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CASE REPORT

Bell's palsy complicated by a hypertensive crisis: a case report and review of diagnostic and therapeutic challenges

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ABSTRACT

Background: Bell's palsy is a neurological condition characterized by acute unilateral facial paralysis. While it is often idiopathic, it can sometimes be associated with other underlying conditions, complicating diagnosis and management. **Objectives:** This case report aims to present a unique case of Bell's palsy complicated by a hypertensive crisis, emphasizing the importance of differential diagnosis and careful pharmacotherapy management. **Case Presentation:** A 63-year-old male presented to Ngletih Community Health Center with a four-day history of right-sided facial paralysis and slurred speech, following exposure to a fan while sleeping. Physical examination revealed a hypertensive crisis with a blood pressure of 185/144 mmHg. Neurological assessment showed no signs of stroke, such as limb weakness or other lateralizing signs. **Conclusion:** This case underscores the importance of a comprehensive diagnostic approach and an individualized treatment plan for patients with Bell's palsy and concurrent hypertensive crisis. Awareness of such complications can aid clinicians in optimizing patient outcomes while minimizing risks.

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Highlights

1. Unique Clinical Challenge: This case study presents a rare and complex scenario of Bell's palsy complicated by a hypertensive crisis, emphasizing the need for a careful and precise differential diagnosis to distinguish it from cerebrovascular accidents (CVA).
2. Pharmacotherapy Management: This case underlines the critical importance of managing

hypertension in patients with Bell's palsy, considering potential contraindications of corticosteroids, typically used in Bell’s palsy treatment, highlighting the intricacies of individualized treatment plans.

3. Clinical Practice Implications: The findings stress the necessity for comprehensive diagnostic approaches and vigilant monitoring in primary health care settings to optimize patient outcomes and minimize the risks associated with concurrent severe medical conditions such as hypertensive crises.

BACKGROUND

Bell's palsy, a condition characterized by the sudden onset of unilateral facial paralysis, is the most common acute mononeuropathy and the leading cause of facial paralysis worldwide. It results from inflammation or viral infection of the facial nerve (cranial nerve VII), leading to muscle weakness or paralysis on one side of the face (Zhang et al., 2020). The incidence of Bell's palsy is estimated to be approximately 15-30 cases per 100,000 people annually (Baugh et al., 2013).

Globally, Bell's palsy affects individuals of all ages, with a slightly higher prevalence in those aged 15-45 years (Schwartz et al., 2014), although some studies noted a higher incidence in the elderly (Dewi et al., 2020). The condition does not exhibit significant gender predilection, although some studies suggest a slight increase in incidence among women during pregnancy (Schwartz et al., 2014). In Indonesia, the prevalence of Bell's palsy mirrors global trends, but comprehensive national data are limited. Anecdotal evidence from healthcare facilities suggests that it is a relatively common neurological disorder, warranting further epidemiological studies.

Several risk factors have been identified for Bell's palsy, including viral infections (e.g., herpes simplex virus), genetic predisposition, diabetes mellitus, hypertension (Jeong et al., 2021; Riga et al., 2012), immunocompromised states (Dewi et al., 2020), epilepsy, obesity, and vaccination (Alanazi et al., 2022). Pregnant women, particularly during the third trimester or the first week postpartum, are also at increased risk (Baugh et al., 2013). A significant proportion of cases have been associated with underlying metabolic disorders, such as diabetes, obesity, and hypertension, which can influence the clinical presentation and prognosis (Mueanchoo et al., 2024; Savadi-Oskouei et al., 2008).

This case report of a 63-year-old male presenting with Bell's palsy complicated by a hypertensive crisis emphasizes the importance of differential diagnosis in distinguishing Bell's palsy from cerebrovascular accidents (stroke). The presence of severe hypertension poses unique challenges in management, particularly concerning the use of corticosteroids, which are commonly prescribed for Bell's palsy but may exacerbate hypertension (Madhok et al., 2016).

OBJECTIVE

This case report aims to highlight the diagnostic and therapeutic challenges associated with Bell's palsy in the context of a hypertensive crisis, providing valuable insights into the careful pharmacological management of such patients. The significance of this report lies in its potential to guide clinicians in optimizing treatment strategies, improving patient outcomes, and raising awareness of the interplay between Bell's palsy and hypertensive conditions.

CASE

A 63-year-old male presented to the Ngletih Community Health Center with a chief complaint of sudden right-sided facial paralysis and slurred speech for the past four days. The patient reported that the symptoms began after exposure to a fan while sleeping. He denied any recent trauma, fever, or upper respiratory tract infection.

The patient had a history of hypertension, managed with antihypertensive medication for the past five years. On examination, his blood pressure was significantly elevated at 185/144 mmHg. There was no history of diabetes, recent infections, or similar episodes in the past.

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Physical examination revealed pronounced right-sided facial weakness. The patient was unable to raise his right eyebrow, had incomplete closure of the right eye, and exhibited drooping of the right corner of his mouth, with loss of the nasolabial fold on the affected side. The forehead muscles on the right side showed a lack of movement, indicative of peripheral facial nerve palsy. No other neurological deficits were observed.

A comprehensive neurological examination was performed, including:

1. Cranial Nerve Examination:
    a. Cranial Nerve I (Olfactory): Normal smell perception.
    b. Cranial Nerve II (Optic): Visual acuity and fields were normal.
    c. Cranial Nerves III, IV, VI (Oculomotor, Trochlear, Abducens): Normal eye movements, no ptosis, pupils equal and reactive to light.
    d. Cranial Nerve V (Trigeminal): Normal facial sensation and corneal reflex.
    e. Cranial Nerve VII (Facial): Inability to raise the right eyebrow, incomplete closure of the right eye, drooping of the right mouth corner, loss of the nasolabial fold on the right side.
    f. Cranial Nerve VIII (Vestibulocochlear): Normal hearing and balance.
    g. Cranial Nerves IX, X (Glossopharyngeal, Vagus): Normal gag reflex, no dysphagia.
    h. Cranial Nerve XI (Accessory): Normal shoulder shrug and head rotation against resistance.
    i. Cranial Nerve XII (Hypoglossal): Midline tongue position, no atrophy or fasciculations.

The results of the neurologic examination of cranial nerve VII are shown in Tables 1 and 2.

Table 1. Examination of the facial nerve (N. VII) at rest

Rest	Right side	Left side
Forehead wrinkles	**(-)**	(+)
Angle of the eye	**Drooped**	Normal
Nasolabial creases	**Shallow**	Normal

Table 2. Examination of the facial nerve (N. VII) during movement

Movement	Right side	Left side
Frowning	(-)	(+)
Closing eyes	(-)	(+)
Grimace (showing teeth)	(-)	(+)
Whistle	(-)	(-)
Tasting on the front 2/3 of the tongue	Not done	Not done
Hyperacusis	(-)	(-)
Tear secretion	(+)	(-)

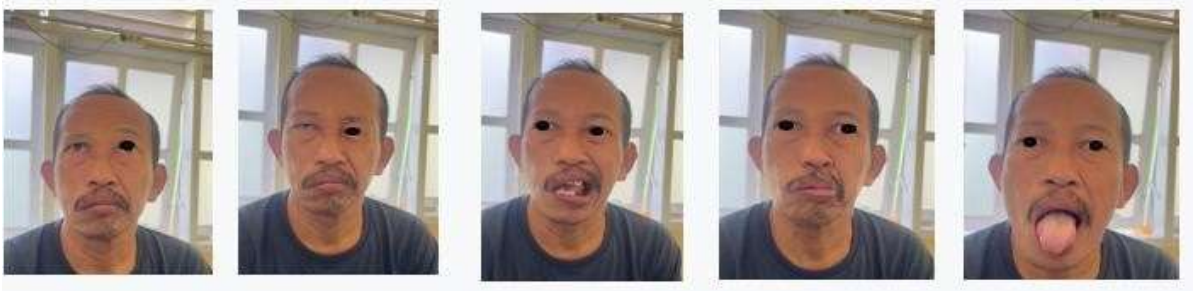

Figure 1. Patient's face during neurological examination of N. VII

2. Meningeal Signs: Negative for Kernig's and Brudzinski's signs, indicating no meningeal irritation.
3. Motor Examination:
    a. Muscle strength was normal in all extremities (5/5).
    b. No atrophy or fasciculations observed.
4. Sensory Examination:

    Normal sensation to light touch, pinprick, vibration, and proprioception in all extremities.
5. Reflexes:
    a. Physiological reflexes (biceps, triceps, brachioradialis, patellar, Achilles) were normal and symmetrical.
    b. Pathological reflexes (Babinski, Hoffmann) were absent, indicating no upper motor neuron lesion.

Basic laboratory tests, including a complete blood count, electrolyte panel, and fasting blood glucose, were all within normal limits as provided below.

Table 3. Results of laboratory tests

Laboratory test	Parameters	Results	Normal range
Complete Blood Count (CBC)	Hemoglobin (Hb)	15.2 g/dL	13.5-17.5 g/dL
	Hematocrit (Hct)	45%	41-50%
	Leucocytes (WBC)	5,800/uL	4,500-11,000/uL
	Thrombocytes (Platelet)	220,000/uL	150,000-450,000/uL
Electrolytes	Natrium (Na)	140 mmol/L	135-145 mmol/L
	Kalium (K)	4.2 mmol/L	3.5-5.1 mmol/L
	Chloride (Cl)	102 mmol/L	98-106 mmol/L
Metabolites	Fasting Blood Glucose	92 mg/dL	70-99 mg/dL

Given the primary care setting, advanced imaging such as a CT scan was not available.

The patient was diagnosed with Bell's palsy (G51), with a hypertensive crisis (I10) noted as a significant comorbidity. Due to the hypertensive crisis, careful consideration was given to the pharmacological management of the patient's condition. He was prescribed with Amlodipine 10 mg once daily to manage hypertension; Aspilet (Aspirin) 80 mg three times daily as an antiplatelet; vitamin B-Complex once daily; and artificial tears twice daily to protect the affected eye from dryness.

Non-pharmacological management included advice on controlling blood pressure through dietary and lifestyle modifications, such as adopting a low-sodium diet and avoiding high-fat foods. The patient was instructed to follow up if symptoms persisted or worsened and to return for a reassessment of blood pressure and facial nerve function after one week.

DISCUSSION

Bell's palsy, an acute, idiopathic, unilateral facial nerve paralysis, is a condition that necessitates prompt and effective management to maximize recovery and minimize complications (Baugh et al., 2013). The etiology may involve viral, inflammatory, autoimmune, and vascular factors, resulting in swelling or hemorrhage of the facial nerve (De Almeida et al., 2014). Management strategies for Bell's palsy primarily focus on pharmacological treatments, physical therapies (De Almeida et al., 2014), and supportive care, with the aim of improving facial function and facilitate recovery (Baugh et al., 2013) by minimizing the risk of incomplete recovery and reducing the likelihood of morbid sequelae (e.g., facial weakness, synkinesis, autonomic dysfunction, and contracture of the facial tissues) (De Almeida et al., 2014). Bell's palsy is assumed to result from inflammation of the facial nerve, making steroids a key therapeutic option, with systemic steroid treatment (SST) as the gold standard (Demir et al., 2020).

A study found that patients with Bell's palsy had significantly higher blood pressure, fasting blood glucose, lipid profile (except HDL-c), body mass index (BMI), and waist circumference compared to

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those without Bell's palsy (Mueanchoo et al., 2024b). This highlights the association between Bell's palsy and metabolic syndrome, including obesity, hypertension, and glucose and lipid metabolism impairments. The presence of comorbid conditions, such as hypertensive crisis in this case, complicates the management approach (Savadi-Oskouei et al., 2008). Metabolic syndrome, including hypertension, is associated with a lower recovery rate in Bell's palsy patients (Jung et al., 2018). In hypertensive patients, facial nerve palsy may arise from vascular lesions that thicken blood vessels or cause edema, raising intracranial tension (Lloyd et al., 1966). Hemorrhage may also compress the nerve in the facial canal (Lavin and Weissman, 1985), due to increased intravascular pressure from hypertension, with hemorrhagic complications being a common concern (Cifkovâ, 2014).

Identifying the affected side is crucial for guiding medical management, followed by determining whether it is due to an upper or lower motor neuron lesion (Masterson et al., 2015). The severity of facial weakness informs the prognostic outcome of the therapy (De Almeida et al., 2014). Corticosteroids are widely recognized as the cornerstone of pharmacological treatment, significantly enhancing the likelihood of complete recovery if administered early in the disease course (Baugh et al., 2013). The American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) guidelines recommend a high-dose steroid regimen within 72 hours of symptom onset (Schwartz et al., 2014). However, corticosteroids should not be initiated if labile hypertension is still present (Ellis et al., 1999), despite their proven efficacy in alleviating facial paralysis (Adour et al., 1972). Corticosteroids may exacerbate (Jackson et al., 1981), requiring clinicians to carefully monitor blood pressure during treatment (Jeong et al., 2021). In cases where viral etiology is suspected, alternative treatments, including antiviral agents, may be considered, though their efficacy remains controversial (Riga et al., 2012). The combination of corticosteroids and antiviral agents, such as acyclovir or valacyclovir, may be considered in certain clinical scenarios to reduce the rates of synkinesis (Dalrymple et al., 2023), although this approach is not universally endorsed (Madhok et al., 2016). A case report involving elderly patients with uncontrolled hypertension highlighted the use of antihypertensive therapy to alleviate facial paralysis (Adour et al., 1972), with the benefit of lowering blood pressure per se (Cifkovâ, 2014). For patients requiring steroids, intratympanic steroid injection (ITSI) is a preferred option as it targets the most likely site of the disease through the natural dehiscence of the facial canal and the canaliculus of the chorda tympany without the unwanted side effects of oral steroids (Demir et al., 2020).

The prescription of artificial tears may help to protect the cornea from ulceration in patients who are unable to blink or close their eyes (De Almeida et al., 2014), and those with reduced tear flow (McCaul et al., 2014). Other protective measures, such as night eye patches, ophthalmic ointments before sleep, and eyeglasses (Prabasheela et al., 2017) may also be recommended for eye protection. The use of antiplatelet drugs such as Aspilets can reduce thrombotic complications associated with hypertension (Cifkovâ, 2014). Vitamin B supplementation may help to relieve the symptoms of moderate peripheral neuropathy (Silviana et al., 2021). Vitamin B12 is used clinically to repair nerve damage (Wang et al., 2015), acts as an antioxidant, and regulates growth factors, macrophage function, and the coagulation system. Vitamin B12 deficiency can lead to demyelination and degeneration of the nervous system (Choi et al., 2022). Vitamin B3, as a precursor of coenzyme 1, has also been used as a hypolipidemic agent to prevent atherosclerosis (Prousky, 2012).

Clinical Significance of Hypertensive Crisis as a Comorbid Factor

Hypertensive crisis presents a significant challenge in the management of Bell's palsy. Studies have demonstrated an independent association between hypertension and the incidence of Bell's palsy (Savadi-Oskouei et al., 2008). Severe hypertension has been associated with Bell's palsy, particularly in pediatric populations (Jörg et al., 2013), as well as with type 2 diabetes mellitus (Romadhoni et al., 2024). The pathophysiological mechanisms underlying this association are not entirely understood but may involve vascular and metabolic disturbances that predispose individuals to facial nerve ischemia (Liu et al., 2023; Wang et al., 2024). In this case report, the patient's hypertensive crisis (185/144 mmHg) posed a dual challenge: managing the acute facial paralysis while simultaneously addressing the severe elevation in blood pressure. The management of such cases requires a multidisciplinary

approach, integrating the expertise of neurology, primary care, and possibly cardiology to ensure optimal outcomes (Jiang et al., 2024; Wang et al., 2024; Wu et al., 2024).

Comparison with Stroke

One of the differential diagnoses of Bell’s palsy is stroke (Dalrymple et al., 2023). Distinguishing Bell's palsy from stroke is of paramount importance, especially in primary care settings. While both conditions can present with facial weakness, the underlying pathophysiology, prognosis, and treatment strategies differ significantly. Stroke, typically caused by an interruption of blood flow to the brain, can result in a range of neurological deficits, including hemiparesis, aphasia, and altered consciousness, depending on the affected brain region. In contrast, Bell's palsy is a peripheral neuropathy of the facial nerve, with symptoms generally limited to facial muscles. Bell’s palsy has a sudden onset developing over hours and usually resolves within six months. In contrast, stroke is caused by cerebral hemorrhage and infarction and can produce neurological symptoms such as vomiting, severe headache, rapid progression of neurological deficits, and even coma (Silviana et al., 2021).

Despite their differences, there are some similarities between stroke and Bell’s palsy, including slurred speech and facial drooping. However, stroke is often accompanied by noticeable arm drift. Additionally, while both stroke and Bell’s palsy may present with equal, round, and reactive pupils, Bell’s palsy patients have difficulty closing the affected eye (Mayhew and Carhart, 2015). Given these similarities, the differentiation between these two conditions is crucial to avoid misdiagnosis and inappropriate management. Stroke typically requires urgent intervention, such as thrombolytic therapy to restore cerebral perfusion (Charchar et al., 2024; Coelho et al., 2021; Mueanchoo et al., 2024; Nuraini, 2015), whereas Bell's palsy management focuses on reducing inflammation and supporting facial nerve recovery. The presence of hypertension in Bell's palsy patients can mimic stroke symptoms, underscoring the importance of thorough clinical assessment and appropriate diagnostic imaging (Jeong et al., 2021).

Aligning with the Objective of the Study

This case report aims to highlight the importance of differential diagnosis and careful management of patients presenting with facial paralysis, particularly in primary care settings such as community health centers (*puskesmas*). Distinguishing Bell's palsy from more severe conditions such as cerebrovascular accidents is critical, as the treatment approaches differ significantly (Baugh et al., 2013; Schwartz et al., 2014; Vakharia and Vakharia, 2016). The emphasis on managing comorbid conditions, such as hypertension, underscores the need for a holistic approach to patient care.

Furthermore, this case underscores the clinical significance of hypertensive crises as a potential exacerbating factor for Bell's palsy. The interplay between vascular health and nerve function warrants further investigation to elucidate the underlying mechanisms and improve management strategies (Jiang et al., 2024). By documenting and analyzing such cases, we can contribute to the existing body of knowledge and improve clinical outcomes for future patients.

In addition to the established pharmacological treatments, emerging therapies for Bell's palsy merit consideration. Physical therapy, including facial exercises and neural mobilization, has shown promise in enhancing recovery and preventing long-term complications such as synkinesis (Alharbi et al., 2023). Acupuncture and photobiomodulation therapy are alternative modalities that have demonstrated beneficial effects on facial nerve function and edema, although their widespread adoption requires further validation through randomized controlled trials (Wu et al., 2024). Hyperbaric oxygen therapy (high-pressure oxygen) is another potential treatment option for Bell's palsy (Holland et al., 2012).

Moreover, understanding the genetic and metabolic predispositions to Bell's palsy may help identify at-risk populations and inform preventive strategies. Recent studies have highlighted the potential genetic associations between hypertension, diabetes, and Bell's palsy, suggesting a multifactorial etiology that extends beyond idiopathic origins (Liu et al., 2023). Incorporating genetic screening and metabolic assessments into routine clinical practice could enhance early detection and intervention, ultimately improving patient outcomes.

From a public health perspective, raising awareness of the early signs and symptoms of Bell's palsy, particularly in primary care settings, is crucial. Educational initiatives targeting healthcare providers

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and the general public can facilitate prompt recognition and timely intervention, thereby reducing the disease burden (Warner et al., 2023). Additionally, advocating for regular blood pressure monitoring and management can help mitigate the risk of hypertensive crises and associated complications (Vakharia and Vakharia, 2016).

From the Patient's Perspective

From the patient's perspective, experiencing facial paralysis alongside a hypertensive crisis can be profoundly distressing. Mr. A reported a sudden onset of right-sided facial paralysis and slurred speech, which significantly impacted his daily life and emotional well-being. Understanding the psychosocial impact of Bell's palsy is crucial, as patients often experience anxiety, depression, and social withdrawal due to visible disfigurement and functional impairment (Alharbi et al., 2023).

Effective communication, empathetic support, and clear explanations of the diagnosis and treatment plan are vital in alleviating patient anxiety and fostering adherence to prescribed therapies. Educating patients about the typically favorable prognosis of Bell's palsy, with most individuals experiencing significant recovery within three to six months, can provide reassurance and hope (Alharbi et al., 2023; Wu et al., 2024).

Patient-Centered Care

Adopting a patient-centered approach in managing Bell's palsy involves addressing the individual's physical, emotional, and social needs. Providing comprehensive care plans that include pharmacological treatment, physical therapy, and psychosocial support can enhance recovery and quality of life for affected individuals (Patel and Levin, 2015). Encouraging patient engagement and active participation in their care journey fosters a sense of empowerment and improves adherence to therapeutic regimens.

Strength and Limitations

This case report provides valuable insights into the management of Bell's palsy with comorbid hypertension. However, it has several limitations. First, the findings are based on a single case, which limits the generalizability of the conclusions. Future research should include larger, multicenter studies to validate the observations and recommendations presented here. Additionally, the retrospective nature of the case analysis may introduce recall bias and limit the ability to establish causality.

Further research is needed to explore the pathophysiological mechanisms linking hypertension and Bell's palsy. Longitudinal studies examining the impact of blood pressure control on the incidence and prognosis of Bell's palsy could provide deeper insights into preventive strategies. Moreover, investigating the role of genetic and metabolic factors in Bell's palsy may uncover novel therapeutic targets and improve personalized treatment approaches.

Emerging therapies such as neural mobilization, acupuncture, and photobiomodulation therapy warrant further investigation through rigorous clinical trials to establish their efficacy and safety. Additionally, understanding the psychosocial impact of Bell's palsy and developing interventions to support patients' mental health and social well-being are critical areas for future research.

CONCLUSIONS

This case report underscores the critical importance of distinguishing Bell’s palsy from stroke in patients presenting with facial paralysis and hypertension, particularly in primary care settings such as *puskesmas*. Accurate diagnosis and timely management are vital to avoid misdiagnosis and ensure optimal treatment.

Our findings highlight the necessity for heightened clinical vigilance and comprehensive neurological assessments in such cases. From a public health perspective, improving awareness and training among primary care providers can significantly enhance patient outcomes and reduce the burden of

misdiagnosed neurological conditions. Future research should focus on refining diagnostic protocols and exploring the interplay between hypertension and Bell's palsy.

Conflict of Interest

The authors declare no conflict of interest in relation to this manuscript.

Ethics Consideration

The Health Research Ethics Committee of the Faculty of Medicine at Hang Tuah University in Surabaya reviewed and approved this study, with a certificate number I/051/UHT.KEPK.03/VII/2024 on July 17, 2024. The approval documentation is attached to the manuscript.

Consent Form

The patient has provided informed consent for the publication of this case. All patient data are kept confidential and only used the purpose of this case report.

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