Acta Neurologica Indonesia 2026; 04(01)

Acta Neurologica Indonesia
Department of Neurology, Faculty of Medicine Universitas Indonesia
ISSN 3032-6303

Case Series

EPILEPSY AS A RARE INTRACRANIAL COMPLICATION OF PANSINUSITIS: A CASE SERIES

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ABSTRACT

Introduction Epilepsy is a condition characterized by recurrent and unpredictable seizures with an underlying mechanism that remains incompletely understood. A hallmark feature is over-synchronized neuronal discharge due to the loss of inhibitory control over glutamatergic neurons, with neuroinflammation being a potential contributing factor.

Case Summary We report two cases of epilepsy in patients with a history of pansinusitis. A 14-year-old male experienced a first-onset generalized seizure, while a 23-year-old female presented with recurrent focal-to-bilateral seizures. CT scans revealed pansinusitis, predominantly affecting the bilateral frontal sinuses with bilateral ostiomeatal complex blockage in one patient, and the maxillary sinuses with an open bilateral ostiomeatal complex in the other patient. No evidence of intracranial infection or other epilepsy causes were found, suggesting epilepsy as an intracranial complication (IC) of pansinusitis. One patient was managed with medication, while the other underwent functional endoscopic sinus surgery. (FESS), revealing sinonasal polyps on histology.

Discussion ICs, including epilepsy, are rare but serious complications of pansinusitis and can lead to disabling neurological sequelae. The most common source of IC is frontal sinusitis, followed by ethmoid, sphenoid, and maxillary sinusitis. ICs occur via direct infection spread through dehiscent bone or through natural pathways, such as the olfactory foramina. Neuroinflammation can activate microglia and trigger reactive astrogliosis, leading to inflammatory cascades, neurotransmitter imbalances, and ion channel dysfunction.

Conclusion We have presented rare cases of epilepsy as an IC of pansinusitis, highlighting the need for thorough evaluations in epilepsy patients without known intracranial infections, including investigations for other potential infectious sources.

Keywords: epilepsy; pansinusitis; intracranial infection; neuroinflammation

INTRODUCTION

Epilepsy is a chronic neurological condition characterized by recurrent and unpredictable seizures, with an underlying mechanism that remains incompletely understood.[1] It continues to represent a global disease burden, with an estimated prevalence of 7.6 per 1,000 people and affecting approximately 50 million individuals worldwide. It is the second most common neurological disorder after headaches.[2,3] Various contributing factors can lead to epilepsy. In children, it is commonly caused by genetic factors, perinatal insults, and malformations. In adults without

genetic predisposition, common causes include brain injuries, brain tumors, stroke, infections, neuroinflammation, neurodegenerative disorders, and congenital abnormalities. Approximately 30% of all epilepsy cases continue to experience refractory seizures despite treatment with antiepileptic drugs (AEDs), which can increase morbidity and mortality and reduce quality of life. Several predictive risk factors for intractable epilepsy have been identified, one of which is abnormal findings on imaging and neuropathological examination. Patients with predictive factors for intractable epilepsy need to be identified and the underlying causes addressed to prevent further deterioration.[4]

Over the past decade, growing evidence has indicated a significant contribution of neuroinflammation and infections in the pathophysiology of epilepsy.[5] One possible infectious source is sinusitis. Sinusitis, or inflammation of the sinuses, is a common condition and ranks as the fifth most frequently treated illness with antibiotics. Sinusitis can be categorized into acute and chronic types. Acute sinusitis is usually viral, whereas chronic sinusitis is mostly caused by gram-negative bacteria, such as Staphylococcus aureus, or fungi.[6] For diagnosis, both forms require at least two of the following: purulent nasal or nasopharyngeal discharge, nasal congestion, pain, tenderness, or swelling around the sinuses.[7]

Anatomically, the sinuses are located close to the eyes and skull, so when locoregional complications occur, they typically involve oculo-orbital and cranio-encephalic pathologies.[8] Complicated sinusitis is diagnosed when symptoms last for more than 10 days, the patient has a fever above 38°C, or there are signs of extra-sinus or systemic involvement. With appropriate treatment, complications of sinusitis are rare. However, approximately 3.7% of cases may develop intracranial complications of sinusitis (ICS), which can lead to life-threatening conditions and require hospitalization.[6,9]

The most common ICS include subdural empyema (55.6%), brain abscess (18.5%), and meningitis (11.1%). Infectious spread can occur through two main mechanisms, either via (1) thrombi or septic emboli that travel retrograde through the valveless diploic veins of the

skull, or (2) through direct extension of infection from the bone via congenital or traumatic defects, existing foramina, or erosion of the sinus walls.[6] These complications are supposed to be rare, so any concern about such cases should be taken seriously. Early and well-coordinated multidisciplinary management can significantly improve prognosis.[10] In this study, we present two cases of epilepsy as an intracranial complication of pansinusitis, aiming to highlight the importance of early diagnosis and appropriate treatment to prevent further deterioration.

CASE REPORT

Case 1

A 14-year-old boy presented with a first-onset generalized tonic-clonic seizure, lasting approximately 2 minutes, during which his eyes rolled upward. The patient denied any headache, fever, vomiting, or prior history of seizures, but he did have a history of acute rhinosinusitis. During the event, the patient had no recollection of the seizure and denied any head injury. After the seizure, it took around 5 minutes to regain full consciousness. He only complained of left shoulder pain but denied any other neurological symptoms. He was healthy at birth, had normal developmental milestones, and was fully vaccinated according to age.

On physical examination, neurological and motor examinations were normal, although the range of motion (ROM) in the left shoulder was limited. Mucopurulent discharge was noted in both nostrils. Laboratory results, including complete blood count, renal and liver function tests, glucose, and electrolytes, were unremarkable. A head CT scan revealed pansinusitis with obstruction/occlusion of the right and left ostiomeatal complexes, a mild rightward deviation of the nasal septum, and adenoid hypertrophy. Head MRI with contrast showed fluid and markedly enhancing mucosal thickening, nearly filling the bilateral maxillary, ethmoidal, and sphenoidal sinuses, although no sinus wall expansion was observed (Figure 1).

He was diagnosed with a first-onset seizure, suspected epilepsy, acute pansinusitis, and left humeral contusion. The patient was treated with mometasone furoate 50 mcg/dose 1 spray once daily, montelukast sodium 10 mg once daily, desloratadine 5 mg once daily,

and levetiracetam 500 mg twice daily, which successfully controlled the seizures.

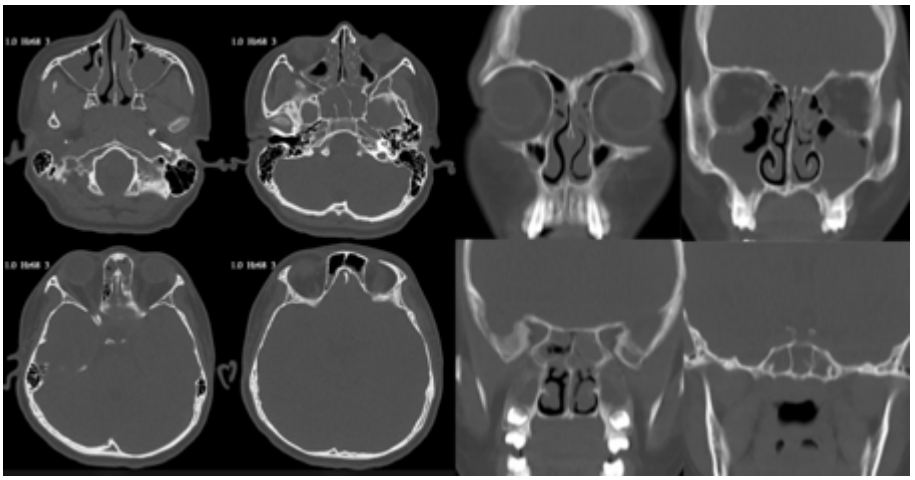

Figure 1. Non-contrast head CT scan showed mucosal thickening of the bilateral frontal, maxillary, ethmoidal, and sphenoidal sinuses, consistent with pansinusitis. The right and left ostiomeatal complexes were obstructed, with evidence of adenoid hypertrophy and a mild rightward deviation of the nasal septum.

Case 2

A 23-year-old female presented with recurrent focal-to-bilateral seizures. The first episode occurred 10 months prior to hospital admission, and the second episode, consisting of a total of 11 seizures, occurred 2 weeks before admission and was diagnosed as status epilepticus. The patient denied any headache, fever, vomiting, or previous history of seizures, but reported experiencing chronic nasal congestion, which had worsened over the past month. During the event, the patient had no recollection of the seizures and denied any head injury. After the seizure, she reported no noticeable neurological symptoms. She was born healthy, with no perinatal complications, and had normal developmental milestones until the first seizure occurred. The patient noted that she had been feeling unwell due to working night shifts as part of her nursing profession.

On physical examination, neurological and motor exams were normal, but mucopurulent discharge was observed from both nostrils. Laboratory tests, including complete blood count, renal and liver function tests, glucose, and electrolytes, were all within normal limits. A head CT scan showed pansinusitis, predominantly in the bilateral maxillary sinuses, with both ostiomeatal complexes open, findings suggestive of rhinitis, and a leftward deviation of the nasal septum (Figure 2).

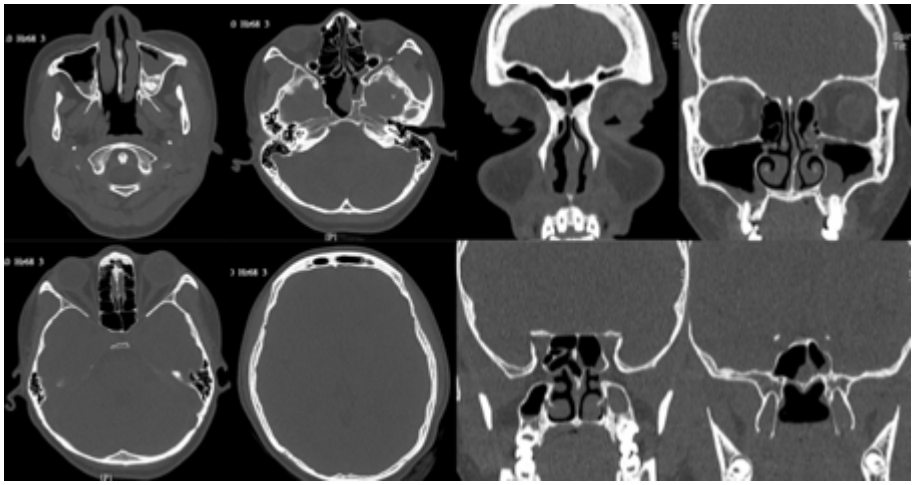

Figure 2. Non-contrast head CT scan showed mucosal thickening of the bilateral frontal, maxillary, ethmoidal, and sphenoidal sinuses, consistent with pansinusitis. The ostiomeatal complexes were open, accompanied by conchal hypertrophy and a leftward deviation of the nasal septum.Non-contrast head CT scan (axial, sagittal, and coronal views) of a 61-year-old male presenting with persistent hiccups.

The patient was diagnosed with idiopathic epilepsy and chronic pansinusitis. She was treated with phenytoin 200 mg twice daily, levetiracetam 500 mg twice daily, and folic acid 400 mcg once daily. The patient subsequently underwent Functional Endoscopic Sinus Surgery (FESS) and nasal septum resection. Postoperatively, there was only minimal discharge, pain was tolerable, and no further seizure episodes occurred.

Histopathological examination revealed features of a sinonasal polyp, consisting of a protruding mass of tissue lined by pseudostratified ciliated columnar epithelium. The underlying stroma contained glandular structures and showed a mild infiltration of chronic inflammatory cells.

DISCUSSION

Epilepsy is a well-known chronic neurological disorder; however, the underlying mechanism of epilepsy remains incompletely understood.[11] Properly regulated neuronal activity plays a crucial role in maintaining the excitation-inhibition balance of the central nervous system (CNS). Therefore, one of the hallmark features of epilepsy is believed to be over-synchronized neuronal discharge due to the loss of inhibitory control over glutamatergic neurons.[12]

The development of epilepsy, which occurs gradually, is known as epileptogenesis. It typically follows a brain insult or occurs during the insult, eventually leading to the first spontaneous seizure. This process involves multiple brain regions,

including the cortex, midbrain, basal ganglia, thalamus, hippocampus, and cerebellum.[12] During epileptogenesis, neurons undergo various modifications that increase excitatory tendencies, form crucial interconnections, and undergo complex morphological alterations. The changes in epileptogenesis are generally categorized into three phases: (1) Acute phase, lasting minutes to days, characterized by modifications in receptor regulation, ion channels, and post-translational alterations; (2) Subacute phase, lasting hours to weeks, marked by the activation of the inflammatory cascade, neuronal loss, and increased expression of neurotrophic factors; (3) Chronic phase, lasting weeks to months, driven by anatomical changes such as mossy fiber sprouting, gliosis, neurogenesis, and network reorganization. The chronic phase may result in late-onset seizures and the development of chronic epilepsy, which is often unresponsive to pharmacotherapy.[5]

In this case series, the first patient experienced a first-onset generalized tonic-clonic seizure without any other identifiable etiology apart from an underlying inflammatory sinus process. Therefore, the ongoing sinusitis, if left untreated, could potentially lead to recurrent seizures and further epileptogenesis. The patient was likely in the acute or subacute phase of epileptogenesis, as the parents could not recall the exact onset of the rhinosinusitis, only that it had begun either within the past few days or weeks. The second patient experienced recurrent seizures, including an episode of status epilepticus, with a background of chronic pansinusitis and nasal polyps. Her epileptogenesis appears to fall into the chronic phase, which is consistent with her limited responsiveness to pharmacological therapy, ultimately requiring surgical intervention for adequate seizure control.

Inflammation represents a complex response that occurs post-infection or post-injury, aimed at eliminating pathogens and initiating tissue regeneration. However, inflammation can also lead to pathological consequences, including cell injury or loss. Both innate and adaptive immune responses can occur within the CNS or the periphery, eventually reaching brain tissue through alterations in blood-brain barrier (BBB) permeability. This may happen via

cytokine-regulated stimulation of metalloproteases or disruption of tight junctions. Such BBB impairment is considered a hallmark mechanism of brain injury. In addition to the infiltration of inflammatory molecules and leukocytes, a disrupted BBB also permits the extravasation of serum proteins into the brain. This leakage of serum proteins contributes to neural network overexcitability, playing a significant role in the pathogenesis of epilepsy.[5] As seen in both patients in this case series, the presentation of seizures, coupled with sinonasal infections, mucosal inflammation, and sinus opacification on MRI, suggests a strong correlation between the inflammatory sinus process and increased neuronal excitability, particularly in young or otherwise healthy individuals with no prior seizure history or other known risk factors.

Rhinosinusitis is an inflammation of the mucosa of the paranasal sinuses, with or without involvement of the underlying osseous tissue of the nasal cavity. The term rhinosinusitis is preferred over sinusitis because inflammation of the paranasal sinuses almost always occurs together with inflammation of the nasal cavity. Based on duration, rhinosinusitis is classified into acute and chronic. According to the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS), acute rhinosinusitis is defined as the presence of at least two typical symptoms, (1) nasal blockage (obstruction or congestion) and (2) rhinorrhea (anterior or postnasal), accompanied by confirmation from either mucopurulent nasal discharge, the presence of nasal polyps, endoscopic findings of edema in the middle meatus, or a CT scan showing mucosal changes in the ostiomeatal region. EPOS further categorizes acute rhinosinusitis based on frequency and symptom pattern into: (1) acute viral rhinosinusitis (common cold), where symptoms last for 10 days or less; (2) acute post-viral rhinosinusitis, in which symptoms persist beyond day 10 or worsen after day 5; and (3) acute bacterial rhinosinusitis, diagnosed when three or more of the following are present: purulent (discolored) nasal discharge, severe localized facial pain, fever of 38°C or higher, elevated CRP or ESR, and "double sickening" (a second worsening of symptoms following initial improvement).[13] The first patient in this case series

experienced acute rhinosinusitis, without fever, headache, or toxic appearance. However, the presence of persistent symptoms lasting ≥10 days without improvement, along with mucopurulent nasal discharge, classifies the condition as acute post-viral rhinosinusitis. Therefore, antibiotics were not administered; instead, the patient received supportive management only.

Recurrent acute rhinosinusitis (RARS) is defined as four or more episodes of acute rhinosinusitis within a year, with symptom-free intervals during which symptoms resolve completely within 12 weeks. Chronic rhinosinusitis (CRS), on the other hand, is defined as inflammation of the nasal and paranasal sinus mucosa lasting 12 weeks or more. It is characterized by four cardinal symptoms: mucopurulent nasal drainage (anterior or posterior), facial pain or pressure, nasal obstruction, and a reduced or absent sense of smell.[14,15] CRS can be further classified into three types: CRS with nasal polyps (1-4% in the general population), CRS without nasal polyps (11% in the general population), and allergic fungal rhinosinusitis. These classifications are important in determining treatment approaches.[13,16] According to the criteria for CRS, the second patient presented with nasal obstruction lasting ≥12 weeks, mucopurulent drainage from both nostrils, along with radiological evidence of CRS and histological confirmation of nasal polyps, thus classifying the condition as CRS with nasal polyps.

The pathophysiology of rhinosinusitis involves three major factors: abnormal ciliary function, obstruction of sinus drainage pathways, and alterations in mucus composition or quantity. Under normal conditions, the sinuses produce mucus that is transported by ciliary action through the sinus ostia into the nasal cavity, down the pharynx, and eventually swallowed. However, upper respiratory

infections, inflammatory processes, edematous mucosa, and anatomical variations can impede this mucus outflow. This results in mucus retention, negative pressure in the sinus cavity, bacterial proliferation, and the development of acute rhinosinusitis.[13]

Several risk factors contribute to rhinosinusitis, including rhinitis, anatomical abnormalities, comorbid conditions, and environmental factors. The condition is usually not caused by a single pathogen but rather by a combination of organisms. The most common pathogens include Streptococcus pneumoniae (20–43%), Haemophilus influenzae (22–35%), Moraxella catarrhalis (2–10%), and Staphylococcus aureus (10%). In cases of severe or complicated rhinosinusitis, the most frequently implicated pathogens are Streptococcus milleri, α-hemolytic streptococci, and anaerobic bacteria.[13]

The majority of acute bacterial rhinosinusitis cases can recover spontaneously without the use of antibiotics. Therefore, the AAO-HNS guidelines recommend two possible management options: watchful waiting or prescription of antibiotics. Watchful waiting is appropriate when follow-up is available, whereas antibiotics should be considered in cases where there is no improvement within 7 days, worsening of symptoms, or the presence of severe disease, typically indicated by fever and unilateral facial pain. The first-line antibiotic for adults is co-amoxiclav for 5–7 days, while for children, either amoxicillin or co-amoxiclav is recommended for 10–14 days. In pediatric cases where oral antibiotics cannot be administered, due to vomiting, ceftriaxone may be given intravenously or intramuscularly at a single dose of 50 mg/kg, followed by oral therapy once symptoms improve after 24 hours.

However, in cases where maximum-dose medical therapy fails to provide symptom relief, or in cases of recurrent acute rhinosinusitis, chronic rhinosinusitis, or when specific infectious complications are present, surgical intervention may be considered. A study by Laila et al. reported that 30% of patients were managed exclusively with antibiotics, while the remaining 70% required surgery, with 30% undergoing nasoendoscopic procedures, 30% undergoing

neurosurgical intervention, and 10% requiring both types of surgery.[17]

In CRS, treatment options include both medical and surgical approaches. Oral antibiotics, particularly macrolides, are often used, although long-term therapy of up to 3 months remains controversial. Topical steroids are also an effective treatment of choice due to the inflammatory nature of CRS, and their long-term use for 3–6 months is still recommended, particularly in obstructive cases or as supportive therapy after surgery. Although oral corticosteroids have potent anti-inflammatory effects, their systemic side effects must be carefully considered, and they are generally not recommended for long-term use. Medical treatment is typically the initial step, while in persistent CRS, regardless of whether CRS is with or without polyps, endoscopic sinus surgery (ESS) is commonly performed as it helps to remove polyps, open obstructed ostiomeatal complexes, clear retained secretions, and facilitate delivery of topical agents.

In rare cases, aggressive rhinosinusitis may develop, especially in immunocompromised patients, adolescents, and children older than 6 years.[18,19] Complications of rhinosinusitis can be divided into three major categories: intracranial, orbital, and bony/soft tissue complications. Intracranial complications occur in approximately 3–13% of acute bacterial rhinosinusitis cases, with a mortality rate of 2–7% among those affected. These complications may include meningitis, encephalitis, brain abscess, subdural or epidural empyema, and venous sinus thrombosis. Infection can spread either directly through bone or other structures that are in close proximity between the sinuses and intracranial spaces, or hematogenous, as in the majority of cases. Transmission to the meninges can occur through the diploic veins located behind the frontal sinus, which lack venous valves. Clinical manifestations are often nonspecific, such as headache, lethargy, fever, or focal neurological symptoms, making diagnosis more challenging and often resulting in delayed diagnosis. In the United States, the average time to diagnosis is approximately 8 days after symptom onset.[7] Patients with ICS may develop long-term sequelae, including epilepsy, permanent visual disturbances, and focal paresis.[6] Both patients in this case series were otherwise healthy but belonged to the adolescents and older pediatric

groups, which placed them at increased risk for developing ICS.

Radiologic evaluation is essential when ICS is suspected. CT scan is the first-line neuroimaging modality and is considered the gold standard for visualizing both the sinuses and intracranial structures. MRI is usually performed before surgery due to its higher resolution and superior soft tissue contrast.[6] As observed in both patients, they developed ICS which may initially involve microscopic or inflammatory changes, such as BBB disruption, cytokine diffusion, and perivascular inflammation, before progressing to gross structural lesions. These early neuroinflammatory processes may not yet be detectable on imaging. Moreover, not all ICS cases manifest as space-occupying lesions. The brain can experience functional disturbances without permanent anatomical damage, such as neuronal hyperexcitability, glial activation, or cytokine-induced neurotoxicity, all of which may lower the seizure threshold and provoke epilepsy. Therefore, even in the absence of radiographic abnormalities, both patients experienced seizures coinciding with active pansinusitis involving the frontal and ethmoidal sinuses, which are adjacent to the anterior cranial fossa, supporting the possibility of inflammatory spread causing cortical irritation and seizure activity.

The treatment of ICS typically requires a multidisciplinary approach involving both neurologists and ENT specialists. There is no universally established standard or duration of treatment, but management of sinusitis requires broad-spectrum intravenous antibiotics capable of penetrating the BBB. In most cases, long-term IV antibiotics are recommended, typically for at least 6–8 weeks, with the duration tailored according to the clinical course and radiologic findings on follow-up.[20] In addition to medical therapy, patients who do not show improvement within the first 48 hours often require surgical intervention, usually in the form of FESS or surgical drainage of affected sinuses, which are the most commonly performed procedure.[6,9] Both patients in this case series responded to sinusitis-targeted treatment. The first patient showed improvement with conservative medical therapy, as the condition was most likely viral, while the second patient experienced seizure

remission following FESS and septoplasty.

This case series has several limitations, primarily the small sample size of two patients, which may limit the generalizability of the findings and the ability to establish a causal relationship between sinusitis and epilepsy. Second, although both patients presented with seizures in the context of active pansinusitis and no other identifiable etiology or contributing factors, causality cannot be definitively established due to the absence of radiological evidence of intracranial involvement. Third, long-term follow-up was limited, making it difficult to assess the risk of seizure recurrence or the development of chronic epilepsy.

CONCLUSION

Intracranial complications of sinusitis (ICS) are rare, particularly in immunocompetent patients. However, due to the anatomical proximity of the sinuses to critical structures and the fragility of the surrounding physiology, patients with pansinusitis or rhinosinusitis involving the frontal sinus can develop life-threatening complications, with isolated epilepsy being one of the most common manifestations. In addition, patients presenting with red flag signs such as fever, toxic appearance, and systemic symptoms should raise a strong suspicion for ICS. Therefore, although ICS is uncommon, it carries significant morbidity and mortality. Early recognition and appropriate treatment of intracranial complications of pansinusitis are essential to prevent disease progression and improve clinical outcomes, especially in cases where symptoms are nonspecific and imaging does not immediately reveal the extent of intracranial involvement. Functional disturbances, such as neuroinflammation or blood-brain barrier (BBB) disruption may represent the primary pathophysiological mechanisms of epileptogenesis. Therefore, the absence of radiological findings does not exclude ICS, especially in its early or inflammatory stages.

ACKNOWLEDGEMENT

The authors would like to thank the patients and their family, Department of Neurology, and Faculty of Medicine Pelita Harapan University for the support and contributions in making this report.

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