CASE REPORT

Horner Syndrome Following Permcath Insertion in a Child with End-Stage Renal Disease: A Case Report

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ABSTRACT

Horner syndrome, characterized by the triad of unilateral ptosis, miosis, and anhidrosis, typically arises following a stroke, surgical interventions in the neck and chest, or trauma. Horner syndrome is rare in children. This study presents the case of a 9-year-old girl with End-Stage Renal Disease (ESRD) caused by renal hypodysplasia. After permcath insertion, she developed a severe headache and anisocoria, followed by ptosis and a progressively expanding neck hematoma. Physical examination and MRI of the neck revealed that Horner syndrome, caused by hematoma formation following permcath placement, was the diagnosis. Supportive interventions were implemented, leading to significant improvement in Horner syndrome over a six-month period. Complications from permcath insertion can be a cause of Horner syndrome.

Introduction

Horner syndrome is characterized by unilateral ptosis, miosis, and anhidrosis. This syndrome results from sympathetic innervation disruption to the eye because of diverse etiologies, including congenital and acquired origins (1). Congenital Horner syndrome typically arises from birth trauma, while acquired cases occur post-stroke, after surgical procedures in the cervical and upper thoracic regions, or due to trauma (2).

Horner syndrome, resulting from lesions of the internal carotid artery, typically presents with unilateral head and/or neck pain, along with ptosis and miosis, but not anhidrosis. This presentation

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arises due to a lesion affecting the sympathetic fibers within the internal carotid plexus while sparing the external carotid plexus, which is responsible for innervating the facial sweat glands. Horner syndrome often occurs due to trauma or dissection of the cervical internal carotid artery, resulting in painful symptoms. Furthermore, mass lesions in the neck can potentially compress the carotid sympathetic neurons, consequently leading to Horner syndrome. The recovery course from Horner syndrome induced by jugular vein catheterization varies, ranging from complete resolution to persistent residual symptoms (3).

In addition, rare occurrences of Horner syndrome have been documented following internal jugular vein catheterization, with an incidence estimated to be less than 5% (4, 5). This case study presents the case of a 9-year-old girl with End-Stage Renal Disease (ESRD) who developed a severe headache and anisocoria following permcath placement. Subsequent investigations revealed that a permcath-induced hematoma precipitated Horner syndrome. By reporting this case, the present study aims to inform clinicians about Horner syndrome's rare and possible etiology.

Case Report

Clinical Presentation

A 9-year-old girl, presenting with a serum creatinine level of six and uremic symptoms, became a candidate for hemodialysis. Six months earlier, she was diagnosed with chronic kidney disease due to renal hypodysplasia following an evaluation for short stature and poor weight gain (height 113 cm and weight 18 kg, both under the 5th percentile). Despite supportive therapy, her glomerular filtration rate (GFR) continued to decline, and uremic symptoms worsened, necessitating hemodialysis for the first time. She had no history of neurologic disease. To facilitate vascular access for hemodialysis, a right internal jugular permcath was placed.

The patient experienced severe headache and light sensitivity within a few hours post-procedure. Blood pressure recorded during the headache episode was 140/90 mmHg. Despite successful control of hypertension, the headaches persisted.

Subsequently, bleeding was reported at the permcath insertion site, and management was done with pressure dressing, vitamin K supplementation, tranexamic acid administration, and an infusion of fresh frozen plasma. The permcath site exhibited recurrent bleeding. Furthermore, episodes of epistaxis, hematemesis, and bleeding from the intravenous line were observed on the following day post-surgery. Given the patient's underlying chronic kidney disease, the possibility of platelet dysfunction secondary to uremic state was considered. Therefore, platelet transfusion was initiated, resulting in successful control of bleeding.

Diagnostic Workup

Due to persistent severe headaches during the bleeding episodes, the patient was further examined, and anisocoria and ptosis were observed on the right eye. A large hematoma was found two days after permcath insertion on the right side of the neck. Chest X-ray yielded normal findings (Figure 1). The patient underwent a CT scan of the head and neck to rule out cerebral hemorrhage. The emergency CT scan revealed no evidence of cerebral hemorrhage. However, it did show significant inflammatory changes and a probable hematoma at the base of the neck on the right side, extending into the pharyngeal space (Figure 2).

Neurology and neurosurgery consultations were

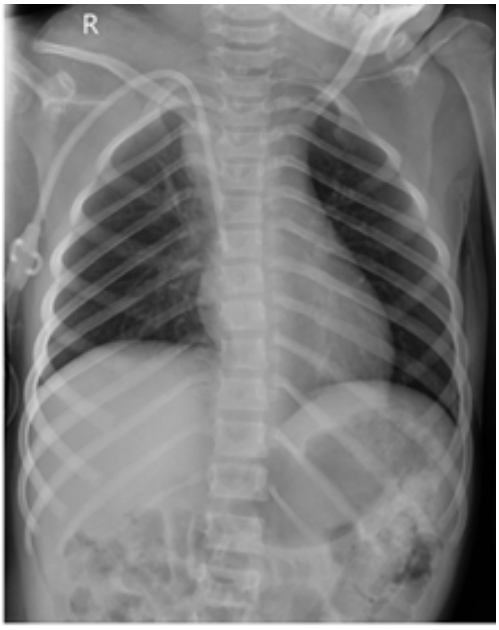

Figure 1. Chest X-ray of the 9-year-old patient after Permcath insertion

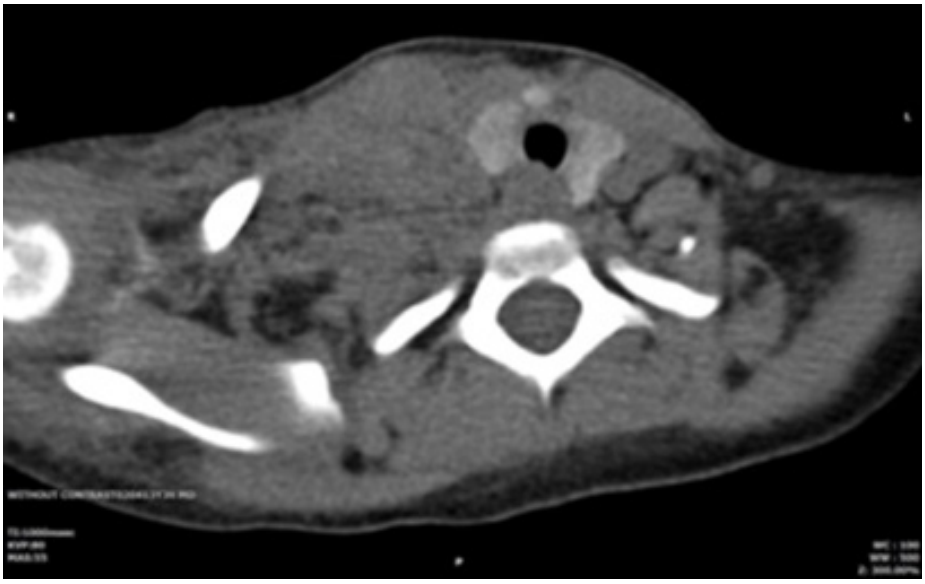

Figure 2. Right-sided neck hematoma on cervical CT scan of the 9-year-old patient with ESRD following permcath insertion

conducted. In neurological examination, no evidence of damage to other neural structures or deficits was revealed. However, miosis and ptosis of the right eye were observed. Ultrasound examination of the neck was unremarkable, demonstrating no signs of vascular injury. Subsequent MRI of the brain and neck also revealed findings consistent with a hematoma on the right side of the neck. Consequently, the Horner syndrome diagnosis was established due to the ptosis, miosis in the right eye, and the observed large neck hematoma.

Subsequent to the prior occasions of bleeding from the permcath insertion site and gastrointestinal tract, further investigations were undertaken. Hematological assessments, including complete

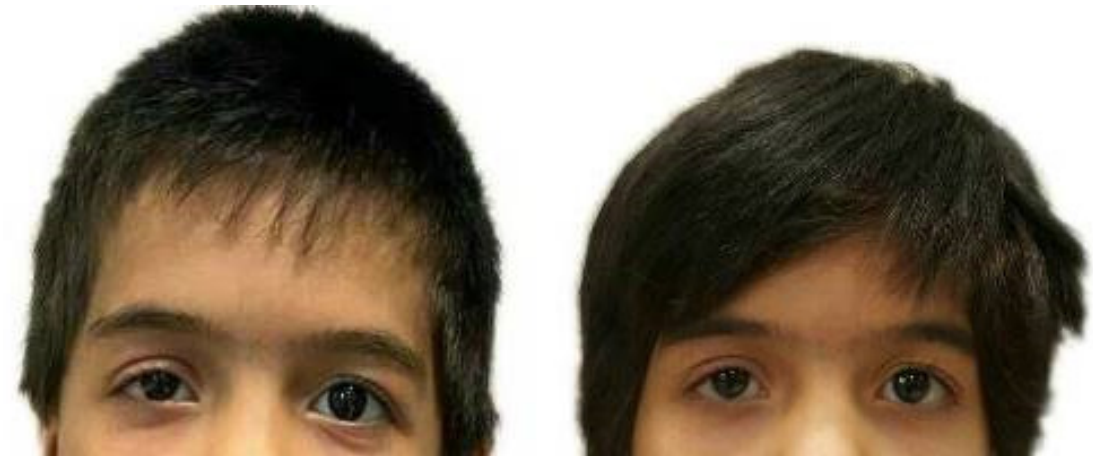

Figure 3. Right eye ptosis one month (left) and six months (right) post-permcath placement in a 9-year-old patient with ESRD

blood count (CBC), prothrombin time (PT), partial thromboplastin time (PTT), bleeding time, clotting time, factor VIII assay, fibrinogen levels, platelet function assay, fibrin degradation products (FDP), D-dimer levels, von Willebrand factor antigen (VWF Ag), and ristocetin cofactor test, all returned within normal limits. Following clinical stabilization, an endoscopic examination was conducted because of the occurrence of hematemesis, which revealed findings consistent with gastritis.

Management

One week following the reduction in cervical hematoma, headaches, and photophobia significantly decreased. Supportive interventions were implemented for Horner syndrome. Subsequently, Horner syndrome significantly improved within six months (Figure 3).

Discussion

Horner syndrome is a neurological disorder originating from impairment of the oculosympathetic pathway. This pathway includes a 3-neuron chain that arises in the hypothalamus and descends to innervate different structures. Consequently, the functions of this pathway comprise vasoconstriction of facial blood vessels, facial sweating, dilation of the pupil, and preservation of an open position of the eyelids (6). Horner syndrome should be included in the differential diagnosis of patients presenting with anisocoria. After confirmation of Horner syndrome, a comprehensive evaluation is needed to determine its underlying etiology.

A detailed history and physical examination are crucial for identifying potential causes of Horner syndrome. Factors such as repeated puncture attempts, accidental carotid artery puncture, or hematoma formation may increase the risk of Horner syndrome following permcath placement (7, 8). Patients with chronic kidney disease are prone to increased bleeding tendencies, leading to various manifestations such as gastrointestinal bleeding, bleeding from cannulation sites, retinal hemorrhage, subdural hematoma, epistaxis, hematuria, ecchymosis, and purpura(9). In this case, hematoma formation was the etiology of Horner syndrome. The hematoma formation was not associated with carotid artery puncture but could be linked to bleeding tendencies observed in ESRD patients, potentially because of soft

tissue damage or venous wall injury (10).

The occurrence of Horner syndrome as a complication of medical procedures involving vascular access has been documented in several cases. Nowak et al. documented a case involving a young woman who was undergoing chemotherapy for gestational trophoblastic disease and required venous port implantation due to poor peripheral vein access. Even with ultrasound guidance, the procedure failed and resulted in a local hematoma. This hematoma caused compression of the sympathetic nerves, leading to Horner syndrome (10). Similarly, Vinod et al. described a 37-year-old man with end-stage chronic kidney disease who developed Horner syndrome after a right internal jugular double lumen central venous catheter (CVC) was placed on the second attempt without ultrasound guidance. Following hemodialysis, the patient developed anisocoria, partial ptosis, miosis, and enophthalmos on the right side (7).

Yamamoto et al. documented a case of a 60-year-old male patient with colon cancer who developed transient Horner syndrome after undergoing central venous port placement for chemotherapy. The day after the procedure, the patient experienced difficulty seeing out of his right eye and was diagnosed with Horner syndrome, characterized by right ptosis and miosis, caused by a transient nerve block from local anesthesia. Further evaluation confirmed that the syndrome was due to sympathetic nerve injury caused by the puncture needle (5).

These cases highlight the importance of careful technique and vigilance during vascular access procedures, as hematoma formation and subsequent nerve compression can lead to complications such as Horner syndrome. Clinicians should be aware of this potential etiology and consider it in the differential diagnosis when patients present with symptoms consistent with Horner syndrome following such procedures.

In Conclusion

This case highlights Horner syndrome as a rare complication of permcath placement in a pediatric ESRD patient, likely resulting from hematoma formation. Anisocoria following such procedures should trigger a comprehensive investigation, specifically given the increased bleeding risk in ESRD. This case underscores the need for diligent post-procedure monitoring and prompt recognition of neurological symptoms. Although the patient experienced significant improvement with supportive care over six months, further research into preventive measures and management strategies for similar complications in ESRD patients may be valuable.

Acknowledgment

None.

Authors' Contribution

Masoumeh Mohkam and Shiva Fatollahierad contributed to the diagnosis and clinical management of the patient, as well as the drafting and revision of the manuscript. Farzad Ahmadabadi was involved in patient management and the finalization of the manuscript. Mitra Khalili contributed to the diagnosis and provided critical revisions to the final manuscript. All authors have read and approved the final version of the manuscript.

Conflict of Interest

None.

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