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Transient Acquired Speech Dysfluency Temporally Associated with Dexmedetomidine- Assisted Sedation During Spinal Anesthesia: A Case Report

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Case Report

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Abstract

Background

Dexmedetomidine (DEX), a highly selective α_2-adrenoceptor agonist, is widely used for perioperative sedation because it provides cooperative sedation while maintaining spontaneous respiration and reducing the risk of respiratory depression. Although its pharmacological characteristics and safety profile have been extensively investigated, neurological adverse events involving speech production remain rarely reported. We describe a case of transient acquired speech dysfluency temporally associated with dexmedetomidine-assisted sedation during spinal anesthesia.

Case Presentation

A 63-year-old woman (American Society of Anesthesiologists physical status II) underwent elective arthroscopic surgery of the left knee under spinal anesthesia. Dexmedetomidine was administered for intraoperative sedation using a loading infusion followed by continuous maintenance infusion. Approximately 30 minutes after discontinuation of dexmedetomidine, the patient suddenly developed difficulty initiating speech characterized by repetition of initial sounds and syllables and sound prolongation. She remained fully conscious and oriented throughout the episode, with preserved comprehension, naming ability, semantic expression, and articulation. Neurological examination revealed no focal neurological deficits except for the speech abnormality, corresponding to a National Institutes of Health Stroke Scale score of 1. Brain magnetic resonance imaging, diffusion-weighted imaging, and intracranial magnetic resonance angiography demonstrated no evidence of acute cerebral infarction or major intracranial vascular abnormalities. No specific neurological treatment was administered. The speech disturbance resolved spontaneously approximately 97 minutes after symptom onset. The patient recovered completely without recurrence during hospitalization or the 7-day follow-up period.

Conclusions

This case describes transient acquired speech dysfluency occurring in close temporal association with dexmedetomidine-assisted sedation during spinal anesthesia. Although a causal relationship cannot be established from a single case, dexmedetomidine-related modulation of speech-associated neural networks may represent one possible explanation, particularly in patients with pre-existing basal ganglia abnormalities. Acute cerebrovascular events should always be excluded when new-onset speech abnormalities occur during the perioperative period.

Introduction

Speech fluency depends on the coordinated activity of distributed cortical and subcortical networks responsible for speech initiation, motor planning, sequencing, and temporal regulation. Speech dysfluency refers to abnormalities in the continuity, rhythm, or initiation of speech and commonly manifests as repetition of sounds or syllables, sound prolongation, and speech blocks. When these abnormalities develop after previously normal speech due to neurological injury, systemic disease, or medication exposure, they are generally classified as acquired speech dysfluency. Acquired stuttering is one of the most recognized forms and has been reported after stroke, traumatic brain injury, neurodegenerative disorders, epilepsy, and other neurological conditions.[1,2]

The neural mechanisms underlying speech fluency involve complex interactions among the cortex, basal ganglia, thalamus, and supplementary motor regions. Functional and structural abnormalities within the cortico-striato-thalamo-cortical network have been implicated in both developmental and acquired stuttering.[3] In particular, basal ganglia circuits play an essential role in speech initiation, motor timing, and selection of appropriate speech motor patterns. Dysfunction within these networks may result in impaired speech fluency even without widespread neurological deficits.

Dexmedetomidine (DEX) is a highly selective α_2-adrenoceptor agonist that produces sedation primarily through inhibition of noradrenergic activity in the locus coeruleus. It has become widely used in perioperative medicine because it provides cooperative sedation with minimal respiratory depression and allows patients to remain responsive to verbal stimulation.[4] Compared with conventional sedatives, DEX is associated with favorable respiratory and recovery profiles; however, adverse effects including bradycardia, hypotension, hypertension during loading administration, and excessive sedation have been reported.[4,5] Neurological complications involving speech function remain uncommon.

New-onset speech abnormalities during the perioperative period require immediate evaluation because they may represent manifestations of acute cerebrovascular disease. Transient ischemic attack (TIA) and acute ischemic stroke should be considered first, particularly in patients with vascular risk factors. However, transient neurological symptoms may also result from reversible dysfunction of neural networks, medication effects, or interactions between pharmacological agents and pre-existing structural abnormalities.[6]

In this report, we describe a patient who developed transient acquired speech dysfluency shortly after dexmedetomidine-assisted sedation during spinal anesthesia. The diagnosis, differential considerations, possible mechanisms, and limitations of causal interpretation are discussed according to the principles of CARE guidelines.

Case Report

Patient Information and Clinical Findings

A 63-year-old woman (height, 158 cm; weight, 60 kg; body mass index, 24.03 kg/m²) was admitted to our hospital because of progressive pain and restricted mobility of the left knee. She had experienced

intermittent left knee pain for approximately 10 years, with significant aggravation during the week preceding admission. Arthroscopic debridement, radiofrequency ablation, and meniscal repair of the left knee were scheduled.

Her medical history included hypertension for more than 10 years, which was treated with enalapril maleate and sustained-release nifedipine; type 2 diabetes mellitus for more than 12 years, which was managed with metformin and acarbose; and hypothyroidism for 5 years, which was treated with levothyroxine sodium. She denied any previous history of stroke, transient ischemic attack, epilepsy, psychiatric disorders, migraine, smoking, or alcohol consumption. She had never experienced stuttering, speech dysfluency, dysarthria, aphasia, or other language disorders. No family history of neurological disease or speech impairment was reported.

On admission, the patient was alert and fully oriented. Her body temperature was 36.4°C, heart rate was 74 beats/min, respiratory rate was 16 breaths/min, and blood pressure was 149/75 mmHg. General physical examination was unremarkable except for tenderness and restricted movement of the left knee.

Baseline neurological examination showed normal cranial nerve function, intact muscle strength and sensation in all extremities, normal coordination, and no pathological reflexes. Speech evaluation demonstrated normal spontaneous speech, comprehension, naming ability, semantic expression, and articulation.

Preoperative Assessment

Routine laboratory investigations revealed mild anemia, with a hemoglobin concentration of 110 g/L. Liver function, renal function, coagulation parameters, serum electrolytes, and arterial oxygen saturation were within normal limits.

The fasting plasma glucose level was 4.88 mmol/L, the 2-hour postprandial glucose level was 7.08 mmol/L, and glycated hemoglobin was 6.46%, indicating relatively stable glycemic control.

Transthoracic echocardiography demonstrated impaired left ventricular diastolic function without significant structural cardiac abnormalities. Duplex ultrasonography of both lower extremities revealed atherosclerotic plaque formation without evidence of significant arterial stenosis or deep venous thrombosis.

Preoperative cranial computed tomography (CT) demonstrated multiple chronic lacunar infarctions involving the bilateral basal ganglia. No acute intracranial hemorrhage, recent cerebral infarction, or mass lesion was identified. Although chronic basal ganglia lesions were present, the patient had no history of neurological symptoms or previous cerebrovascular events.

Anesthetic Management

After routine monitoring was established in the operating room, including electrocardiography, noninvasive blood pressure measurement, and pulse oximetry, oxygen was administered through a face mask at 4 L/min.

With the patient positioned in the right lateral decubitus position, spinal anesthesia was performed at the L4–L5 interspace using a midline approach. After confirmation of free cerebrospinal fluid flow, 3 mL of 0.25% bupivacaine hydrochloride was administered intrathecally. A sensory block reaching the T10 dermatome was achieved, providing satisfactory anesthesia for the surgical procedure.

Dexmedetomidine sedation was initiated after confirmation of adequate spinal anesthesia. A loading dose of 0.5 μg/kg was administered by continuous infusion at 3 $\mu g \cdot kg^{-1} \cdot h^{-1}$ over 10 minutes, followed by a maintenance infusion of 0.3 $\mu g \cdot kg^{-1} \cdot h^{-1}$. The infusion was discontinued approximately 10 minutes before completion of surgery, and the total administered dose was approximately 0.06 mg.

Sedation depth was monitored using the Richmond Agitation–Sedation Scale (RASS). The lowest intraoperative RASS score was − 2, and the patient remained responsive to verbal stimulation throughout the procedure. Cefuroxime sodium was administered for perioperative antimicrobial prophylaxis. No opioids, benzodiazepines, propofol, or inhalational anesthetics were administered.

The surgical procedure was completed uneventfully. Hemodynamic and respiratory parameters remained stable throughout the operation. Blood pressure ranged from 155 to 178/72–100 mmHg, heart rate ranged from 60 to 82 beats/min, and peripheral oxygen saturation remained ≥ 99%. No episodes of hypotension, bradycardia, hypoxemia, respiratory depression, or requirement for vasoactive medication occurred.

Postoperative Neurological Event

At the end of surgery, the patient was awake, cooperative, and able to communicate normally, with a RASS score of − 1. She was transferred to the ward after routine postoperative observation.

Approximately 30 minutes after discontinuation of dexmedetomidine, the patient suddenly developed difficulty initiating speech. Her speech was characterized by repetition of initial sounds and syllables accompanied by occasional sound prolongation. Despite these abnormalities, she remained fully conscious and oriented, recognized family members and medical staff, and responded appropriately to questions.

Language comprehension, naming ability, semantic expression, and articulation remained intact throughout the episode. No slurred speech or abnormal pronunciation was observed. The clinical presentation was inconsistent with aphasia because language processing abilities were preserved. Dysarthria was considered unlikely because articulation and speech motor function remained normal. Furthermore, the absence of inconsistent articulatory errors or impaired speech motor programming made apraxia of speech less likely.

At symptom onset, the patient's blood pressure was 156/78 mmHg, heart rate was 80 beats/min, respiratory rate was 16 breaths/min, body temperature was 36.5°C, and oxygen saturation was 100% while breathing room air. The RASS score was 0, indicating full alertness.

Neurological Evaluation and Diagnostic Assessment

Because acute cerebrovascular disease could not be excluded, an urgent neurological consultation was obtained.

Neurological examination demonstrated equal and reactive pupils, symmetrical facial movement, normal upper-extremity strength and sensation, intact coordination, and absence of pathological reflexes. Assessment of lower-extremity motor and sensory function was limited because residual spinal anesthesia had not completely resolved.

The National Institutes of Health Stroke Scale (NIHSS) score was 1, with the single abnormal point attributed to the speech abnormality. No facial weakness, visual field deficit, upper-extremity weakness, sensory disturbance, neglect, or cerebellar signs were identified.

Brain magnetic resonance imaging (MRI), diffusion-weighted imaging (DWI), and intracranial magnetic resonance angiography (MRA) were performed while the patient remained symptomatic. DWI showed no evidence of acute diffusion restriction or acute cerebral infarction. MRA demonstrated no intracranial arterial occlusion, arterial dissection, or severe intracranial arterial stenosis. Chronic bilateral basal ganglia lacunar infarctions were again identified, consistent with the preoperative CT findings.

Because imaging findings did not indicate acute ischemic stroke and the patient's neurological status remained stable, thrombolytic therapy or other targeted neurological intervention was not initiated. Close clinical observation was continued.

Clinical Outcome and Follow-up

The patient's speech gradually returned to normal without pharmacological treatment. Approximately 97 minutes after symptom onset, difficulty initiating speech, repetition of initial sounds and syllables, and sound prolongation had completely resolved.

Repeat neurological examination demonstrated normal consciousness, language comprehension, naming ability, semantic expression, and articulation. No focal neurological deficits were identified.

The postoperative course was otherwise uneventful. The patient was discharged on postoperative day 3 without neurological sequelae.

Telephone follow-up performed 7 days after surgery confirmed complete recovery. The patient reported normal speech function without recurrence of speech dysfluency, limb weakness, sensory disturbance, headache, or dizziness.

Discussion

Dexmedetomidine (DEX) is a highly selective α_2-adrenoceptor agonist that has become widely used for procedural and perioperative sedation because it produces cooperative sedation while preserving spontaneous respiration and allowing patients to remain responsive to verbal stimulation. Compared with conventional sedatives, DEX is associated with less respiratory depression and favorable postoperative recovery characteristics. The most commonly reported adverse effects include bradycardia, hypotension, transient hypertension during loading administration, and excessive sedation.[4,5] Neurological adverse events affecting speech function are uncommon. In this case, the patient developed transient acquired speech dysfluency shortly after discontinuation of dexmedetomidine-assisted sedation during spinal anesthesia. Although the temporal relationship suggests a possible association, the findings do not establish a direct causal relationship between DEX exposure and the neurological event.

Speech dysfluency is characterized by abnormalities in speech continuity, rhythm, or initiation, including repetition of sounds or syllables, sound prolongation, and speech blocks. Unlike aphasia, which results from impaired language processing, or dysarthria, which reflects impaired speech articulation caused by neuromuscular dysfunction, speech dysfluency primarily involves the initiation and temporal organization of speech output. In the present patient, comprehension, naming, semantic expression, and articulation remained intact throughout the episode. The preservation of these functions argued against aphasia and dysarthria. In addition, the absence of inconsistent articulatory errors or impaired speech motor planning made apraxia of speech less likely. Therefore, the observed phenomenon was considered most consistent with transient acquired speech dysfluency.

Acquired speech dysfluency has been described in patients with neurological disorders involving cortical and subcortical speech networks. Previous studies have reported that stroke affecting the basal ganglia, thalamus, supplementary motor area, and their connecting pathways may result in acquired stuttering or other abnormalities of speech fluency.[1,2] The cortico-striato-thalamo-cortical network plays an important role in speech initiation, motor timing, and regulation of fluent speech production. Functional and neuroanatomical abnormalities within this network have also been associated with developmental and acquired forms of stuttering.[3]

The most important diagnostic consideration in this patient was acute cerebrovascular disease. Sudden-onset speech abnormalities after anesthesia or sedation require immediate neurological evaluation because isolated speech symptoms may represent early manifestations of ischemic stroke or transient ischemic attack (TIA).[6] This patient had several vascular risk factors, including hypertension, diabetes mellitus, atherosclerotic changes, and chronic bilateral basal ganglia lacunar infarctions.

However, multiple findings argued against acute ischemic stroke as the primary explanation. Brain MRI and diffusion-weighted imaging performed during the symptomatic period showed no evidence of acute cerebral infarction. Intracranial magnetic resonance angiography demonstrated no major arterial occlusion, arterial dissection, or severe stenotic lesions. Furthermore, the patient exhibited no persistent

neurological abnormalities other than transient speech dysfluency, and complete spontaneous recovery occurred within approximately 97 minutes without thrombolytic therapy or other cerebrovascular intervention.

Nevertheless, a diffusion-negative transient ischemic attack cannot be completely excluded. According to the current definition, TIA refers to transient neurological dysfunction caused by focal cerebral ischemia without evidence of acute infarction on imaging.[6] Therefore, normal diffusion-weighted imaging does not entirely eliminate the possibility of transient ischemia, particularly in patients with brief symptoms or small vascular territory involvement. In this case, the possibility of TIA was considered; however, the isolated nature of the symptom, the absence of additional focal neurological findings, the close temporal relationship with DEX administration, and the complete spontaneous resolution without specific treatment made a transient functional disturbance of speech-related neural networks a plausible alternative explanation.

The presence of chronic bilateral basal ganglia lacunar infarctions may have contributed to individual susceptibility. The basal ganglia are involved not only in motor control but also in speech initiation and fluency regulation through extensive connections with the supplementary motor area, premotor cortex, thalamus, and frontal speech-related regions.[3] Although the patient had no previous clinically apparent speech impairment, chronic structural abnormalities within these circuits may have reduced functional reserve and increased vulnerability to transient alterations in neural activity.

The potential contribution of dexmedetomidine requires cautious interpretation. DEX induces sedation mainly through activation of α_2-adrenoceptors located in the locus coeruleus, resulting in reduced central noradrenergic activity and altered communication between cortical and subcortical networks.[4] Functional neuroimaging and electrophysiological studies have demonstrated that DEX produces reversible changes in cortical and subcortical activity patterns while maintaining a degree of environmental responsiveness.[7] These reversible changes in neural network function may theoretically influence speech-related circuits in susceptible individuals. However, direct evidence linking DEX exposure to transient speech dysfluency is currently unavailable, and the proposed mechanism remains hypothetical.

To further evaluate the possibility of a drug-related adverse event, the Naranjo Adverse Drug Reaction Probability Scale was applied.[8] The patient obtained a total score of 3 points, corresponding to a possible adverse drug reaction. This assessment was mainly supported by the clear temporal relationship between DEX exposure and symptom onset, together with objective documentation of the neurological event. However, the absence of previous reports of an identical reaction, the lack of drug rechallenge, and the presence of alternative explanations limited the strength of causal inference. Therefore, the current case should be interpreted as a possible association rather than confirmed DEX-induced neurotoxicity.

Several alternative perioperative explanations were considered. Residual effects of spinal anesthesia were unlikely because sensory and motor blockade does not selectively affect speech initiation or

speech fluency. Perioperative hypoxemia, hypercapnia, hypoglycemia, electrolyte imbalance, and hemodynamic instability were excluded by continuous monitoring and laboratory evaluation. The patient did not receive opioids, benzodiazepines, propofol, or inhalational anesthetics, reducing the possibility of interactions with other centrally acting medications. A functional neurological disorder was also considered because abrupt onset and spontaneous recovery can occur in functional symptoms. However, the patient had no psychiatric history, no inconsistent neurological findings, and a reproducible speech pattern characterized by repetition and sound prolongation, making this explanation less likely, although it cannot be completely excluded.

This case highlights several clinically relevant considerations. First, transient acquired speech dysfluency may occur during the perioperative period even when conventional neuroimaging does not reveal acute structural abnormalities. Second, new-onset speech abnormalities after sedation should always prompt urgent neurological assessment because potentially reversible or treatable cerebrovascular events must be excluded. Third, patients with pre-existing basal ganglia lesions may represent a subgroup with increased susceptibility to transient disturbances of speech-related neural networks after pharmacological modulation.

Several limitations should be acknowledged. This report describes only a single patient, and no definitive causal relationship between DEX administration and speech dysfluency can be established. Formal speech-language pathology assessment and acoustic analysis were not performed because the symptoms resolved rapidly. Advanced functional neuroimaging or electrophysiological studies were also unavailable, limiting further exploration of the underlying mechanism. Although acute ischemic stroke was unlikely based on imaging findings and clinical evolution, transient ischemia cannot be completely ruled out.

In conclusion, this case describes transient acquired speech dysfluency temporally associated with dexmedetomidine-assisted sedation during spinal anesthesia. The event occurred in a patient with chronic bilateral basal ganglia lacunar infarctions, resolved spontaneously, and was not accompanied by evidence of acute cerebral ischemia. Dexmedetomidine-related modulation of speech-associated neural networks may represent one possible contributing factor, but further clinical observations are required to determine whether this represents a reproducible neurological adverse effect.

Timeline

Admission

A 63-year-old woman was admitted because of progressive left knee pain and restricted mobility caused by osteoarthritis. Elective arthroscopic surgery was planned.

Preoperative assessment

Routine laboratory investigations, cardiopulmonary evaluation, and cranial computed tomography were completed. CT revealed chronic bilateral basal ganglia lacunar infarctions without previous neurological symptoms.

Intraoperative period

Spinal anesthesia was successfully performed at the L4–L5 interspace. Dexmedetomidine-assisted sedation was administered after adequate sensory blockade was achieved. The surgery proceeded uneventfully with stable hemodynamic and respiratory parameters.

End of surgery

Dexmedetomidine infusion was discontinued approximately 10 minutes before completion of surgery. The patient was awake, cooperative, and communicating normally.

Approximately 30 minutes after DEX discontinuation

The patient suddenly developed difficulty initiating speech characterized by repetition of initial sounds and syllables and sound prolongation. Consciousness, comprehension, naming, and articulation remained intact.

Immediate neurological evaluation

Urgent neurological consultation was obtained. The NIHSS score was 1, attributable only to the speech abnormality.

Diagnostic imaging

Brain MRI, diffusion-weighted imaging, and intracranial MRA demonstrated no evidence of acute cerebral infarction, major arterial occlusion, or other acute vascular abnormalities.

Approximately 97 minutes after symptom onset

Speech fluency returned completely without specific neurological treatment.

Postoperative day 3

The patient was discharged without neurological sequelae.

Seven-day follow-up

Telephone follow-up confirmed complete recovery without recurrence of speech dysfluency or other neurological symptoms.

Timeline

Time	Clinical Event
Admission	A 63-year-old woman was admitted because of progressive left knee pain and restricted mobility caused by osteoarthritis. Elective arthroscopic surgery was planned.
Preoperative assessment	Routine laboratory investigations, cardiopulmonary evaluation, and cranial computed tomography were completed. CT revealed chronic bilateral basal ganglia lacunar infarctions without previous neurological symptoms.
Intraoperative period	Spinal anesthesia was successfully performed at the L4–L5 interspace. Dexmedetomidine-assisted sedation was administered after adequate sensory blockade was achieved. The surgery proceeded uneventfully with stable hemodynamic and respiratory parameters.
End of surgery	Dexmedetomidine infusion was discontinued approximately 10 minutes before completion of surgery. The patient was awake, cooperative, and communicating normally.
Approximately 30 minutes after DEX discontinuation	The patient suddenly developed difficulty initiating speech characterized by repetition of initial sounds and syllables and sound prolongation. Consciousness, comprehension, naming, and articulation remained intact.
Immediate neurological evaluation	Urgent neurological consultation was obtained. The NIHSS score was 1, attributable only to the speech abnormality.
Diagnostic imaging	Brain MRI, diffusion-weighted imaging, and intracranial MRA demonstrated no evidence of acute cerebral infarction, major arterial occlusion, or other acute vascular abnormalities.
Approximately 97 minutes after symptom onset	Speech fluency returned completely without specific neurological treatment.
Postoperative day 3	The patient was discharged without neurological sequelae.
Seven-day follow-up	Telephone follow-up confirmed complete recovery without recurrence of speech dysfluency or other neurological symptoms.
Patient Perspective	

The patient reported that the sudden difficulty in speaking was frightening because she remained fully conscious and understood what she wanted to express but was temporarily unable to initiate speech smoothly. She was particularly concerned about the possibility of stroke and felt anxious while undergoing neurological evaluation.

After brain imaging excluded major acute cerebrovascular abnormalities and her speech gradually returned to normal without treatment, she felt reassured. At follow-up, she reported complete recovery and expressed satisfaction with the clinical management and outcome.

Conclusion

This case describes transient acquired speech dysfluency temporally associated with dexmedetomidine-assisted sedation during spinal anesthesia. The patient developed isolated impairment of speech fluency without aphasia, dysarthria, focal neurological deficits, or radiological evidence of acute cerebral ischemia.

Although a direct causal relationship between dexmedetomidine and speech dysfluency cannot be established, the temporal relationship, clinical course, and exclusion of other perioperative triggers suggest that dexmedetomidine-related modulation of speech-associated neural networks may have contributed to the event in a patient with pre-existing basal ganglia abnormalities.

Clinicians should remain vigilant when new-onset speech abnormalities occur after anesthesia or sedation and should promptly exclude acute cerebrovascular events before considering medication-related or functional mechanisms.

Declarations

Ethics Statement

Ethical approval was not required according to institutional regulations for single case reports without experimental intervention.

This manuscript was prepared according to the CARE guidelines for case reports.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical information.

The patient agreed to publication of anonymized clinical data.

Author Contributions (CRediT)

Wudong Zhuang: Conceptualization, Investigation, Data curation, Writing – original draft.

Ruizhao Lyu: Conceptualization, Supervision, Validation, Writing – review and editing.

Rui Liu: Clinical management, Data curation.

Yang Bai: Clinical data acquisition and perioperative management.

Xiaoyuan Li: Literature review and manuscript preparation.

Yukun Xu: Clinical evaluation and follow-up.

Haoyang Geng: Investigation, Manuscript revision, Supervision.

All authors read and approved the final manuscript.

Conflict of Interest

The authors declare that they have no competing interests.

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Clinical Trial Number

Clinical trial number: not applicable.

Data Availability Statement

The clinical data supporting the findings of this case report are available from the corresponding author upon reasonable request. Due to patient privacy considerations, the data are not publicly available.

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