NU Rounds
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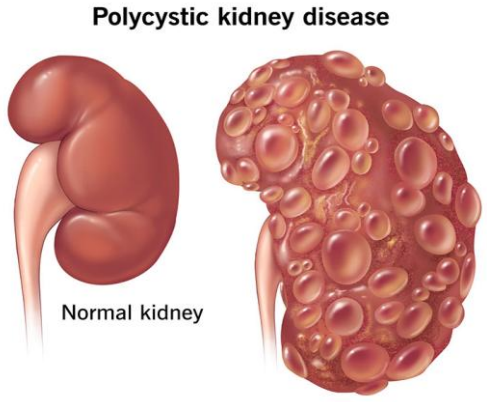
Evaluation of the gut microbiota pattern with respect to kidney function in patients with Autosomal Dominant Polycystic Kidney Disease

Introduction



The gut microbiota is a collection of symbiotic microbes in the gastrointestinal tract that play various physiological roles such as regulating the immune system and protecting against pathogens, supporting energy metabolism, and generating short-chain fatty acids (SCFAs) which have renoprotective properties.[1] Several factors affect the nature and function of the microbiota, including age, diet, medications, and disease. About 15% of adults suffer from chronic kidney disease (CKD), with autosomal dominant polycystic kidney disease (ADPKD) being the primary genetic cause. CKD causes biochemical and biophysical changes, including uremic toxin retention, altered intestinal pH, and longer colonic transit time.[2] These factors may therefore result in metabolic, immunological, and endocrine disorders as well as changes to the gut microbiota caused by the growth of proteolytic pathobionts at the expense of saccharolytic symbionts.[3] In CKD, the uremic environment promotes the growth of proteolytic bacteria that produce uremic toxins like p-cresyl sulfate and indoxyl sulfate. As kidney function declines, these toxins accumulate in the plasma, causing oxidative stress, inflammation, and fibrosis. Thus, low-protein diets containing pro-, pre-, and synbiotics (oligofructose-enriched inulin, B. infantis, and L. acidophilus) that encourage symbiont growth may reduce inflammation and slow the progression of CKD.[4] Further, there is limited research on the impact of CKD on the microbiota in Sub-Saharan Africa (SSA). In this region with high CKD prevalence and limited access to kidney replacement therapy, so nephroprotection is crucial for managing the condition. Dietary differences among SSA populations may impact gut microbiota changes compared to other regions.[5] This study examined the relationships between kidney function in individuals with autosomal dominant polycystic kidney disease (ADPKD) and representative symbionts (Bifidobacterium and Lactobacillus) and pathobionts (Enterobacteriaceae).

Methods

This cross-sectional study included 29 patients with ADPKD and 15 controls matched for age and sex (2:1 ratio). Participants aged 18-65 years agreed to participate in the study. Nephrologists diagnosed ADPKD patients using Ravine's criteria based on ultrasounds. Participants were excluded if they had diabetes, chronic diarrhea, or chronic infections (HIV,

hepatitis B or C), had undergone hemodialysis, were taking antibiotics, prebiotics, probiotics, laxatives, or NSAIDs in the previous four weeks, or did not provide all samples. Dietary intake was evaluated using food frequency questionnaire (FFQ) and a 24-hour diet recall, classifying protective or non-protective for gut microbiota. Biological samples (urine, blood, stool) and clinical data (blood pressure, urine dipstick) were collected during fasting. Blood Pressure was measured using automated blood pressure measuring device and serum creatinine was measured using kinetic Jaffé method. The estimated glomerular filtration rate (eGFR) was calculated from serum creatinine using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation from 2009. Microbiological DNA extracted from stool samples and amplified using qPCR was used to measure the abundance of Enterobacteriaceae, Bifidobacterium, and Lactobacillus. The data analysis was conducted using IBM SPSS version 26.0

Results

Among 44 participants, 29 (65.9%) had ADPKD, while 15 (34.1%) were controls. The participants' mean age (SD) was 40.65 (±11.9) years. Among ADPKD participants, 62.1% reported flank pain and 48.3% had hypertension. The median eGFR in ADPKD was 74.4 [51.2–94.6] ml/ min/1.73m^2, with 38% in stage G3-5 of CKD, while 62% were in stage G1-2. All stool samples contained Enterobacteriaceae. Lactobacillus abundance was lower in ADPKD participants with severe kidney function decline (CKD G3-5: 0.58 ng/µL) compared to those with milder damage and controls (G1-2: 0.64 ng/µL, p=0.047; controls: 0.71 ng/µL, p=0.043), while Enterobacteriaceae abundance was higher in ADPKD patients with lower kidney function (CKD G3-5: 78.6 ng/µL) compared to those in the other two groups (G1-2: 71.6 ng/µL, p=0.048; controls:70.5 ng/µL, p=0.045) (Figure 1).

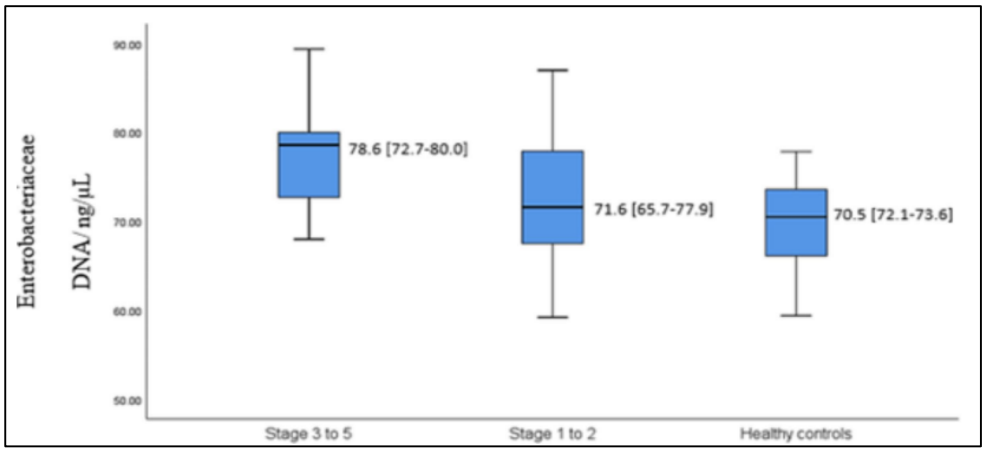


Figure 1. Comparison of Enterobacteriaceae abundance in CKD patients and healthy controls

Discussion

This study demonstrates that declining kidney function in ADPKD patients is associated with significant gut dysbiosis, characterized by an increase in Enterobacteriaceae and a decrease in Lactobacillus. These findings align with previous studies that the uremic environment of CKD promotes the growth of proteolytic bacteria, which produce uremic toxins like p-cresyl sulfate and indoxyl sulfate. These toxins contribute to increased oxidative stress and inflammation, further exacerbating CKD progression.[4] As kidney function declines, the amount of Lactobacillus in the gut decreases, indicating a possible target for therapeutic interventions like dietary changes or synbiotic supplements to improve dysbiosis. Similarly, Gryp et al. found that as kidney function declined, Lactobacillus abundance also decreased.[6]

Conclusion

In ADPKD patients, decreased kidney function was linked to decreased symbiont and increased pathobiont abundance, indicating a possible role for the microbiota in disease progression and potential targets for future research.

Gut microbiota imbalances in ADPKD may contribute to CKD progression, highlighting the potential for dietary or probiotic interventions to support kidney health.

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Role of kidney and gallbladder stones and the risk of prostate cancer

Introduction

Prostate cancer is the most common cancer among men in the Western world, posing a significant health risk. In 2022, around one million cases of prostate cancer were reported globally, with nearly 400,000 deaths.[1] Epidemiological studies suggest a potential link between prostate inflammation and prostate cancer development.[2] Although a history of urinary calculi increases the risk of urinary tract cancers, the association between urinary stones and systemic

cancers remains unclear.[3,4] Also, according to recent estimates, gallbladder stones affects 10 to 15% of the adult population and there was a significant associations between gallbladder stones and prostate cancer, but none addressed the aggressiveness of the cancer.[4] So, the study aimed to investigate the relationship between kidney or gallbladder stones and prostate cancer risk.

Methods

This study utilized data from the EPICAP (EPIdemiological Study of Prostate Cancer), a population-based case-control study that investigates the effects of environmental and genetic factors on prostate cancer. The study included 819 histologically confirmed incident prostate cancer cases and 879 controls from the general population who were matched for age and socioeconomic status. Eligible participants were men diagnosed with incident prostate cancer between 2012 and 2013, under the age of 75, and residing in the He'rault department, with all cases confirmed by histological examination. Data regarding sociodemographics, lifestyle habits (e.g. diet, tobacco, alcohol consumption, physical activity), occupational and residential history, family history of prostate cancer, and anthropometric measurements were collected. Medical information, including Gleason scores and Prostate Specific Antigen (PSA) levels, were extracted from patients' records at diagnosis and validated by the He'rault cancer registry later. Unconditional logistic regression was used to calculate odds ratios (OR) and 95% confidence intervals (95% CI) for the risk of prostate cancer associated with kidney and gallbladder stones. The statistical analysis software SAS (9.4 version) was used for all analyses.

Results

There was a significant association between kidney stones and the risk of prostate cancer (OR: 1.46 95% CI: 1.13–1.90), specifically in men with a history of kidney infections (OR: 5.43, 95% CI: 1.16–25.4). The risk remained significant for low-grade cancer (OR: 1.49, 95% CI: 1.13-1.98), but no longer significant for high-grade cancer (OR: 1.16, 95% CI: 0.75-1.80). Gallbladder stones did not significantly increase the risk of prostate cancer (OR: 1.19, 95% CI: 0.83-1.70), including both low-grade (OR: 1.14, 95% CI: 0.77-1.69) and high-grade cancers (OR: 1.43, CI: 0.83-2.45) (Figure 1). Gallbladder stones did not show a significant overall link to prostate cancer; however, in individuals with high triglyceride levels, the presence of gallbladder stones was associated with an increased risk (OR: 2.27, 95% CI: 0.99–5.28).

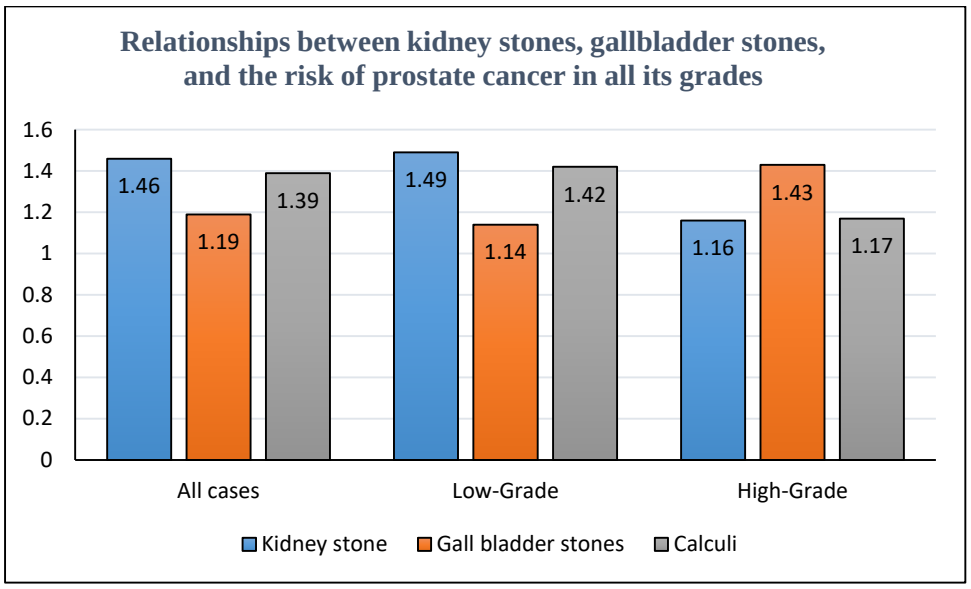

Figure 1. Association between kidney stones and gallbladder stones and the risk of prostate cancer.

Discussion

There was a significant association between the history of kidney stones and an increased risk of prostate cancer, particularly among men with a history of pyelonephritis. The observed association may be linked to chronic inflammation. Kidney stones can cause mechanical irritation or obstruct the urinary tract, resulting in urine retention and stasis. This can promote bacterial colonization and exacerbate inflammation. Chronic inflammation can cause DNA damage, cellular proliferation, and lead to tumor progression.[4] This study findings align with two prior studies suggesting a potential association between kidney stones and prostate cancer risk.[5] In contrast, studies from Sweden found no significant association between kidney stones and prostate cancer risk, possibly due to shared metabolic and lifestyle factors.[4,6]

Conclusion

There was a relationship between kidney stones and prostate cancer, particularly in men with a history of pyelonephritis and kidney infections. This highlights the potential role of chronic inflammation in prostate carcinogenesis. Also, the intricate nature of these relationships was brought to light by the detailed examination of gallbladder stones, especially regarding elevated triglyceride status.

The history of kidney stones is associated with an increased risk of prostate cancer, particularly in men with prior kidney infections, suggesting a link to chronic inflammation.

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Clinical outcome of prostate-pelvic syndrome theory in diagnosing the adult patients with type-III prostatitis

Introduction

Type-III prostatitis, also known as chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), is the most common prostate disease among adult males under 40 years old. It has a significant negative impact on patients' quality of life.[1] The pathogenesis of type-III prostatitis is not fully understood, and the diagnostic and treatment plan based on the NIH classification system published in 1995 has left doctors and patients dissatisfied. Shoskes et al. proposed the UPOINT classification method in 2009, which improved the effectiveness of diagnosing type-III prostatitis. However, the diagnostic process remains complex. UPOINT still requires tests for expressed prostatic secretion (EPS) and lower urinary tract pathogen localization (LUTPL), which have limited clinical significance. Research indicates no significant correlation between the severity of type-III prostatitis symptoms and leukocyte and bacterial counts in EPS.[2,3] Therefore, the Chinese urological experts proposed the new theory of “prostate-pelvic syndrome (PPS)” and recommended to using it to replace the term of type-III prostatitis.[4] The PPS theory offers advantages such as eliminating LUTPL and invasive EPS tests, diagnosing and treating prostate diseases through non-invasive tests, and providing personalized treatment. It has potential for high clinical guidance and application in urology and male outpatient work. However, practical application results are rare.[5] This study aimed to validate the PPS theory for diagnosing type-III prostatitis in adults under 40 years old. It also analyzed the factors associated with the main symptoms of PPS in young adult males, providing recommendations for prevention and treatment.

Methods

This retrospective study analysed the Medical records of 548 adult outpatients with type III prostatitis under the age of 40. Patients were diagnosed retrospectively using PPS diagnostic criteria in this cohort study. The study's selection criteria included adult type-III prostatitis patients under 40 years old with ultrasonography records of their urinary system and prostate. The exclusion criteria used in this study were as follows: (a) Lack of ultrasound for the urinary system and prostate; (b) Urinary infections, tuberculosis, tumors, and trauma; (c) Abnormal prostatic nodules detected by ultrasound; (d) Urinary calculus, bladder diseases, urethral stricture, chronic epididymitis, seminal vesiculitis, varicocele, diabetes, neurogenic bladder, mental illness, etc. affecting urination function and pelvic pain; (e) malignant (h) Addicts of alcohol or drugs; (i) Patients undergoing pelvic or prostate. Clinical data of the patients including age, medical history, medication history, clinical characteristics, routine urine and results of urological ultrasound examinations including the prostate were collected. A telephone follow-up was conducted to supplement psychological symptoms, educational levels, and economic income. Statistical analyses were performed using SPSS 22.0 software.

Results

Among the 548 patients, 229 experienced lower urinary tract symptoms, 159 experienced pelvic pain, and 160 experienced both lower urinary tract and pelvic pain. These patients were classified as having voiding symptoms (VS), pain symptoms (PS), and voiding + pain symptoms (VS+PS) in accordance with the PPS concept. The three primary PPS symptom groups differed significantly in PV and disease duration. The PV in the VS group was higher than that in the PS group. Spearman correlation analysis revealed a positive correlation between VS, PV, and disease duration. However, four secondary symptoms (sexual dysfunction, psychosocial symptoms, reproductive dysfunction, and others) were not related to PV. The PV ≥20 mL group had a higher proportion of VS patients compared to the PV <20 mL group. A multivariate logistic analysis revealed that PV and disease duration were independent predictors of VS in adult PPS patients under 40 years old (Figure 1).

Variables	Odds ratio (95% CI)	*P* value	Forest Plot
Constant	0.668	0.008	
Sedentariness	0.490 (0.325-0.740)	0.001	
Psychological symptoms	0.048 (0.019-0.127)	0.000	
PV≥20 ml	5.348 (2.755-10.382)	0.000	
Disease duration (month)	1.038 (1.017-1.061)	0.000	

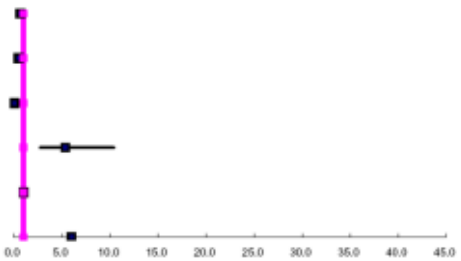

Figure 1. Multivariate logistic regression analysis of VS-related factors including PV ≥20 ml in PPS patients (Forward Stepwise).

Discussion

The study highlights prostate pelvic syndrome as a diagnostic criterion for type-III prostitis in adult males under the age of 40. There is a significant association between prostate volume and voiding symptoms, with PPS patients with PV ≥20 mL having a 5.348 times higher risk of VS than those with PV <20 Ml.[5] Benign prostatic obstruction (BPD) may be caused by enlarged prostate glands (PVs), similar to prostate obstruction symptoms in males over 40. However, the pathogenesis of BPD in adult patients under 40 may differ from males over 40 and may be linked to chronic prostate inflammation, leading to congestion and edema. BPH and chronic inflammation can exacerbate the increase in PV.[6]

Conclusion

The study found that type III prostatitis in adult males under 40 years old can be diagnosed using PPS theory. PV and disease duration were independent predictors of VS in adult PPS patients under 40. Also, this study noted that the PPS theory simplified the diagnosis protocol for type-III prostatitis in adult males under 40 years old, making it suitable for clinical use.

Prostate-pelvic syndrome theory offers a more practical and efficient approach to diagnose and manage type-III prostatitis in men under 40 years.

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Case report on prostate abscess misdiagnosed as benign prostatic hyperplasia

Introduction

Benign prostatic hyperplasia (BPH) is a common condition that affects elderly men. Symptoms include difficulty urinating, frequent urination, urgency, and nocturia. As men age increases, prostate tissue growth puts pressure on the urethra, causing low urine flow and, in severe cases, urinary retention.[1] Studies shown a significant increase in the prevalence of BPH among men over 50, with rates reaching up to 90% in those over 70 years.[2] Prostate abscess is a rare complication caused by prostatitis or infection. Symptoms include acute urinary difficulty, retention, fever, and general discomfort.[3,4] Prostatic abscess is often misdiagnosed as benign prostatic hyperplasia in clinical settings due to its symptom overlap with prostate hyperplasia, which delays treatment. Elderly men with diabetes are at a higher risk of developing prostatic abscesses, even without obvious infection symptoms. Therefore, increased awareness of the possible presence of a prostatic abscess is necessary when evaluating lower urinary tract symptoms in older males, especially those with a history of diabetes. This case provides valuable insights for diagnosing prostatic abscess in clinical practice.[4]

Case presentation

A 72-year-old male patient visited the hospital with a one-week history of difficulty urinating along with symptoms including incomplete dripping and increased frequency. The patient denied having lower back pain, chills, fever, weight loss, rash, fatigue, or dry mouth. He has type 2 diabetes for 5 years and currently takes metformin hydrochloride sustained-release tablet 1g BID and glipizide controlled-release tablet 10mg QD orally. His fasting blood glucose level was 8.43 mmol/L at admission. His physical examination showed no abnormalities in the cardiovascular, respiratory, or abdominal systems, and a digital rectal examination revealed a tight anal sphincter, smooth rectal wall, soft prostate, and shallow central sulci. Auxiliary examination revealed postoperative hematologic inflammation indicators, including procalcitonin at 0.080 ng/ml and a prostate tumor marker at 1.66 μg/L. Urinalysis revealed glucose at 1+, occult blood at 3+, protein at +−, and leukocytes at 2+. Quantitative urine sediment analysis revealed urine red blood cells at 115/μl, urine white blood cells at 118/μl, and urine white blood cell clumps at 7/μl. The bladder has a residual urine volume of approximately 655 ml. Urinary system ultrasound revealed prostate hyperplasia with calcification (size: 5.0 × 3.6 × 3.8 cm). A CT scan confirmed prostate hyperplasia (Figure 1) and an MRI is recommended if necessary. The patient had acute urinary retention and underwent emergency room catheterization. He was treated with oral hypoglycemic therapy and monitored for peripheral blood glucose levels. To treat urinary retention caused by benign prostatic hyperplasia, the patient underwent transurethral plasmakinetic resection of the prostate and transurethral incision of the prostatic abscess under spinal-epidural anesthesia. During the procedure, multiple abscess cavities were found within the prostate, with abundant

purulent fluid. After meticulously incising the abscess cavities, the prostatic abscess was diagnosed and treated with 'Meropenem' for better infection control. The patient's vital signs remained stable during and after surgery, and empirical anti-infective therapy continued postoperatively. After 7 days of postoperative anti-infective therapy, the patient's urinary catheter was removed, allowing them to urinate freely. It was recommended to continue oral antibiotic therapy for 1 month after discharge. The postoperative pathology revealed prostatic hyperplasia and no bacterial growth in the pus culture obtained during surgery.

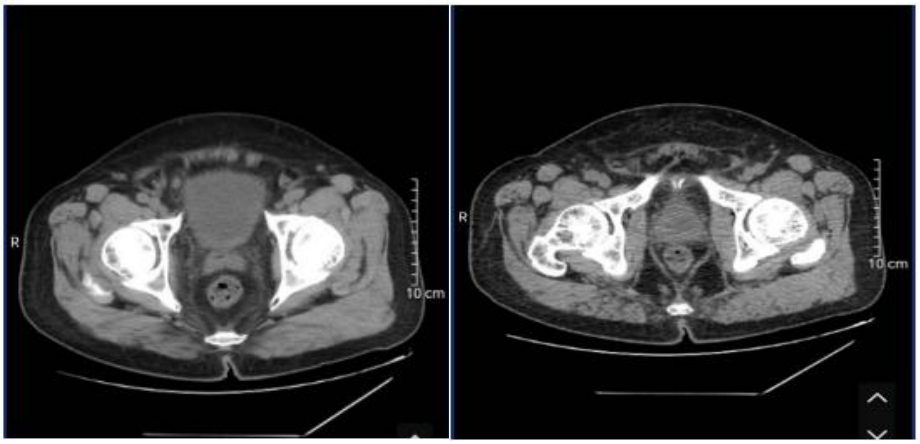

Figure 1. CT scan results indicating prostate hyperplasia.

Discussion

This case highlights the diagnostic challenges of prostatic abscess, particularly on elderly diabetic patients, due to its symptom overlap with benign prostatic hyperplasia. Although no fever or tenderness symptoms were present at admission, the elevated white blood cell count and C-reactive protein levels in blood routine analysis suggest that a urinary tract infection may not be the sole cause of these findings. Clinicians should maintain a high level of suspicion and investigate multiple potential sources of infection to ensure accurate diagnosis.[5] Prostate MRI and transrectal ultrasound examinations can detect abscesses early, allowing for timely intervention. Prostate abscesses can be effectively managed if identified early.[6] Effective treatment of prostatic abscess involves anti-infective therapy and incision drainage. The patient experienced significant improvement in urinary symptoms after surgery, demonstrating the effectiveness of this treatment strategy.[7]

Conclusion

This case emphasizes the importance of accurate diagnosis of prostatic abscess, particularly in elderly male diabetic patients with lower urinary tract symptoms. Early detection through appropriate imaging, prompt anti-infective therapy and timely surgical intervention can

improve patient outcomes and reduce the risk of severe complications after a prostate abscess diagnosis.

Early recognition of prostatic abscess is crucial to prevent misdiagnosis and avoid complications.

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