

Scientific Letters**Severe Adult Protein-Energy Malnutrition Mimicking Haematological Malignancy: Diagnostic Lessons from severe Cytopenias****Keywords:** Kwashiorkor; Parvovirus B19; Pancytopenia.**Published:** November 01, 2026**Received:** May 31, 2026**Accepted:** October 10, 2026**Citation:** Seraj L., Bruci A., Voci A., Derraj X., Serani K. Severe adult protein-energy malnutrition mimicking haematological malignancy: diagnostic lessons from severe cytopenias. *Mediterr J Hematol Infect Dis* 2026, 18(1): e2026079, DOI: <https://dx.doi.org/10.4084/MJHID.2026.079>

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

To the Editor.

Severe cytopenias with hepatosplenomegaly, polyserositis, coagulation abnormalities and markedly elevated lactate dehydrogenase (LDH) reasonably prompted urgent investigation for a primary haematological disorder. We report a 53-year-old man with severe dietary restriction during a major depressive episode who developed progressive cytopenias, more than 40 kg weight loss, oedema and multisystem abnormalities. He arrived with an outside private-laboratory complete blood count reporting haemoglobin 30 g/L, white cells $1.48 \times 10^9/\text{L}$ and platelets $14 \times 10^9/\text{L}$. Review of that sample showed platelet clumping, making the automated platelet count unreliable. Repeat in-house testing showed platelets in the normal range ($175\text{--}179 \times 10^9/\text{L}$) before any platelet transfusion, while severe anaemia and leukopenia persisted. The outside platelet value was therefore considered spurious pseudothrombocytopenia and was not used for longitudinal interpretation. The overall phenotype was ultimately most consistent with severe oedematous protein-energy malnutrition, with a kwashiorkor phenotype, but this was not histologically proven. The case is educational because the presentation strongly mimicked haematological malignancy. At the same time, several striking laboratory findings, including LDH/CRP discordance and low-level parvovirus B19 DNA, were non-specific when interpreted against the longitudinal course.

The patient had long-standing insulin-treated diabetes mellitus. A complete blood count approximately one month before admission had already shown cytopenias, but he declined further assessment and treatment; during the following weeks his intake remained markedly reduced and his condition deteriorated. On admission, measured weight was 59 kg at 188 cm (body mass index 16.7 kg/m^2), although oedema and polyserositis inflated the measured weight; estimated dry weight was 47–49 kg. Examination

showed marked muscle wasting, bilateral pitting oedema, jaundice and sparse depigmented scalp hair. There was no diarrhoea, steatorrhoea or other clinical feature suggesting malabsorption. Cross-sectional imaging showed hepatomegaly (18 cm), splenomegaly (17 cm), bilateral pleural effusions, pericardial fluid and ascites, without pathological lymphadenopathy or cirrhotic morphology.

Peripheral blood examination showed no circulating blasts, dysplasia, atypical or immature leucocytes, significant poikilocytosis or other specific red-cell abnormalities suggesting an acute primary marrow disorder. Reticulocytes were 3%, with a corrected reticulocyte index of approximately 0.6% and a reticulocyte production index of approximately 0.3. The earliest retrievable complete red-cell index panel, obtained after initial stabilisation, showed a haematocrit of 11.8%, MCV 90.5 fL, MCH 29.5 pg, MCHC 32.6 g/dL, and RDW 35.2%. Acute leukaemia was therefore less likely based on peripheral blood, but not conclusively excluded. We deferred bone marrow examination during the clinically unstable phase because significant coagulopathy and thrombocytopenia evolved during hospitalisation. Meanwhile, repeated peripheral blood assessments showed no blasts, dysplasia, or other abnormal circulating cells. Cytopenias had already been documented approximately one month before admission and had worsened in parallel with continued nutritional deterioration. We planned marrow examination after stabilisation if the cytopenias persisted. Following supportive treatment and transfer to intensive care, the patient improved clinically and haematologically; in the absence of new peripheral-smear abnormalities and with sustained count recovery, marrow examination was reconsidered but ultimately judged unlikely to alter management.

The nutritional phenotype was supported by severe hypoproteinaemia, hypoalbuminaemia,

hypcholesterolaemia and micronutrient depletion. Initial total protein was 38 g/L, and albumin was approximately 17.7 g/L when estimated from the albumin fraction on serum protein electrophoresis; later, directly measured serum values were higher after plasma, albumin, and nutritional support. Cholesterol was 1.57 mmol/L, serum iron 5.22 μ mol/L, transferrin saturation 8.9%, ferritin 22.89 μ g/L, vitamin B12 borderline low at 147.6 pmol/L, and folate low at 7.93 nmol/L. Low-grade urinary protein was transiently present but was not nephrotic-range and was negative on 9 July. Protein-losing enteropathy was clinically unlikely in the absence of gastrointestinal symptoms, and chronic liver disease was not supported by imaging, viral hepatitis testing or subsequent recovery. Pre-support serum protein electrophoresis showed a polyclonal pattern with a gamma fraction of 30.2%. Early C3 and C4 were low and later normalised. Haemophagocytic lymphohistiocytosis was considered but was not supported by the low ferritin, low triglycerides (0.54 mmol/L) and only brief low-grade fever; soluble interleukin-2 receptor testing was unavailable.

Reconstruction of the original laboratory record clarified the extreme LDH elevation. LDH measured 5684 U/L on 5 July and 5324 U/L on 6 July, then declined to 3877 U/L and 2630 U/L on 9 July and 2307 U/L on 10 July. Early CRP values were low or only minimally raised (approximately 2.9–5.6 mg/L). The enzyme pattern did not identify the source of LDH: early AST was only modestly elevated (up to 66.5 U/L), ALT was 13.7–17.2 U/L, and creatine kinase was low (13.9 U/L, later 11.4 U/L). Direct antiglobulin testing was negative, and no schistocytes were seen; haptoglobin, plasma-free haemoglobin and LDH isoenzymes were unavailable. We therefore do not attribute the LDH elevation to a single mechanism. Importantly, during the later post-ICU readmission CRP rose to 142.3 mg/L, demonstrating preserved capacity for an acute-phase response. The initially low CRP is therefore reported only as a non-specific contextual observation and not as evidence of impaired CRP synthesis or as a discriminator against malignancy or infection.

The coagulation course was similarly complex. A marked consumptive coagulopathy with DIC-like features evolved during hospitalisation,¹ with markedly elevated D-dimer, prolonged PT/INR, hypofibrinogenaemia and a subsequent fall in platelets. Severe malnutrition and hepatic synthetic dysfunction were important competing contributors, and serial measurements were influenced by active replacement therapy. Early in admission, PT was 44–50%, INR 1.64–1.85, and fibrinogen 1.24–1.76 g/L. On 9 July, platelets were 148×10^9 /L, INR 1.52, fibrinogen 1.57 g/L and D-dimer >10 mg/L; on 10 July, platelets were 125×10^9 /L, INR 1.72, fibrinogen 1.13 g/L and D-dimer 8.85 μ g/mL; and on 11 July, platelets were 98×10^9 /L, INR 1.56,

fibrinogen 1.14 g/L and D-dimer 7.60 μ g/mL. These measurements were obtained during active replacement therapy and cannot be interpreted as spontaneous correction. After ICU treatment, PT was 71%, and INR 1.27; by 18 August, PT was 72%, and INR 1.25; and on 22 September, PT was 78%, INR 1.19, fibrinogen 3.83 g/L, and D-dimer 1.62 μ g/mL. Subsequent follow-up confirmed complete normalisation of the coagulation profile.

Pleural-fluid protein was 24.4 g/L, albumin 16.8 g/L, LDH 1546 U/L and N-terminal pro-brain natriuretic peptide 2005 pg/mL; these values were pleural-fluid, not serum, measurements. In the setting of profound hypoproteinaemia and evidence of cardiac contribution, the effusion was considered mixed and predominantly hypo-oncotic/cardiac rather than assigned a single mechanism.¹⁴ Cytology was negative for malignant cells. Beta-2 microglobulin was 4.997 mg/L but, because it is non-specific, was not used to support or exclude malignancy. Autoimmune testing was negative; HIV, hepatitis B and C, Leishmania and Schistosoma testing was negative, while Epstein-Barr virus and cytomegalovirus serology indicated past exposure.

Parvovirus B19 DNA was detected by quantitative PCR at 210 IU/mL; the reporting laboratory considered values below 100 IU/mL negative. Simultaneous B19 IgM and IgG were not obtained. The low viral load and spontaneous PCR negativity at the two-month follow-up favour low-level persistence or resolving infection, but no universal viral-load threshold can exclude clinically meaningful B19 disease because interpretation depends on assay, host and timing.^{9–11} Multilineage cytopenia also does not exclude a contribution from B19. We therefore present the result as a probable confounder rather than a proven causal or definitively incidental finding.

Treatment required substantial supportive care. During the first eight days at Memorial Regional Hospital, the patient received two units of fresh frozen plasma and two cryoprecipitate administrations daily, together with parenteral nutrition and refeeding precautions,^{3,13} intravenous albumin, intravenous iron, intramuscular vitamin B12 and folate replacement. He received eight packed red-cell units in total: four during the initial admission until haemoglobin reached 64 g/L, two further units before transfer, and two during the subsequent ICU stay. No platelet transfusions were given. After eight days, he was transferred to the intensive care unit at Mother Teresa University Hospital, where intravenous fibrinogen replacement was continued for approximately four days. He was then briefly transferred to Gastroenterology and returned the same day to our hospital for about one more week of multidisciplinary care. No further red-cell transfusions were required after his return.

The early haemoglobin rise was therefore transfusion-related and was not used as evidence of

Table 1. Reconstructed clinical, laboratory and treatment timeline.

Referral / 5 Jul	Outside private-lab CBC: Hb 30 g/L, WBC $1.48 \times 10^9/L$, platelets $14 \times 10^9/L$. Platelet clumping was documented on review, so the platelet value was considered spurious and excluded from longitudinal interpretation. Earliest full-index panel after stabilization: Hct 11.8%, MCV 90.5 fL, MCH 29.5 pg, MCHC 32.6 g/dL, RDW 35.2%. Reticulocytes 3%; RPI ~0.3.	LDH 5684 U/L. Early CRP low/near-normal.	Initial total protein 38 g/L; albumin ~17.7 g/L estimated from SPEP. Early PT 44–50%, INR 1.64–1.85, fibrinogen 1.24–1.76 g/L.	PRBC, FFP/cryo, albumin, nutrition and micronutrient replacement started. No platelet transfusion.
6 Jul / in-house verification	Hb 37 g/L before transfusion → 54 g/L after transfusion; WBC $1.86\text{--}1.74 \times 10^9/L$; platelets $175\text{--}179 \times 10^9/L$. The platelet count was verified in-house and was not transfusion-related.	LDH 5324 U/L.	Bilirubin ~2.50 mg/dL (direct 1.51).	Transfusion-supported phase.
9–11 Jul	9 Jul: Hb 79, WBC 1.49, platelets 148. 10 Jul: Hb 71, WBC 2.34, platelets 125. 11 Jul: Hb 84, WBC 2.21, platelets 98 (all Hb g/L; counts $\times 10^9/L$).	LDH 2630 U/L (9 Jul) → 2307 U/L (10 Jul). CK low.	9 Jul: INR 1.52, fibrinogen 1.57 g/L, D-dimer >10 mg/L. 10 Jul: INR 1.72, fibrinogen 1.13, D-dimer 8.85 $\mu\text{g/mL}$. 11 Jul: INR 1.56, fibrinogen 1.14, D-dimer 7.60.	Six PRBC units received before ICU transfer; 2 FFP + 2 cryoprecipitate administrations daily during the first 8 days.
ICU + return to Memorial	Two further PRBC units in ICU (8 total). After return and no further PRBC: Hb 105 g/L, WBC $12.17 \times 10^9/L$, platelets $643 \times 10^9/L$.	Post-ICU CRP 142.3 mg/L, confirming preserved acute-phase response.	After return: total protein 68.8 g/L, albumin 41.3 g/L, PT 71%, INR 1.27.	~4 days ICU with IV fibrinogen replacement; brief Gastrohepatology transfer; ~1 further week at Memorial. Psychiatric care initiated.
18 Aug 2025	Follow-up during recovery.	Not repeated.	Total protein 67.1 g/L; albumin 37.5 g/L; bilirubin 0.62 mg/dL; PT 72%, INR 1.25; AST/ALT 98/64 U/L. Ferritin 480 ng/mL after transfusion/iron/inflammatory phase.	No further transfusions.
22 Sep 2025 (~2 months)	Hb 110 g/L, WBC $7.94 \times 10^9/L$, platelets $403 \times 10^9/L$; MCV 76.8 fL.	CRP 4.4 mg/L. B19 PCR negative at the two-month milestone.	Albumin 40.1 g/L; total protein 74.8 g/L; AST/ALT 29/28 U/L; PT 78%, INR 1.19; fibrinogen 3.83 g/L; D-dimer 1.62 $\mu\text{g/mL}$; C3/C4 normalized.	Outpatient oral iron, folate, B12 and antidepressant treatment.
June 2026	Hb 138 g/L, WBC $7.18 \times 10^9/L$, platelets $273 \times 10^9/L$; MCV 77.3 fL, MCH 25.0 pg.	Not repeated.	Ferritin 30.04 ng/mL.	Sustained multilineage recovery; mild microcytosis persisted.

Hb, haemoglobin; Hct, haematocrit; MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RPI, reticulocyte production index; LDH, lactate dehydrogenase; CRP, C-reactive protein; INR, international normalised ratio; SPEP, serum protein electrophoresis; PRBC, packed red blood cells; FFP, fresh frozen plasma. The outside 5 July platelet count of $14 \times 10^9/L$ was considered spurious because platelet clumping was documented; repeat in-house counts were in the normal range before any platelet transfusion. Early haemoglobin values were transfusion-influenced; later sustained recovery after transfusion cessation is more informative.

endogenous marrow recovery. On 11 July, while still in the transfusion-supported phase, haemoglobin was 84 g/L, white cells $2.21 \times 10^9/L$ and platelets $98 \times 10^9/L$. After returning from the ICU and stopping red-cell transfusion, haemoglobin was 105 g/L, white cells $12.17 \times 10^9/L$, and platelets $643 \times 10^9/L$; serum total protein was 68.8 g/L, albumin 41.3 g/L, and INR 1.27. Psychological and psychiatric evaluation confirmed a major depressive episode with food refusal, and antidepressant therapy was initiated. After discharge, he continued oral ferrous fumarate providing 130 mg elemental iron, folic acid 1200 μg and vitamin B12 1000 μg , together with antidepressant treatment.

Recovery was sustained. On 18 August 2025, total protein was 67.1 g/L, albumin 37.5 g/L and total

bilirubin 0.62 mg/dL; PT had normalised to 72%, and INR had improved to 1.25, although AST/ALT remained elevated at 98/64 U/L. By 22 September, haemoglobin was 110 g/L, white cells $7.94 \times 10^9/L$ and platelets $403 \times 10^9/L$; albumin was 40.1 g/L, total protein 74.8 g/L, bilirubin 0.37 mg/dL, AST/ALT 29/28 U/L, CRP 4.4 mg/L, fibrinogen 3.83 g/L and C3/C4 had normalized. Repeat B19 PCR was negative at the two-month follow-up. By June 2026, haemoglobin was 138 g/L, white cells $7.18 \times 10^9/L$ and platelets $273 \times 10^9/L$, confirming sustained recovery of all three lineages; mild microcytosis persisted (MCV 77.3 fL, MCH 25.0 pg) with ferritin 30.04 ng/mL.

Adult kwashiorkor or severe oedematous protein-energy malnutrition is uncommon in well-resourced

settings but has been reported in adults with severe dietary restriction, psychiatric disease, malabsorption and gastrointestinal surgery.^{4,15} Severe malnutrition can produce cytopenias, including through gelatinous transformation of bone marrow, although this lesion is neither specific to malnutrition nor inferable without marrow examination.⁵⁻⁷ Micronutrient depletion may also have contributed in this patient: iron indices were deficient, vitamin B12 was borderline low, and folate was low, and all were replaced as part of nutritional rehabilitation. Severe starvation may also be associated with reversible hepatic injury.⁸ These adult data support the biological plausibility of the presentation while reinforcing the need for caution when assigning causality. The main limitations of this case remain the absence of marrow histology, B19 serology, haptoglobin and LDH isoenzymes, and the inability to assign a single mechanism to the extreme LDH elevation. The nutritional phenotype and longitudinal recovery strongly supported the diagnosis of severe oedematous protein-energy malnutrition, but did not definitively prove it.

In summary, severe adult protein-energy malnutrition can present with severe cytopenias, organomegaly, serosal effusions and coagulation abnormalities that closely resemble a primary haematological disorder. In this patient, the nutritional history, peripheral blood morphology, multisystem phenotype and sustained recovery after the acute transfusion-supported phase made malnutrition the most likely unifying diagnosis. The central diagnostic lesson is not a single laboratory ratio, but the value of integrating morphology, nutritional history, competing causes, treatment effects and longitudinal evolution before escalating invasive investigation.

Acknowledgments. The authors thank the nursing, biochemistry, microbiology, and transfusion service teams at Memorial Regional Hospital (Spitali Rajonal Memorial), Fier, and Qendra Spitalore Universitare “Nënë Tereza”, Tirana, for their contributions to this patient's care. The authors are grateful to the patient and his family for their consent to publication.

Authors' Contributions. LS conceived the case report, was the haematologist responsible for the haematological assessment and diagnostic synthesis, reconstructed the clinical timeline, performed the literature review and drafted the manuscript. AB was the gastroenterologist and hepatologist responsible for assessment of hepatic involvement and contributed to clinical interpretation and manuscript revision. AV was the cardiologist responsible for assessment of the pericardial and pleural effusions and contributed to clinical interpretation and manuscript revision. XD was the intensive care physician responsible for inpatient stabilisation, transfusion and coagulopathy management and contributed to clinical interpretation and manuscript revision. KS contributed to inpatient critical care management during the transfer episode and reviewed the manuscript. All authors read and approved the final version.

Ethics Approval And Consent. This case report was prepared in accordance with the Declaration of Helsinki and institutional guidelines. The patient provided written informed consent for publication of this case report and accompanying clinical data; a copy is available for review by the Editor-in-Chief.

Liri Seraj¹, Aureta Bruci², Amarildo Voci³, Xhoana Derraj⁴ and Krisli Serani⁵.

¹ Department of Haematology, Memorial Regional Hospital (Spitali Rajonal Memorial), Fier, Albania.

² Department of Gastroenterology and Hepatology, Memorial Regional Hospital, Fier, Albania.

³ Department of Cardiology, Memorial Regional Hospital, Fier, Albania.

⁴ Department of Intensive Care, Memorial Regional Hospital, Fier, Albania.

⁵ Department of Intensive Care, Qendra Spitalore Universitare “Nënë Tereza” (Mother Teresa University Hospital), Tirana, Albania.

Competing interests: The authors declare no competing interest.

Correspondence to: Liri Seraj, MD. Department of Hematology, Memorial Regional Hospital (Spitali Rajonal Memorial), Fier, Albania. E-mail: liri.seraj@srmf.gov.al.

References:

1. Levi M, Toh CH, Thachil J, Watson HG. Guidelines for the diagnosis and management of disseminated intravascular coagulation. *Br J Haematol*. 2009;145:24-33. <https://doi.org/10.1111/j.1365-2141.2009.07600.x> PMID:19222477
2. Pepys MB, Hirschfield GM. C-reactive protein: a critical update. *J Clin Invest*. 2003;111:1805-1812. <https://doi.org/10.1172/jci200318921> PMID:32499051
3. Crook MA. Refeeding syndrome: problems with definition and management. *Nutrition*. 2014;30:1448-1455. <https://doi.org/10.1016/j.nut.2014.03.026> PMID:25280426
4. Arcieri ST, Cheung S, Belkin A, Pillai A, Gupta R. Kwashiorkor on the south shore. *J Community Hosp Intern Med Perspect*. 2021;11:528-530. <https://doi.org/10.1080/20009666.2021.1933718> PMID:34211663
5. Miguelañez Matute L, van Noord D, Rayman N, Guillen SS. Gelatinous bone marrow transformation due to extensive weight loss in median arcuate ligament syndrome. *BMJ Case Rep*. 2022;15:e246916. <https://doi.org/10.1136/bcr-2021-246916> PMID:35039369

6. Gunaratne MDSK, Shi M, Go RS. A comprehensive review on gelatinous transformation of the bone marrow. *Expert Rev Hematol*. 2024;17:547-554.
<https://doi.org/10.1080/17474086.2024.2385472> PMID:39060221
7. Böhm J. Gelatinous transformation of the bone marrow: the spectrum of underlying diseases. *Am J Surg Pathol*. 2000;24:56-65.
<https://doi.org/10.1097/00000478-200001000-00007> PMID:10632488
8. Restellini S, Spahr L, Rubbia Brandt L. Severe starvation-induced hepatocyte autophagy as a cause of acute liver injury in anorexia nervosa: a case report. *Case Rep Hepatol*. 2013;2013:749169.
<https://doi.org/10.1155/2013/749169>
9. Maple PAC, Hedman L, Dhanilall P, Kantola K, Nurmi V, Söderlund-Venemo M, Brown KE, Hedman K. Identification of past and recent parvovirus B19 infection in immunocompetent individuals by quantitative PCR and enzyme immunoassays: a dual-laboratory study. *J Clin Microbiol*. 2014;52:947-956.
<https://doi.org/10.1128/JCM.02613-13> PMID:24403307
10. Heegaard ED, Brown KE. Human parvovirus B19. *Clin Microbiol Rev*. 2002;15:485-505.
<https://doi.org/10.1128/cmr.15.3.485-505.2002> PMID:12097253
11. Eid AJ, Brown RA, Patel R, Razonable RR. Parvovirus B19 infection after transplantation: a review of 98 cases. *Clin Infect Dis*. 2006;43:40-48.
<https://doi.org/10.1086/504812> PMID:16758416
12. Morlese JF, Forrester T, Jahoor F. Acute-phase protein response to infection in severe malnutrition. *Am J Physiol*. 1998;275:E112-E117.
<https://doi.org/10.1152/ajpendo.1998.275.1.e112> PMID:9688881
13. Stanga Z, Brunner A, Leuenberger M, Grimble RF, Shenkin A, Allison SP, Lobo DN. Nutrition in clinical practice-the refeeding syndrome: illustrative cases and guidelines for prevention and treatment. *Eur J Clin Nutr*. 2008;62:687-694.
<https://doi.org/10.1038/sj.ejcn.1602854> PMID:17700652
14. Romero-Candeira S, Fernandez C, Martin C, Sanchez-Paya J, Hernandez L. Influence of diuretics on the concentration of proteins and other components of pleural transudates in patients with heart failure. *Am J Med*. 2001;110:681-686.
[https://doi.org/10.1016/s0169-5002\(05\)81038-4](https://doi.org/10.1016/s0169-5002(05)81038-4)
15. Dixon W, Bromfield B, Redston M, Sack J. Kwashiorkor mimicking portal hypertensive ascites after bariatric surgery. *ACG Case Rep J*. 2026;13:e02260.
<https://doi.org/10.14309/crj.0000000000002260> PMID:42578059