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Dasatinib-Induced Transient Ischaemic Attack and Bilateral Pleural Effusions in a Patient with Chronic Myeloid Leukaemia: A Case Report

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Abstract

Dasatinib is a second-generation tyrosine kinase inhibitor (TKI) used in the management of chronic myeloid leukaemia (CML). Although generally well tolerated, it is associated with adverse events including pleural effusions and, more rarely, neurovascular complications such as transient ischaemic attacks (TIAs). We report the case of a 69-year-old male with chronic-phase CML who developed bilateral pleural effusions and transient slurred speech while receiving dasatinib. The patient's symptoms resolved following cessation of dasatinib and initiation of corticosteroid and diuretic therapy. This case highlights the importance of vigilance for both pulmonary and neurological adverse effects of TKIs, and the value of early intervention in preventing morbidity.

Keywords: Tyrosine kinase inhibitor (TKI); Transient ischaemic attacks (TIAs); Chronic-phase CML

Background

Chronic myeloid leukaemia is a myeloproliferative neoplasm characterised by the presence of the Philadelphia chromosome, t(9;22)(q34;q11), which results in the BCR-ABL1 fusion gene encoding a constitutively active tyrosine kinase. Dasatinib is a potent second-generation TKI effective across all phases of CML and is often employed in cases of imatinib intolerance or resistance. While pleural effusions are a well-known dose-dependent complication of dasatinib, the occurrence of neurovascular events such as TIAs is far less common and may go unrecognised. The exact mechanism remains uncertain, though proposed pathways include endothelial dysfunction, platelet inhibition, or off-target kinase effects. Prompt identification of such adverse effects is essential for the safe and effective use of TKIs.

Case Presentation

A 69-year-old male was referred to the emergency department with acute-onset slurring of speech, without associated limb weakness, visual changes, or loss of consciousness. His past medical history included recently diagnosed chronic-phase CML, hypertension, hyperlipidaemia, gastro-oesophageal reflux disease (GORD), and prior transurethral resection of the prostate (TURP). He was a non-smoker and lived independently. The patient had been diagnosed with CML in January 2024, confirmed by peripheral blood and bone marrow analysis showing BCR-ABL1 positivity (84%) with the classic t(9;22)(q34;q11.2) translocation. He commenced dasatinib 100 mg daily on 4 January 2024 and initially responded well haematologically. Over the following months, he developed worsening reflux symptoms and exertional breathlessness. On the day of presentation in April 2025, he experienced transient slurring of speech, which resolved spontaneously within hours. There were no other neurological deficits. His daughter reported that he had been increasingly lethargic and less active in the preceding weeks.

Examination

On arrival, the patient was haemodynamically stable and afebrile. Neurological examination revealed intact cranial nerve function,

normal tone and power in all limbs (5/5), and no sensory deficits. Plantar responses were equivocal and reflexes were symmetrical. Cardiopulmonary and abdominal examinations were unremarkable. There was no peripheral oedema or signs of fluid overload.

Investigations

Magnetic resonance imaging (MRI) of the brain showed no evidence of acute infarction or haemorrhage. However, chronic supratentorial white matter hyperintensities consistent with small vessel ischaemia were present. There was no significant mass lesion or abnormal enhancement (Figure 1).

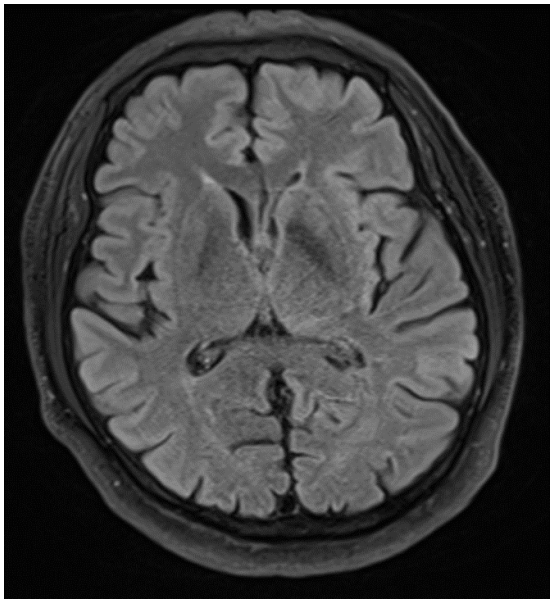

Figure 1: *MRI of brain showing no evidence of acute infarction or haemorrhage but chronic supratentorial white matter hyperintensities consistent with small vessel ischaemia.*

A chest X-ray performed on 29 January 2024 had shown left lower lobe atelectasis but no pleural effusion. By 26 February 2025, repeat chest imaging revealed bilateral pleural effusions, more marked on the right, with associated basal atelectasis. A computed tomography (CT) scan of the chest confirmed the presence of moderate bilateral effusions and reactive mediastinal lymphadenopathy. No pulmonary embolism was identified. Routine blood tests revealed a white cell count of 13.2 × 10^9/L, haemoglobin of 133 g/L, and platelet count of 178 × 10^9/L. Liver function tests were mildly elevated (ALT 61 U/L, AST 44 U/L), and lactate dehydrogenase (LDH) was 273 U/L. Renal function, electrolytes, and calcium were within normal limits. A repeat BCR-ABL1 qPCR test was pending. An echocardiogram was requested to assess for underlying cardiac pathology contributing to the pleural effusions (Figures 2-4).

Figure 2: *Chest X-ray from 29th of January 2024 showing the cardiomediastinal contours within normal limits. There is atelectasis at the left base (arrow).*

Figure 3: *Chest X-ray from 26th of February 2025 showing an enlarged cardiac silhouette and small bilateral pleural effusions (arrowheads) with atelectasis or scarring at the left lung base noted.*

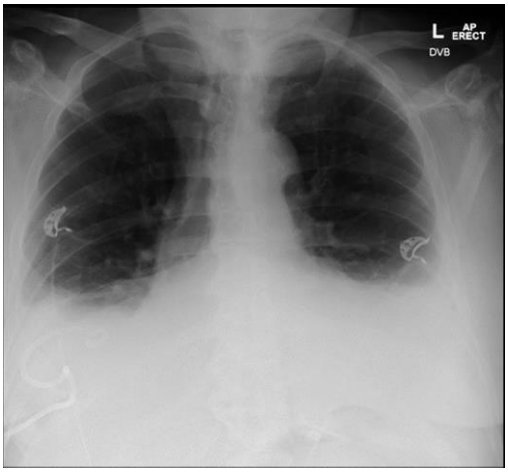

Figure 4: *Admission Chest X-ray on 16th of April 2025 showing bilateral pleural effusion at the level of the 6TH intercostal space (arrowheads).*

Management

In view of the pleural effusions and new-onset neurological symptoms, dasatinib was immediately ceased. The patient was commenced on a short course of oral prednisone to address the inflammatory component of the effusions, along with frusemide for symptomatic fluid offloading. Pantoprazole was continued for management of GORD. The patient was advised to rest, and close outpatient follow-up was arranged with haematology and respiratory teams. Neurology opinion supported a diagnosis of probable TIA in the context of small vessel disease and TKI exposure. Given the absence of infarct and lack of ongoing symptoms, antithrombotic therapy was not initiated.

Outcome and Follow-Up

Following cessation of dasatinib and the introduction of steroids and diuretics, the patient reported significant improvement in his symptoms. His speech returned to normal, and his breathlessness abated over the ensuing days. He remained haemodynamically stable and neurologically intact on review. Pleural effusions were managed conservatively without the need for drainage. Plans were made to re-evaluate therapy and consider a switch to an alternative TKI with a more favourable side effect profile. Serial imaging and cardiac investigations were arranged to monitor for recurrence or alternative pathology.

Discussion

Pleural effusions are a well-recognised adverse effect of dasatinib and occur in up to 30% of treated patients. The pathophysiology is thought to involve off-target inhibition of platelet-derived growth factor receptor-β and SRC family kinases, leading to altered vascular permeability and immune dysregulation. Management typically includes dose reduction, temporary cessation, or use of diuretics and corticosteroids. Rechallenge or switching to an alternative TKI may be considered based on response and tolerance. The occurrence of TIA-like symptoms in this patient raises important concerns regarding the cerebrovascular safety profile of dasatinib. While uncommon, reports of neurological adverse events have emerged, particularly in older patients with vascular risk factors. The mechanism may involve endothelial injury, platelet dysfunction, or dysregulated coagulation. MRI findings in this case were consistent with chronic small vessel ischaemia, but the temporal relationship to dasatinib exposure and rapid resolution of symptoms supports a diagnosis of TIA. Given the potential severity, such events warrant immediate evaluation and reassessment of therapy [1-5].

Conclusion

This case highlights a rare but significant dual complication of dasatinib therapy—bilateral pleural effusions and probable transient ischaemic attack—in a patient with chronic-phase CML. Clinicians must maintain a high index of suspicion for such adverse effects and act promptly with drug cessation and supportive treatment. Multidisciplinary evaluation is essential to balance treatment efficacy with patient safety, especially in elderly individuals with comorbidities.

Learning Points

- Dasatinib is associated with pleural effusions in up to 30% of patients, necessitating surveillance and symptomatic management.
- Neurological symptoms such as TIA are rare but should prompt urgent evaluation and reconsideration of TKI therapy.
- Management may include cessation of the offending agent, corticosteroids, diuretics, and neurologic assessment.
- In elderly patients or those with vascular risk factors, the threshold for investigating neurological symptoms should be low.
- Early recognition and multidisciplinary management can optimise outcomes while maintaining control of underlying CML.

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