal images show heterogeneously hyperintense lesion. No perilesional oedema seen.

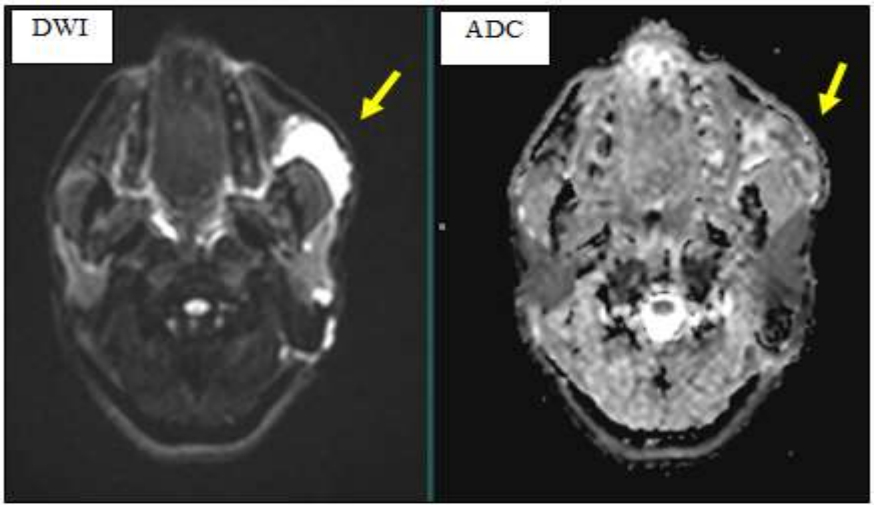

Fig.4. DWI and ADC sequences show significant diffusion restriction with corresponding drop of signal on ADC sequences.

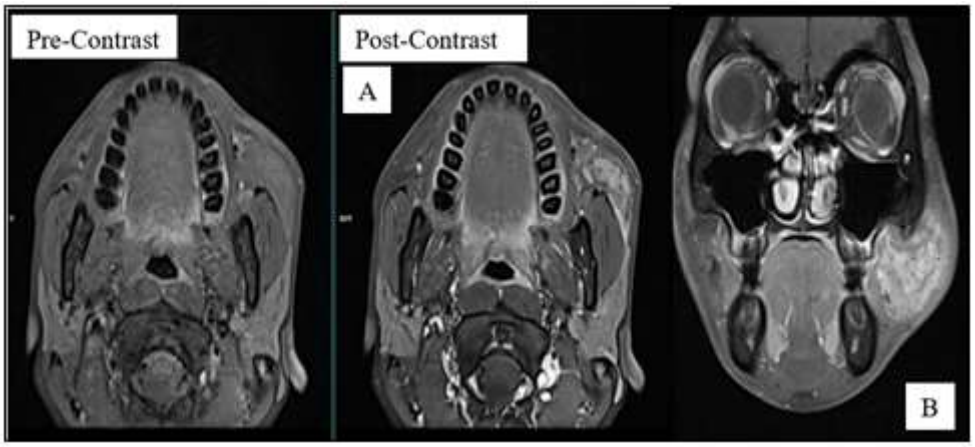

Fig.5. Pre and Post-contrast sequences in axial and coronal views (4.A, 4.B) reveals heterogenous enhancement reflecting rich vascular proliferation.

Fig.6. Post contrast MR images in coronal view shows diffuse enlargement of left lacrimal gland showing intense homogenous enhancement.

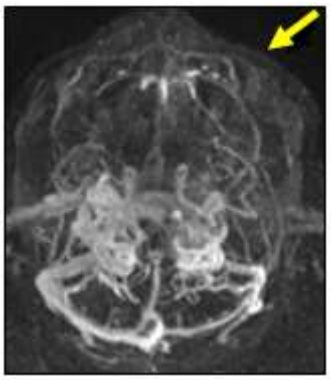

Fig.7. TWIST angiography sequences in axial view reveals no obvious arterial feeders or draining veins.

Ultrasound Findings

USG of the local cheek region (B-mode, colour and doppler) revealed:

Ill-defined heterogeneously hypoechoic lesion in the subcutaneous and intermuscular plane of left cheek with multiple anechoic channel-like areas, initially mimicking vascular spaces. However, colour doppler demonstrates internal vascularity within the lesion without true flow and no spectral waveform within the anechoic channels.

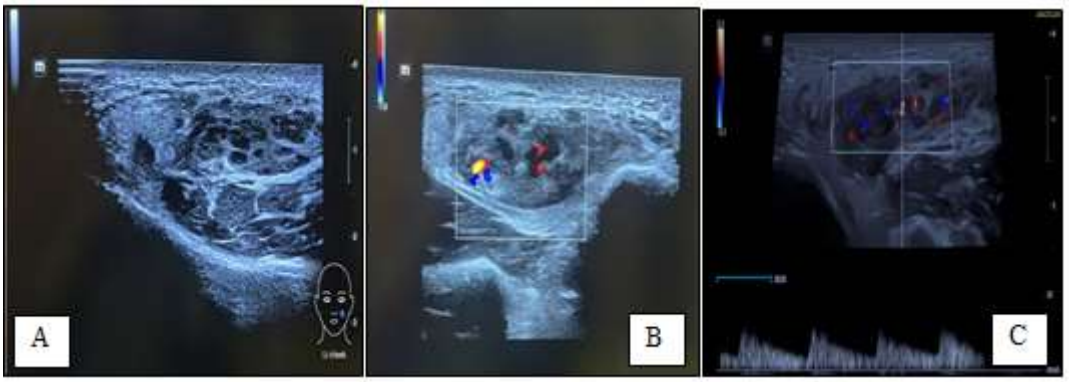

Fig.8. A, B, C - Ultrasound of the left cheek showing a heterogeneously hypoechoic lesion in the subcutaneous plane of left cheek region with multiple anechoic channel-like areas, initially mimicking vascular spaces. Colour Doppler demonstrates internal vascularity within the lesion without true flow within the anechoic channels.

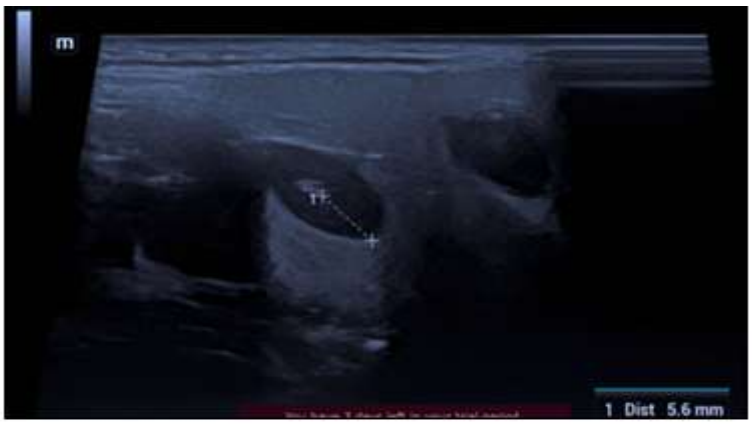

Fig.9. USG Neck – multiple lymph nodes with thickened cortex seen in bilateral Ib, II and III levels of cervical region.

Pathological Findings

Cytology

A polymorphous reactive lymphoid population is seen, predominantly composed of eosinophils, along with surrounding fibrocollagenous and vascular stromal fragments, multinucleated giant cells [1,3], and hyaline material arranged in loose sheets, Fig.10. A.

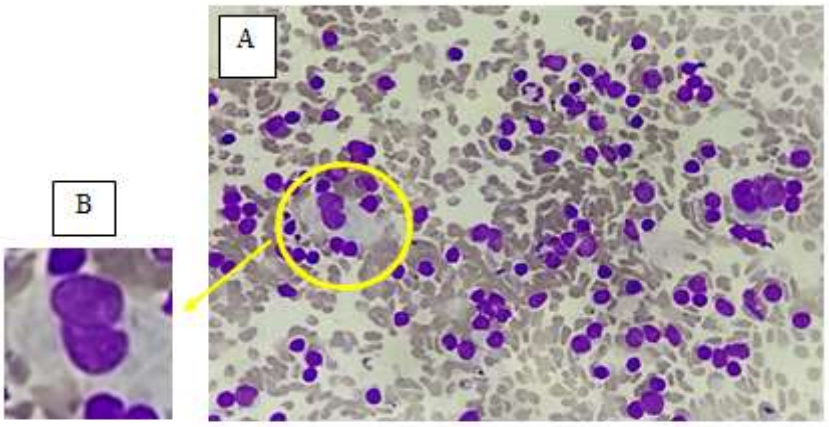

Fig.10. A. Cytology images, 10.B. Zoomed in view of bilobed eosinophil.

Histopathology

A well-circumscribed mass is composed of multiple lymph nodes separated by thick fibrocollagenous bands. The individual lymph nodes show numerous reactive lymphoid follicles with reactive germinal centers, while the surrounding interfollicular areas demonstrate expansion by lymphocytes, plasma cells, and numerous eosinophils [1,3]. A few germinal centers contain central eosinophilic proteinaceous material, and focal areas show eosinophilic microabscess formation. No cytological atypia is identified. Findings confirmed Kimura disease, Fig.11.

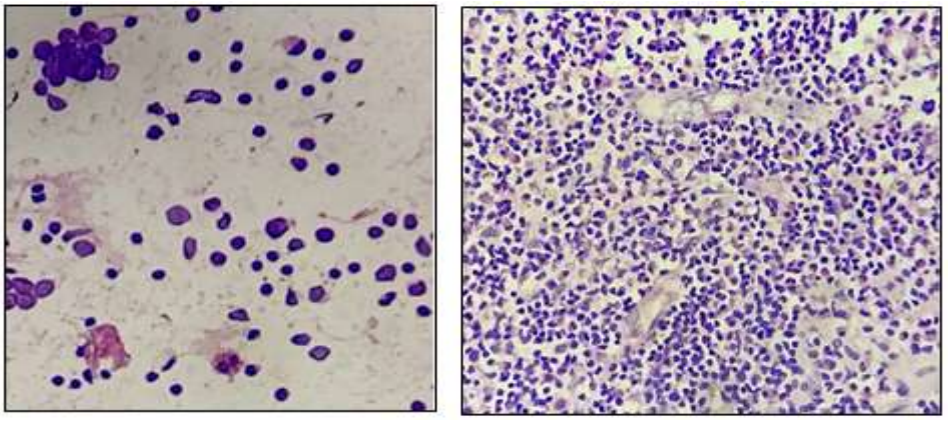

Fig.11. Histopathological images post-excisional biopsy.

Treatment and Outcome

The patient underwent surgical excision of the lesion with collagen graft placement over the inner buccal mucosa. Postoperative recovery was uneventful, and the patient is currently asymptomatic on follow-up. Other treatment modalities available for Kimura disease includes regional or systemic therapy. Though preferred first-line therapy for isolated lesions is surgical excision with less recurrence rates [4], but when surgical intervention is not possible, radiation therapy can be considered as a viable option. To treat more widespread or resistant cases, adjuvant therapy including systemic immunosuppressive medication, such as corticosteroids or cyclosporine may be utilised. Dupilumab, an emerging monoclonal antibody targeting interleukin pathways, have shown promising results.[1,5]

DISCUSSION

Kimura's disease is characterized by a chronic inflammatory process with prominent eosinophilic infiltration and lymphoid hyperplasia [1]. It most commonly involves the subcutaneous tissues of the head and neck, frequently associated with regional lymphadenopathy and systemic immunological markers such as eosinophilia and elevated IgE levels, Fig.12. Imaging plays a crucial role in initial assessment; however, findings are often nonspecific. On MRI, lesions typically appear isointense to muscle on T1-WI and hyperintense on T2-WI, with variable but often marked post-contrast enhancement reflecting underlying vascular proliferation and inflammatory activity.

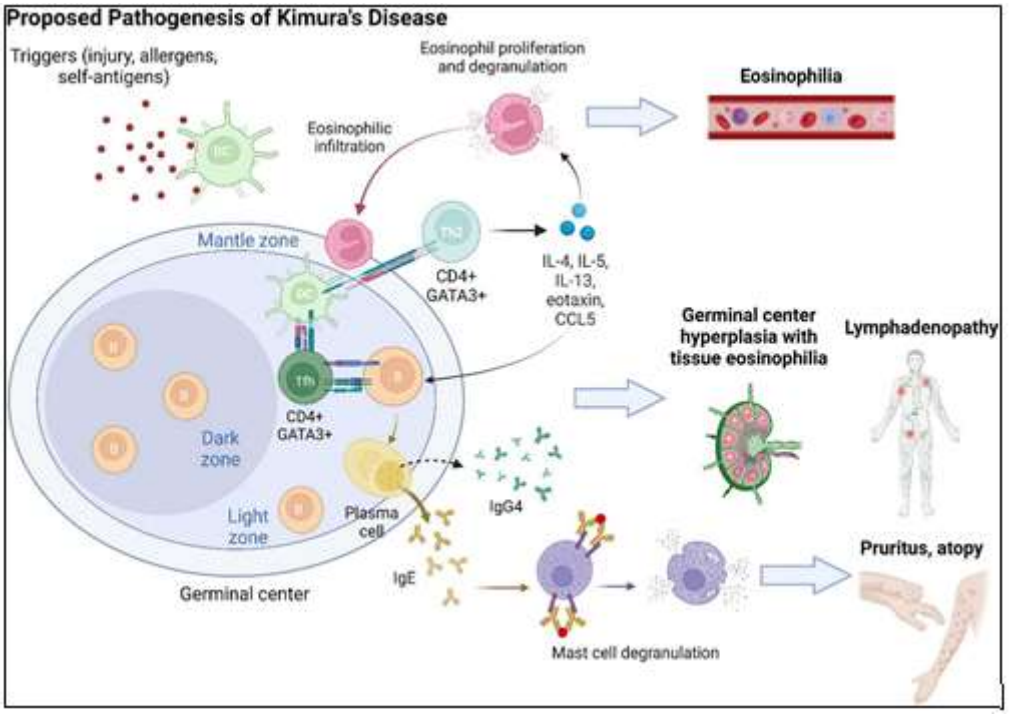

Fig.12. Image depicting pathogenesis of Kimura disease.[1]

A key diagnostic challenge arises when the lesion demonstrates channel-like areas on ultrasound, mimicking a slow-flow vascular malformation [8]. In such scenarios, careful evaluation of Doppler characteristics is essential. The absence of true flow within these apparent channels, along with lack of arterial feeders or early draining veins on angiographic sequences, serves as an important differentiating feature.

The presence of associated lymphadenopathy further supports an inflammatory or lymphoproliferative aetiology rather than a primary vascular anomaly. Additionally, detection of multifocal involvement, such as lacrimal gland enlargement [6] or sublingual lesions, may provide further diagnostic clues favouring Kimura disease.

Histopathological examination remains the gold standard for definitive diagnosis, demonstrating lymphoid follicular hyperplasia with dense eosinophilic infiltrate and characteristic microabscess formation. Recognition of these imaging and clinical patterns is essential to prevent misdiagnosis and avoid unnecessary vascular interventions.

Differential Diagnosis

Due to overlapping features, Kimura disease can be misdiagnosed with other clinical entities, however some striking features helpful in differentiating them are listed below.

Table 1. Differential Diagnosis

Differential Diagnosis	Clinical features	Key Imaging Features	Mimicking features	Diagnostic clue for DD	Differentiating points
Slow- flow venous malformation	Long standing, compressible swelling which increases with Valsalva manoeuvre	Serpiginous T2 hyperintense channels on MRI, phleboliths on CT, slow flow doppler waveform on colour doppler studies	Anechoic vascular channels on USG; T2 hyperintensity	True venous flow within the channels: may show phleboliths; feeder vessel/ draining veins can be seen on angiographic studies	Absence of flow within the channels argues against vascular malformation
Lymphoma	Constitutional B symptoms (fever, night sweats, unintentional weight loss); multiple enlarged lymph nodes	Homogenous nodal enlargement; lesion showing significant diffusion restriction	Lymphadenopathy; diffusion restriction within the lesion	Predominantly nodal disease with less subcutaneous infiltrative mass	Extranodal component argues against lymphoma
ALHE (Angio Lymphoid Hyperplasia with Eosinophilia)	Superficial nodules, more common in females	Superficial lesion with marked internal vascularity	Eosinophilia; vascular proliferation	Superficial lesion: minimal lymphadenopathy	Superficial lesion with deep extension and presence of multiple cervical and sublingual lymph nodes favors Kimura disease
Kikuchi – Fujimoto disease [10]	Young females, fever, painful nodes	Necrotic lymphadenitis	Cervical lymphadenopathy	Painful nodes; systemic symptoms; no dominant extranodal mass	Painful necrotic nodes + fever favors Kikuchi disease more over Kimura disease
Salivary gland tumour	Parotid swelling with ductal symptoms	Well-defined intraglandular mass	Located in cheek region	Lesion seen involving the parotid gland with distorted glandular architecture	Preserved parotid gland architecture with no intraglandular component favors Kimura disease
Differential Diagnosis	**Clinical features**	**Key Imaging Features**	**Mimicking features**	**Diagnostic clue for DD**	**Differentiating points**

Castleman disease	Young to middle-aged, systemic symptoms, usually associated with anaemia and elevated IL-6, can be unicentric or multicentric	Non- necrotic, usually solitary (hyaline vascular type) lymph nodal mass showing intense homogenous enhancement – commonly mediastinal, but can be cervical	Cervical lymphadenopathy	Discrete, well-circumscribed nodal mass + avid enhancement +/- systemic features favours Castleman disease	Avidly enhancing sharply marginated solitary lymph nodal mass – more likely favours unicentric Castleman disease

CONCLUSION

Due to common mimickers, Kimura's disease should be considered in the differential diagnosis of vascular-appearing soft tissue lesions in the head and neck region, especially in young males with associated lymphadenopathy. Every anechoic channel is not a vascular channel unless Doppler and spectral waveform confirms the flow. Overcalling vascular malformation can lead to unnecessary DSA and inappropriate intervention. Thus, early recognition and histopathological confirmation is essential to avoid misdiagnosis and ensure appropriate management.

Abbreviations

MRI Magnetic Resonance Imaging
DWI Diffusion Weighted Imaging
ADC Apparent Diffusion Co-efficient
USG Ultrasonography
T1WI T1-weighted images
T2WI T2-weighted images
DSA Digital Subtraction Angiography
ALHE Angio lymphoid hyperplasia with Eosinophilia
HPE Histopathological Examination

Ethics approval and consent to participate

Informed Consent Obtained.

Consent for publication

Informed Consent Obtained.

Competing interests

None

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