Case Report

DOI: 10.7860/JCDR/2025/75782.20839

Navigating the Complexities: A Case Report on Diagnostic and Management Challenges in Transverse Colon Cancer

Surgery Section

VAIJAYANTHI SARAVANAN[1], VINOJ GOPALAKRISHNAN[2], MARIA INFANT MAJULA SHIFANI MAHENDRAN[3], RAJAN VAITHIANATHAN[4]

ABSTRACT

The transverse colon is the least commonly impacted segment in Colorectal Cancer (CRC), contributing to only a fraction of cases. Its rarity can result in less clinical familiarity and significant diagnostic delays as compared to other CRC sites, such as the rectum or sigmoid colon. Because the transverse colon surrounds the upper abdomen, symptoms may overlap with those of other abdominal organs (for example, the stomach, liver, or pancreas). This frequently results in misdiagnosis or delayed recognition. Patients may present with non specific symptoms such as non specific abdominal pain, bloating, or changes in bowel habits, which are difficult to accurately attribute to cancer in this region. The mobility and location of the transverse colon might make visualisation difficult during colonoscopy, perhaps leading to undetected lesions. Hereby, we present a case study of a 70-year-old male with severe lower abdominal pain, intermittent watery stools, rectal bleeding, and significant weight loss. Examination identified an 8×5 cm palpable mass in the epigastric region extending into both hypochondria. Histopathological analysis revealed colonic mucosal ulceration, chronic inflammation, haemorrhage, and an infiltrating malignant tumour characterised by pleomorphic cells in sheets, nests, and glandular patterns. Magnetic Resonance Imaging (MRI) evidenced the diffuse circumferential wall thickening of the transverse colon, indicative of malignancy. The present case provides valuable insights and enhances the practice of early diagnostic procedures for detecting transverse CRC. By addressing the unique challenges of this rare condition, it deepens diagnostic precision and underscores the importance of timely intervention.

Keywords: Colonoscopy findings, Metastatic, Palliative care, Transverse colorectal cancer

CASE REPORT

A 70-year-old male with a history of hypertension presented to the Outpatient Department with severe lower abdominal pain. The pain was intermittent, mild to moderate in intensity, and non radiating, with no identifiable aggravating or relieving factors. The patient had undergone a haemorrhoidectomy 20 years ago but did not return for follow-ups. There were no records of prior health screenings for CRC or other malignancies. Additionally, no documented family history of CRC or other Gastrointestinal (GI) cancers was available, leaving a gap in assessing genetic predisposition.

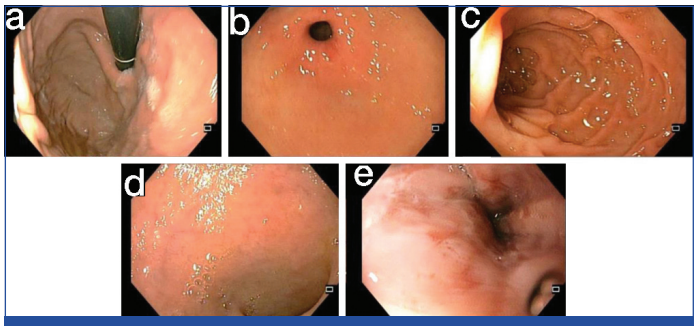
Three months ago, the patient began experiencing intermittent loose, watery stools without any blood or vomiting. Two months ago, the symptoms worsened to include rectal bleeding and significant weight loss. One month ago, vomiting episodes started, which consisted of food particles but were non bilious and non blood-stained. Alongside these symptoms, the patient reported a loss of appetite and melena. On physical examination, a palpable mass measuring 8×5 cm was identified in the epigastric region, extending into both hypochondria. The abdomen showed no tenderness, rigidity, or hepatomegaly. The patient exhibited pallor and bilateral pitting oedema.

Over the examination of rectum, haemorrhoids were noted at the 5o'clock position with normal sphincter tone and an empty rectum. Further, proctoscopy was performed, internal haemorrhoids were observed from the 10 to 11 o'clock position. Initial laboratory tests showed normal parameters except for an elevated glucose level of 170 mg/dL and severe anemia with abnormal Red Blood Cells (RBC) indices. The Complete Blood Count (CBC) results are largely within normal limits, indicating no major abnormalities. Echocardiography indicated mild concentric left ventricular hypertrophy, diastolic dysfunction, trivial mitral and tricuspid regurgitation, and a sclerotic aortic valve, but no thrombus or effusion. Urinalysis revealed mild pyuria, occasional crystals, trace protein, bilirubin, and blood, suggesting potential urinary tract infection or kidney issues [Supplementary Table/Fig-S1-S5].

In response to bleeding per rectum, stool occult blood test was performed for three consecutive days from the day of admission. The test results for three consecutive days were observed to be negative and no signs of blood in the stool was observed.

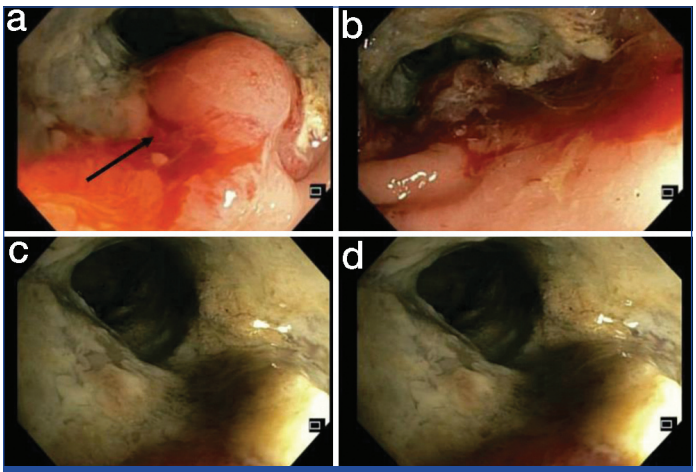
An abdominal ultrasound showed no abnormalities, with normal liver, kidney, and bladder function.

The upper GI endoscopy was performed to rule out irritable bowel disorder. The oesophagus appears normal, with the Oesophago-Gastric (OG) junction located at 40 cm. The stomach, including the fundus, body, antrum, and pylorus, is normal. The duodenum, specifically D1 and D2, is also normal. The impression of the endoscopy indicates the lower oesophageal erosion and no signs of irritable bowel disorder. The mild erosion could be resulted from frequent vomiting [Table/Fig-1].



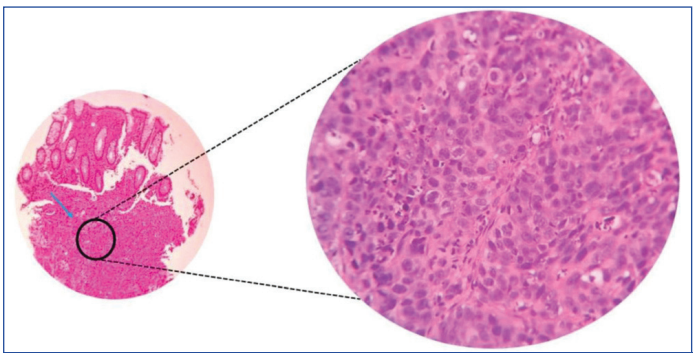
[Table/Fig-1]: (a) Fundus appeared normal. Appears normal with no internal bleeding, erosion or ulceration; (b) Antropyloric norma appears normal with no internal bleeding or erosion; (c) Lower end oesophageal erosion mild erosion of the lower oesophagus has been observed with inflammation; (d) D1 Normal. Duodenum 1 appears normal with no ulceration and erosion; (e) D2 Normal. Duodenum 2 appears normal with no ulceration and erosion.

Further, the patient was investigated for the presence of small mass through colonoscopy. The colonoscopy revealed the presence of an ulcer with distal fleshy growth at the distal end of the transverse colon, 100 cm from the anal verge, along with mucosal sloughing and narrowing of the lumen, resulting in decreased distensibility of the colon, preventing the scope from passing proximally. A biopsy was taken from the fleshy growth 100 cm from the anal verge. The anal canal, rectum, and sigmoid colon are normal. The descending colon and splenic flexure showed no abnormalities. The hepatic flexure, ascending colon, and cecum were not entered. The colonoscopy left the impression of a possible carcinoma of the transverse colon [Table/Fig-2].



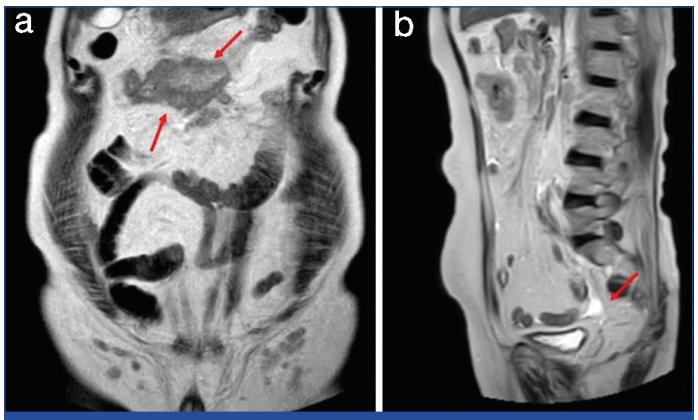
[Table/Fig-2]: a,b) Distal end of the transverse colon, 100 cm from the anal verge, an ulcer with distal fleshy growth was noted (black arrow); c,d) Extensive mucosal sloughing. Mucosal sloughing and narrowing of the lumen, resulting in decreased distensibility of the colon, preventing the scope from passing proximally.

The biopsy was subjected to histopathological analysis to check for carcinoma of transverse colon. Sections studied show multiple fragments of colonic mucosa with ulceration. Sub-epithelium shows mucosal crypts with chronic inflammation and haemorrhage. The fragment shows infiltrating malignant tumour arranged in sheets, nests, and focal glandular pattern. The tumour cells are round to oval with moderate pleomorphic coarse chromatic nucleus, conspicuous nucleoli, and moderate cytoplasm [Table/Fig-3]. Intervening stroma shows desmoplastic reaction. There is no discernible rise of the tumour marker when the Carcinoembryonic Antigen (CEA) level is 0.50 ng/mL.



[Table/Fig-3]: Poorly differentiated adenocarcinoma. The tumour cells are round to oval with moderately pleomorphic, coarsely chromatic nuclei, conspicuous nucleoli, and moderate amounts of cytoplasm. There are 2-3 mitotic figures per high power field. The intervening stroma exhibits a desmoplastic reaction {Haematoxylin and Eosin (H&E), 40x}.

For further confirmation of cancer and to check tumour metastases, MRI was performed. An MRI scan was performed to assess the extent of the disease and to confirm the diagnosis. The imaging revealed diffuse circumferential wall thickening of the transverse colon, a characteristic feature of malignancy [Table/Fig-4]. The thickening was consistent with the presence of an advanced tumour obstructing the normal tissue architecture. Additionally, MRI imaging showed lymph node metastasis, indicating that the cancer had spread beyond the primary site to the surrounding lymphatic system. Mild ascites was also noted, suggesting that the cancer had led to some fluid accumulation in the abdominal cavity, which is commonly associated with advanced-stage cancer [Table/Fig-4]. These findings confirmed the diagnosis of advanced colorectal carcinoma and indicated that the disease had progressed beyond its localised stage.



[Table/Fig-4]: (a) The arrow indicates T2 coronal section showing circumferential wall thickening of the transverse colon, of 11 cm in length, with a maximum thickness of 1.7 cm (from mucosa to serosa), causing luminal narrowing; (b) The arrow indicates T2 sagittal section with mild ascites.

From the investigations it was declared that the patient was experiencing advance stage of cancer. The patient was supported with palliative care during the course of stay in the hospital. The patient was supported with nursing care. Pantaprazole 40 mg PO BD were administered to manage GI concerns and inflammation, crucial for overall patient comfort and recovery. Injection- Emeset 4 mg i.v. TID was used to control nausea. In patient care, a balanced diet plan tailored to specific nutritional requirements is essential for optimal health outcomes. For the patient, 2400 kcal per day, 1 g of protein per kg of body weight, and limited to 15.20 g of fat, while adhering to a salt-restricted diet, careful consideration of food choices and portion sizes had been made crucial as a part of patient care.

However, due to the low socio-economic condition, the patient denied further surgical opinion. In concern of the patient's opinion, he has been discharged against medical advice.

DISCUSSION

Transverse colon cancer is believed to account for 10% of all colon cancers, while estimates for right-sided CRC range from 54% to 67% [1]. Aging, smoking, intestinal inflammatory illness, intestinal polyps, poor eating habits, and genetics all increase the chance to acquire CRC [2]. In a review by Granados-Romero JJ et al., a majority of patients with CRC are over fifty, with a median age of 64 years [3].

The CRC is caused by a complex interaction of genetic abnormalities that affect essential cellular mechanisms controlling proliferation, apoptosis, and Deoxyribonucleic Acid (DNA) repair. Adenomatous Polyposis Coli (APC), Kirsten Rat Sarcoma (KRAS), and Tumour Protein 53 (TP53) gene mutations are important genetic abnormalities that cause unchecked cell proliferation, resistance to apoptosis, and lack of cell cycle regulation [3].

The mutation of the APC gene, which causes dysregulation of the Wnt signaling pathway and excessive cell proliferation [4]. This is frequently followed by KRAS activating mutations, which promote persistent Mitogen-activated Protein Kinase (MAPK) signaling, and TP53 loss-of-function mutations, which decrease DNA damage response and tumour suppression [5]. Furthermore, Phosphatidylinositol-4,5-bisphosphate 3-Kinase Catalytic subunit alpha (PIK3CA) mutations activate the PI3K-AKT pathway, which promotes tumour survival and angiogenesis [6]. Defects in Mismatch Repair (MMR) genes such as MutL homolog 1 (MHL1),

MutS homolog 2 (MSH2), MutS homolog 6 (MSH6) and PMS1 homolog 2 (PMS2) cause Microsatellite Instability (MSI), which is a characteristic of Lynch syndrome and a biomarker for immunotherapy responsiveness [7]. Inactivation of SMAD4 alters the Transforming Growth factor-β (TGF-β) pathway, which generally leads to severe disease and a bad prognosis [8]. Together, these mutations illustrate CRC's complex genetic landscape, providing insights for precision diagnostics and tailored treatment interventions.

Histone modifications and other epigenetic changes, such as DNA methylation, further encourage the development of cancer [9]. Through DNA damage, chronic inflammation which is frequently caused by illnesses like inflammatory bowel disease, creates an environment that is conducive to cancer. Transverse CRC generally, remains asymptomatic as it is bagged with multiple complicated interventions. The anatomical location of transverse colon and variety of symptom, makes diagnosis complicated. Making the distinction between primary and metastatic CRC can be difficult since colorectal metastases from primary colon cancer is uncommon [10].

Imaging techniques are critical for distinguishing between primary and metastatic CRC, especially in difficult instances like transverse CRC, where the tumour's anatomical position complicates diagnosis [11]. Advanced imaging techniques including Computed Tomography (CT), MRI, and Positron Emission Tomography (PET) are critical for determining tumour size, location, and involvement of nearby structures [12]. PET-CT, which combines metabolic and anatomical imaging, is very successful at detecting metastatic lesions that may not be seen on traditional imaging, thereby distinguishing primary illness from metastatic metastasis [13].

In general, staging at the time of diagnosis predicts the survival rates for colon cancer. Uncertainty in the diagnosis can arise from non-specific GI symptoms that are prevalent in advanced colon cancer. Because colon cancer is less common than other benign GI conditions, additional possible causes may be investigated before colon cancer, delaying detection [14].

Consequent obstruction of stool or difficulty in defaecation, accompanied by positive stool occult blood test and higher CEA level are prominent signs of CRC [15,16]. However, in present case study, the patient reports showed negative for the occult blood test, normal CEA level, and presented with loose watery stool, which made diagnosis difficult. Though transverse CRC is reported to be rare, there are incidents reporting the spontaneous regression of tumour after surgical resection [17]. Chida K et al., presented the spontaneous regression of transverse CRC in an 80-year-old man [16]. Patient presented with CRC mostly likely treated with surgical resection of the tumour and supported by chemo and radiation therapy. Approximately 10% of tumours are typically adherent to adjacent organs. Anand Munghate et al., reported the local invasion of inflammation of transverse colon to biliary organs like gallbladder [18].

The majority of colorectal malignancies develop from benign polyps to invasive carcinoma by following the adenoma-carcinoma sequence [18]. Due to the previous medical history was presented with haemorrhoids (20 years back), there is a possibility to develop polyp in the suspected region, which might be hidden under diagnosis. The patient's delay in seeking care was influenced by several socioeconomic barriers. Financial constraints, due to limited resources, hindered timely access to healthcare, preventing the patient from seeking early medical attention. Additionally, the patient's rural location further restricted access to diagnostic facilities, complicating the ability to obtain necessary tests and consultations. Low awareness of the early symptoms of CRC contributed to the delay in medical consultation, as the patient did not recognise the severity of the symptoms until they worsened.

Transverse CRC offers distinct diagnostic and therapeutic issues due to its anatomical position, which frequently results in delayed discovery and poor outcomes. This problem becomes more severe in resource-constrained regions by a lack of extensive screening programs and little understanding of CRC risk factors and symptoms. Addressing these obstacles necessitates targeted, actionable solutions. First, extending CRC screening programs using low-cost, scalable technologies like Faecal Immunochemical Testing (FIT) and stool DNA tests can greatly improve early detection rates. These non invasive tools are inexpensive, do not require advanced infrastructure, and are suitable for large-scale deployment. Community-based programs, such as mobile screening units and home-testing kits, can broaden access to underserved communities. Second, resource-constrained settings should prioritise training primary healthcare professionals to recognise early symptoms of CRC and incorporating basic diagnostic procedures, such as colonoscopy or sigmoidoscopy, into current healthcare systems.

CONCLUSION(S)

Effective care of transverse CRC requires a multidisciplinary approach. A multidisciplinary team of gastroenterologists, oncologists, radiologists, surgeons, and palliative care specialists provides comprehensive care, including correct staging, individualised treatment, and supportive treatments. This collaborative methodology enhances decision-making and outcomes, especially in anatomically complex instances such as transverse CRC.

REFERENCES

[1] Cann C, Dotan E. Transverse colon cancer: A call for focused research in an understudied heterogenous disease. J Gastrointest Oncol. 2024;15(4):1981-86.
[2] Kay J, Thadhani E, Samson L, Engelward B. Inflammation-induced DNA damage, mutations and cancer. DNA Repair. 2019;83:102673.
[3] Granados-Romero JJ, Valderrama-Treviño AI, Contreras-Flores EH, Barrera-Mera B, Herrera Enríquez M, Uriarte-Ruíz K, et al. Colorectal cancer: A review. Int J Res Med Sci. 2017;5(11):4667.
[4] Hong SN. Genetic and epigenetic alterations of colorectal cancer. Intest Res. 2018;16(3):327-37.
[5] Boman BM, Fields JZ. An APC:WNT counter-current-like mechanism regulates cell division along the human colonic crypt axis: A mechanism that explains how APC mutations induce proliferative abnormalities that drive colon cancer development. Front Oncol. 2013;3:244.
[6] Tang Y, Fan Y. Combined KRAS and TP53 mutation in patients with colorectal cancer enhance chemoresistance to promote postoperative recurrence and metastasis. BMC Cancer. 2024;24(1):1155.
[7] Wang Y, Rozen V, Zhao Y, Wang Z. Oncogenic activation of PIK3CA in cancers: Emerging targeted therapies in precision oncology. Genes Dis. 2025;12(2):101430.
[8] Zhao P, Li L, Jiang X, Li Q. Mismatch repair deficiency/microsatellite instability-high as a predictor for anti-PD-1/PD-L1 immunotherapy efficacy. J Hematol Oncol J Hematol Oncol. 2019;12(1):54.
[9] Shan H, Tian G, Zhang Y, Qiu Z. Exploring the molecular mechanisms and therapeutic potential of SMAD4 in colorectal cancer. Cancer Biol Ther. 2024;25(1):2392341.
[10] Audia JE, Campbell RM. Histone modifications and cancer. Cold Spring Harb Perspect Biol. 2016;8(4):a019521.
[11] Kojima S, Sakamoto T, Nagai Y, Honda M, Ogawa F. Metachronous rectal metastasis from primary transverse colon cancer: A case report. Surg Case Rep. 2018;4:90.
[12] García-Figueiras R, Baleato-González S, Padhani AR, Marhuenda A, Luna A, Alcalá L, et al. Advanced imaging of colorectal cancer: From anatomy to molecular imaging. Insights Imaging. 2016;7(3):285-309.
[13] Islam S, Walker RC. Advanced imaging (positron emission tomography and magnetic resonance imaging) and image-guided biopsy in initial staging and monitoring of therapy of lung cancer. Cancer J Sudbury Mass. 2013;19(3):208-16.
[14] Molnar O, Straciuc OM, Mihuțiu S, Lazăr L. Impact of PET/CT imaging with FDG in locally advanced cervical carcinoma—a literature review. Curr Oncol. 2024;31(5):2508-26.
[15] Mueller E, Shaik Z, Addepalli D, Malik S, Schiefelbein P. Obstructing Stage IV adenocarcinoma of the transverse colon in a young patient with vitiligo. Cureus. 2023;15(7):e42679.
[16] Chida K, Nakanishi K, Shomura H, Homma S, Hattori A, Kazui K, et al. Spontaneous regression of transverse colon cancer: A case report. Surg Case Rep. 2017;3(1):65.
[17] Li L, Xing S, Wu M, Ao Y, Zheng X, Cai R, et al. Fecal CEA has an advantage in the diagnosis of colorectal cancer at early stage. Cancer Control J Moffitt Cancer Cent. 2021;28:10732748211048292.
[18] Munghate A, Kumar A, Singh H, Singh G, Singh B, Chauhan M. Carcinoma transverse colon masquerading as carcinoma gall bladder. J Gastrointest Oncol. 2014;5(2):E40-E42.

PARTICULARS OF CONTRIBUTORS:
1. Ph.D. Scholar, MGM Advanced Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India.
2. Assistant Professor, MGM Advanced Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India.
3. Ph.D. Scholar, MGM Advanced Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India.
4. Professor and Head, Department of Surgery, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Vinoj Gopalakrishnan,
Assistant Professor, MGM Advanced Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry-607402, India.
E-mail: vinojvino@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 26, 2024
- Manual Googling: Jan 02, 2025
- iThenticate Software: Jan 04, 2025 (5%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Sep 23, 2024**
Date of Peer Review: **Dec 09, 2024**
Date of Acceptance: **Jan 06, 2025**
Date of Publishing: **Apr 01, 2025**

SUPPLEMENTARY TABLES

Test	Result	Normal range
Urea (Blood)	21 mg/dL	17.1-49.2 mg/dL
Creatinine	0.81 mg/dL	0.8-1.3 mg/dL
Sodium	138 mEq/L	136-143 mEq/L
Potassium	4.2 mEq/L	3.5-5.1 mEq/L
Chloride	106 mEq/L	102-108 mEq/L
Total protein	7.0 g/dL	6.4-8.3 g/dL
Albumin	3.5 g/dL	3.2-4.6 g/dL
Globulin	3.5 g/dL	2.3-3.6 g/dL
Albumin/Globulin (A/G) ratio	1.0:1	-
Bilirubin total	0.6 mg/dL	0.2-1 mg/dL
Bilirubin direct	0.3 mg/dL	0.1-0.3 mg/dL
Bilirubin indirect	0.3 mg/dL	0.2-0.6 mg/dL

[Table/Fig-1]: Initial investigations.

Test	Result	Normal range
SGOT (AST)	19 U/L	<35 U/L
SGPT (ALT)	10 U/L	<45 U/L
Alkaline phosphatase	75 U/L	56-119 U/L

[Table/Fig-2]: Liver function tests.
SGOT (AST): Serum glutamic-oxaloacetic transaminase (amino tranferase); SGPT (ALT): Serum glutamic pyruvic transaminase (alkaline transaminase)

Parameter	Result	Normal range
Epithelial cells	2-4	<5
Pus cells	5-6	<5
Casts	NIL	NIL
Crystals	Occasional	NIL
Specific gravity	1.030	1.003-1.035
Color (Urine)	Straw yellow	Straw yellow
Appearance	Cloudy	Clear
Urine pH	6.0	4.6-8
Urine protein (Albumin)	Trace	Negative
Urine glucose	Nil	Nil
Urine ketone	Negative	Negative
Bilirubin (Urine)	1+	Negative
Urobilinogen (Urine)	Nil	Nil
Nitrite	Nil	Nil
Blood (Urine)	1+	Negative
Leukocyte	Nil	Nil

[Table/Fig-3]: Urinalysis.

Parameter	Result	Normal range
Total WBC count	9600 cells/cumm	4000-10000 cells/cumm
Neutrophils	63.6%	40-80%
Lymphocytes	24.0%	20-40%
Eosinophils	1.2%	1-6%
Basophils	0.5%	<2%
Monocytes	10.7%	2-10%
Band cells	0%	<5%

[Table/Fig-4]: Complete Blood Count (CBC).

Parameter	Result	Normal range	Remark
Total RBC count	4.44 million/cumm	4.5-5.5 million/cumm	Slightly low
PCV (Hematocrit)	27.1%	40-50%	Low
MCV	61.0 fL	83-101 fL	Low
MCH	19.0 pg	27-32 pg	Low
MCHC	31.1%	31.5-34.5%	Low
Platelet count	279,000/cumm	150,000-450,000/cumm	Normal
Haemoglobin (Hb)	8.4 g/dL	13-17 g/dL	Low
RDW-CV	17.9%	11.6-14%	High

[Table/Fig-5]: Red Blood Cell (RBC) indices.
MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; RDW-CV: Red cell distribution width coefficient of variation