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Orange Urine in a Newborn: Could It Be a Surgical Reason

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Abstract

Pediatricians occasionally encounter orange urine in new-borns, mainly breastfed babies. This usually happens in the first few days due to the deposition of urate crystals in concentrated urine and resolves on its own as feeding establishes. However, orange urine can also indicate dehydration or, less commonly, an underlying condition in older infants.

This case report describes a 19-day-old male patient, fifth in birth order, who presented with a chief complaint of orange urine over diaper, frequent non-projectile vomiting, feeding refusal, and signs of dehydration. While not the typical presentation, he was diagnosed with idiopathic hypertrophic pyloric stenosis (IHPS).

Unlike the usual IHPS symptoms of forceful vomiting, wanting to eat more after vomiting, and electrolyte imbalances of hypokalemic, hypochloremic metabolic alkalosis in the firstborn child, this baby did not have forceful vomiting, and he refused to eat. He underwent surgery, but his recovery was complicated by two unrelated illnesses: RSV bronchiolitis and pyelonephritis (kidney infection). Both were treated successfully, and follow-up tests (renal ultrasound and VCUG) were negative.

This case highlights the importance of a high index of suspicion for IHPS in the absence of a typical presentation and investigation of orange urine, as it could signify an underlying serious condition.

Keywords: Orange Urine; Idiopathic Hypertrophic Pyloric Stenosis; Metabolic Alkalosis; Pyloromyotomy; RSV; New-born.

Introduction

Uric acid crystals cause orange-stained diapers in new-borns. This is not linked to higher urinary uric acid levels. It is benign in otherwise healthy new-borns and resolves on its own over time [1].

IHPS is common in firstborn male children. Typically occurring in the first few months of life, infants present with non-bilious, forceful vomiting. Correction of electrolyte abnormality and pyloromyotomy remains the standard treatment [2].

This case presents an atypical presentation of IHPS in a 19-day-old male infant. Unlike the classic presentation, the patient did not have forceful vomiting, electrolyte abnormalities, or a firstborn male status. Instead, the primary symptom was orange-stained diapers.

We aim to present this case to emphasize the importance of a high index of suspicion to diagnose causes of vomiting leading to dehydration in new-born patients, which may need timely surgical repair.

Case Report

A 19-day-old male presented with a one-day history of orange-coloured urine. The patient was sick with cough and cold. He had multiple sick contacts at home had non-bloody, non-bilious, and non-projectile vomiting's. He was seen by another doctor the day before, and the flu test was negative. He had decreased oral intake of both breast milk and reduced urine output. He was not hungry after throwing up and had no fever, but his mother noted that he was unusually cold to the touch.

On examination, the patient had mild dehydration with dry oral mucosa; Temp was 36.2. Notably, he had not regained his birth weight of 4.35 kilograms, weighing only 4.139 kilograms on the 19th day of life.

Given the history of orange-coloured urine, poor feeding, decreased urine output, frequent vomiting, exposure to sick individuals, and potential hypothermia, the patient was sent to the emergency room (ER) for further evaluation. The pediatrician suggested that the orange urine might be due to urate crystals in concentrated urine, sometimes associated with dehydration. Additionally, they raised the concern of potential sepsis and recommended further evaluation and intravenous fluids to address dehydration in the ER.

The patient received a 20 ml/kg normal saline bolus in the ER. Labs were ordered to rule out sepsis. While the C-reactive protein and white blood cell count were normal, the CBC revealed elevated red blood cell count, hemoglobin, and hematocrit, indicating dehydration. Electrolytes were normal, ruling out hypokalemic or hypochloremic metabolic alkalosis. Urine analysis showed microscopic ketonuria, proteinuria, and glucosuria but no signs of urinary tract infection.

Despite normal infection markers, the infant continued to vomit in the ER. An abdominal ultrasound was performed to investigate potential surgical causes. The ultrasound revealed a mildly elongated pylorus measuring 20.5 mm with a muscle thickness of 5.2 mm. importantly, gastric contents were not observed passing through the pylorus, suggesting a diagnosis of pyloric stenosis (Tables 1-3).

	Result	**Normal range**
Hgb	21 g/dl	10.3-17.6g/dl
Hct	62.1%	51.3-53.6 %
RBC	6.25	3.12-5.26 10^6/mcl
RDW-SD	59.8	40-55fl
Sodium	139	135-145 mEq/L
Potassium	4.3	3.5-6 mEq/L
Chloride	100	96-106 mEq/L
BUN	8	2-20mg/dl
Creatinine	0.44	0.3 to 1mg/dl
CRP High sensitivity	0.44	<0.5mg/l
CO2	30	35-45 mEq/L
Anion gap	13.3	Less than 12 mEq/L
Urine clarity	Turbid	Negative
Urine protein	30 mg/dl	Negative
Urine glucose	50 mg/dl	Negative
Urine Ketones	5 mg/dl	Negative
Urine Blood	Negative	Negative
Urine Nitrite	Negative	Negative
Urine Leukocyte esterase	Negative	Negative

Table 1: Lab results.

The patient was referred to a pediatric hospital from the ER for specialized care. On the day 20th of life, he underwent a pyloromyotomy. The surgery was successful and patient tolerated the procedure well. He was discharged home on the 21st day of life.

The following day, day 22nd of life, the patient returned to the ER with a refusal to feed, poor oral intake, decreased urine output, and signs of dehydration. Given the recent history of upper respiratory infection symptoms, which began before the surgery, and exposure to sick individuals at home, tests for respiratory syncytial virus (RSV), flu were performed. The infant tested positive for RSV but negative for flu. Blood tests, including a complete blood count and metabolic panel, were normal except for elevated hemoglobin and hematocrit, indicating dehydration.

He was hospitalized for four days, from the 22nd to the 26th day of life. Although a recent pyloromyotomy had been performed, surgical consultation did not recommend further imaging. The poor oral intake and dehydration were attributed to the RSV infection. The infant received intravenous fluids and supportive care and was discharged home.

	Results	Normal Range
WBC	10	6-20.70 x10^3/mcl
Hb	20.4	10.3-17.6 g/dl
HCT	59.3	51.3-53.6 %
Na	137	135-145mEq/L
K	5.7	3.5-6 mEq/L
Cl	101	96-106mEq/L
HCO3	25	17-28 mEq/L
FLU	Negative	Negative
RSV	Positive	Negative

Table 2: Lab results.

On day 28 of life, the patient presented directly to the pediatric hospital with a fever of 101.4F and one episode of vomiting. The patient appeared septic with tachycardia and paleness.

Blood tests revealed an elevated white blood cell count with a left shift, suggesting infection. A urinalysis confirmed a urinary tract infection. A lumbar puncture was performed to rule out meningitis, and the infant was admitted to the hospital. Intravenous fluids and intravenous Rocephin were initiated.

By the second day of hospitalization, the infant's septic appearance, tachycardia, and pallor had improved. A urine culture identified E. coli as the causative organism. The infant was discharged home after three days on oral Bactrim, an antibiotic chosen based on sensitivity testing.

A renal ultrasound revealed enlargement of the right kidney with upper pole parenchymal edema, decreased corticomedullary differentiation, and mild hyper vascularity, consistent with pyelonephritis. A urology consultation was obtained, and a voiding cystourethrogram was performed to assess the urinary tract. The test results were normal. At discharge, the infant's weight had increased to 4.380 kilograms, surpassing his birth weight for the first time.

	Results	Normal range
WBC	23	5.5 - 22.1
Hb	18.9	10.4 - 18.0
HCT	57.7	51.3-53.6 %
Absolute neutrophil count	12.8	3.47-9.42
Urine leukocyte esterase	3+	Negative
Urine protein	100	Negative
Urine Blood	3+	Negative
Urine WBC	>5000	0-28/mcl
Urine RBC	83	0-22/mcl
Urine Bacteria	>10,000	0-9/mcl

Table 3: Lab results.

Discussion

Many factors can cause orange-coloured urine. A patient is reported with a urinary tract infection caused by Citrobacter sedlakii. This bacteria excretes indole due to the degradation of tryptophan, which causes orange urine. The patient had orange urine in the morning due to the overnight overgrowth of bacteria in the bladder [3].

Food colours such as phenolphthalein beet, medications such as rifampicin, isoniazid prochlorperazine, multivitamins, pyridium, and substances like riboflavin are known to cause orange urine [4,5].

Uric acid crystals in the urine with serum hypouricemia causing persistent orange-coloured urine were reported in a 3-year-old patient diagnosed with renal idiopathic hypouricemia caused by inactivation mutations in the SLC22A12 gene [6].

In our patient, orange-coloured diapers were the presenting symptom of IHPS. It's important to note that orange-coloured urine can also be a sign of underlying genetic disorders. A 3-month-old asymptomatic infant was presented with orange-coloured diapers as the first indication of Lesch-Nyhan disease [7].

The first case of IHPS was documented in the 18th century. IHPS primarily affects firstborn male infants, typically presenting between 2 and 8 weeks of age, with a peak incidence around 6 weeks. However, it can occur as early as 2 days or as late as 5 months of age [8]. Huang et al. found that only less than 10% of patients were diagnosed before 3 weeks of age in a study on 214 infants with IHPS, confirming that neonatal HPS is rare but does exist. 8

Our patient presented earlier than the peak age of occurrence. Early age of occurrence is presented in a 10-day-old male with atypical presentation of IHPS, with an incidental diagnosis of event ration of the left diaphragm. A contrast study showed a gastric outlet obstruction with the possibility of gastric volvulus. Emergency surgery had established the diagnosis of IHPS. The association of IHPS with diaphragmatic defect and gastric volvulus is rare, and few cases have been reported in the literature [9].

Several risk factors are known to be associated with the occurrence of IHPS. Male sex, prematurity, bottle feeding, cesarean delivery, being a firstborn, maternal smoking during pregnancy, maternal age of less than 20 years, erythromycin use in the first two weeks of life, and a family history of IHPS in siblings [10, 11].

Of the known risk factors for IHPS, only the patient's male sex correlates with our patient. The patient was full-term, the fifth child, breastfed, vaginally delivered, born to a mother over 20 years old who did not smoke, and had no history of erythromycin use or family history of IHPS.

A large number of typical and a small number of atypical presentations of IHPS have previously been reported. A retrospective case review of patients with IHPS at an Australian pediatric tertiary Center compared current findings with a previous study. The analysis confirmed the higher prevalence of IHPS in males and revealed an earlier average age of presentation at 5.4 weeks compared to the previous 6 weeks [2].

While complications such as incomplete pyloromyotomy, perforation, and wound infection can occur after surgery for IHPS, our patient did not experience any of these [12]. However, the patient required two additional hospitalizations: one for RSV bronchiolitis, likely due to exposure to sick individuals at home, and another for pyelonephritis, which is unrelated to IHPS.

Conclusion

Orange-coloured urine can be a symptom of various underlying conditions and could be related to serious genetic and surgical disorders. Treatment is directed to identify and address the root cause. While IHPS often presents with typical symptoms that are easily diagnosed and treated, atypical cases can occur even without traditional risk factors. These cases require a high level of clinical suspicion for prompt diagnosis and management to prevent complications. Our case emphasizes the importance of maintaining a high index of suspicion to ensure early diagnosis and appropriate treatment of atypical IHPS presentations.

Patient Consent: The parent gave written informed consent to publish this case report. A copy of the consent is available for review.

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Ethical Considerations: NA.

Conflict of Interest Disclosures (includes financial disclosures): No authors have any conflict of interest.

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