Case report

An unusual case of typhoid fever encephalitis

DOI: 10.5377/alerta.v8i1.17872

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0009-0004-3682-3620

OPEN ACCESS

Un caso inusual de encefalitis por fiebre tifoidea.

Suggested citation:
Chirino Molina WY. An unusual case of typhoid fever encephalitis. Alerta. 2025;8(1): 14-21. DOI: 10.5377/alerta.v8i1.17872

Editor:
Nadia Rodríguez.

Received:
September 19, 2023

Accepted:
November 19, 2024.

Published:
January 22, 2025.

Author contribution:
WYCM: study conception, manuscript design, writing, revising and editing, literature search and data or software management , data analysis and data collection.

Conflicts of interest:
No conflicts of interest.

Abstract
Case presentation. A 13-year-old female patient with no known comorbidities consulted with a ten-day history of fever with chills, diarrhea four times a day and vomiting, treated with acetaminophen. Four days before consulting, the patient experienced adynamia, confusion and difficulty walking. She was taken by her mother to a health facility. **Treatment.** She was admitted to a second level hospital for suspected salmonellosis, encephalitis, water and electrolyte imbalance, acute renal injury and acute gastroenteritis with severe dehydration. She was treated with parenteral hydration and antibiotics, and was referred to a tertiary hospital for respiratory distress with need for mechanical ventilation, where a positive blood culture for Salmonella Typhi was obtained. **Outcome.** The patient presented systemic inflammatory response syndrome due to sepsis that was managed with vasoactive amines; on the sixteenth day of hospitalization the patient presented tachycardia and fever, a blood culture positive for multidrug-resistant Acinetobacter Baumannii was obtained. She presented myocarditis, neurological deterioration with a Glasgow score of 8, developed multisystemic failure and died after 32 days of hospital stay.

Keywords
Typhoid Fever, Salmonella Food Poisonings, Cross Infection..

Resumen
Presentación del caso. Paciente femenina de 13 años de edad, sin comórbidos conocidos quien consultó con historia de diez días de presentar con escalofríos, diarreas líquidas cuatro veces al día y vómitos, fue tratada con acetaminofén. Cuatro días antes de consultar, la paciente había presentado adinamia, confusión y dificultad a la deambulación por lo que fue llevada por su madre a un establecimiento de salud. **Intervención terapéutica.** Inició la atención en un hospital de segundo nivel, fue ingresada por sospecha de salmonelosis, encefalitis, desequilibrio hidroelectrolítico, lesión renal aguda y gastroenteritis aguda con deshidratación grave. Fue tratada con hidratación parenteral y antibióticos, y se remitió a un hospital de tercer nivel, por presentar distrés respiratorio con necesidad de ventilación mecánica, ahí se obtiene un hemocultivo positivo a *Salmonella Typhi*. **Evolución clínica.** La paciente presentó síndrome de respuesta inflamatoria sistémica por sepsis que fue manejada con aminas vasoactivas; el décimo sexto día de hospitalización la paciente presentó taquicardia y fiebre, se obtuvo un hemocultivo positivo a *Acinetobacter Baumannii* multirresistente. Presentó miocarditis, deterioro neurológico con Glasgow de 8 puntos, desarrolló falla multisistémica y falleció con 32 días de estancia hospitalaria.

Palabras clave
Fiebre Tifoidea, Intoxicación Alimentaria por Salmonella, Infección Hospitalaria.

Introduction

Typhoid fever (TF) is a disease caused by *Salmonella typhi*[i] and is transmitted by the oral-fecal route;[ii,iii] consumption of contaminated food or water is the main route of entry.[iii] Humans are the only reservoir of this bacterium.[ii] Although most infections resolve with treatment, 2 % and 5 % of cases progress to a chronic carrier state. These individuals, generally asymptomatic, may excrete and act as reservoirs of transmission for years.[iv]

This disease is characterized by continuous fever, headaches, and relative brady-

cardia. Symptoms vary from mild, including asthenia, cough, chills, anorexia, nausea, vomiting with adynamia, and malaise; 66 % of patients with TF experience diarrhea. Other findings include hepatomegaly, splenomegaly, and complications including altered consciousness, neurological deficits, nephritis, pneumonia, peritonitis, hepatitis, and, in approximately 3 % of cases, hemorrhage and perforation. These perforations can occur in the third week of the disease due to ulceration and death in Peyer's plaques.[i,ii] Without treatment, the lethality rate is estimated to be 10-20 % of cases.[i,ii].

Receiving medical care and treatment in a timely manner reduces the case fatality rate to less than one percent.[ii] Blood culture is one of the diagnostic methods used. The most commonly affected age group is 5 to 14 years. This depends on geographic location and access to primary health care, as well as economic and cultural factors.[x] This disease is common in developing countries with unsanitary conditions.[x,xi].

According to estimates made by the World Health Organization, the annual incidence of TF in 2019 was 27 million cases, of which almost 210 000 died.[ii] This case is presented with the aim of describing the clinical evolution and critical outcome of a case of TF complicated by Acinetobacter bauman,ii highlighting the importance of timely diagnosis and treatment.

Case presentation

A thirteen-year-old female adolescent with a ten-day history of moderate to severe febrile process, unquantified, continuous, accompanied by chills, diarrhea, an average of four bowel movements per day, of liquid consistency, and vomiting of gastric contents (Figure 1).

She presented slight improvement when self-medicating with acetaminophen 500 mg every six hours. However, four days before the consultation, she began to deteriorate in her state of health, with marked hyporexia, adynamia, difficulty in ambulation, and a confusional state. For this reason, they attended the emergency unit of a second-level hospital.

On physical examination, blood pressure (BP) of 100/60 mmHg, heart rate (HR) of 117 bpm, temperature of 36. 9 ˚C, respiratory rate of 30 rpm, and skin pallor were described. Weight was 50 kg and height 155 cm. She had drowsiness, sunken eyes, and dry oral mucosa. Examinations indicated hypokalemia, hyponatremia, mild leukopenia with marked neutrophilia, plateletopenia, and acute renal failure (Table 1).

Treatment

Hospital admission was decided (Figure 1), with suspected TF, viral encephalitis, hydroelectrolyte imbalance, acute kidney injury, and acute gastroenteritis with severe dehydration. Intravenous (IV) hydration with 2 L of 0. 9 % isotonic saline by rapid infusion was indicated. Subsequently, maintenance solutions were administered with crystalloids, ceftriaxone 1 g IV, supplemental oxygen at 3 L/min, and a central vein catheter was placed.

The following day, she was transferred to a tertiary care hospital for respiratory distress. The following vital signs were recorded: BP 98/66 mmHg, HR 90 bpm, and Cheyne-Stokes respiration. Arterial blood gas analysis reported metabolic acidosis with ventilatory failure (Ph: 7. 095, PO2: 57. 6, and Kirby index: 67. 7); support with mechanical ventilation was decided due to severe respiratory failure. Laboratory tests revealed plateletopenia, renal failure, and elevated liver enzymes (Table 1). In the Emergency Unit, treatment was indicated with isotonic solution 0. 9 % IV, potassium chloride 60 mEq, and two cc of magnesium sulfate every 12 hours. She was also administered 10 % calcium gluconate every 8 hours, ceftriaxone 2 g every 12 hours, and a nasogastric tube was placed. She was then transferred with mechanical ventilation to another tertiary hospital specializing in the care of minors, with the diagnostic impression of systemic lupus erythematosus with multiorgan activity.

She was transferred to the intensive care unit with the following diagnoses: systemic inflammatory response syndrome, suspected TF, and suspected SARS-CoV-2. The indicated treatment consisted of 1 L of 5 % IV dextrose, 80 ml of 7. 5 % sodium bicarbonate, 20 ml of 15 % potassium chloride, and 2 ml of magnesium sulfate, administered through a continuous infusion pump at a rate of 105 cc/hour and 25 ml of 10 % calcium gluconate every eight hours. In addition, she started treatment with triple antibiotics, ceftriaxone 2 g every 12 hours, vancomycin every 6 hours, and meropenem 3 g every 12 hours, and transfusions were performed with five units of platelets every 12 hours, for a total of 40 units and one unit of packed red blood cells every day.

Outcome

During the first day of the hospital stay, she was maintained on adrenaline at a rate of 0. 1 mg/kg/minute. Blood gases showed metabolic acidosis under correction with invasive mechanical ventilation. During the

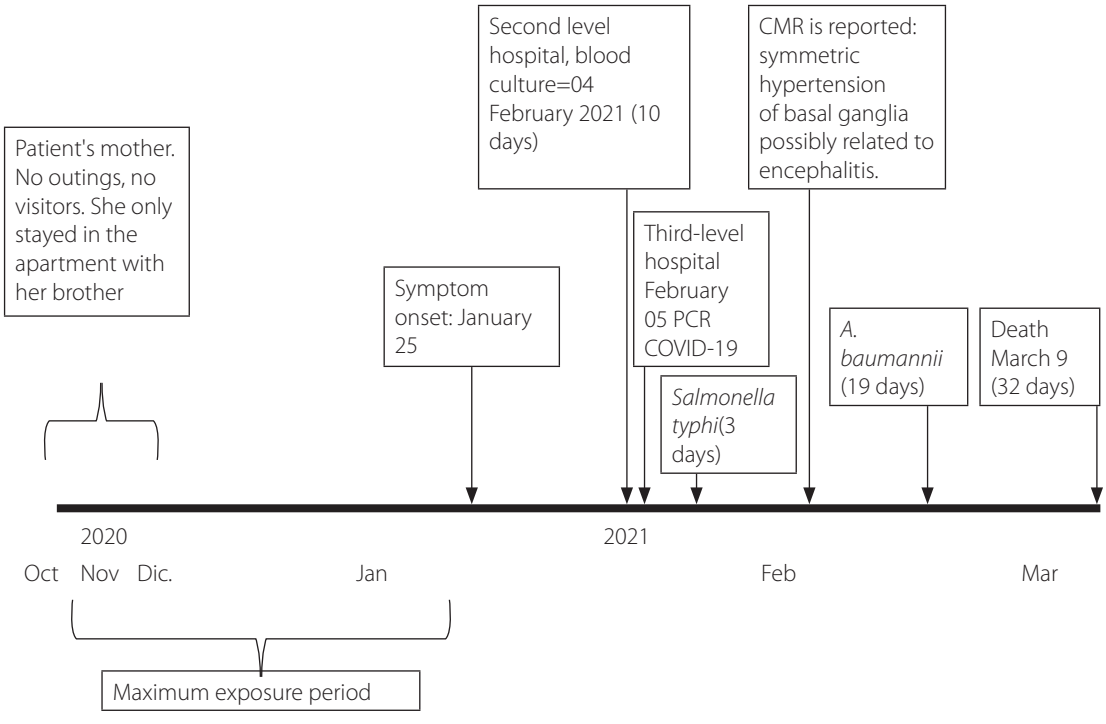

Figure 1. Timeline.

Table 1. Laboratory tests at referral hospitals.

Laboratory data	Second level hospital emergency	Third level hospital emergency	Normal value
Hematocrit (%)	36.2	25.8	33-36
Hemoglobin (g/dL)	12.4	9.2	10.5-12.0
Leukocytes (x103/pl)	5.31	4.920	6-17
Neutrophils (%)	82.2	87.0	55-65
Lymphocytes (%)	15.6	11.4	20-40
Platelets (x10^3/mL)	115 000	82.000	150-350
Na (mEq/L)	114.0	131	135-145
K (mEq/L)	2.16	2.6	3.50-5.50
Glucose (mEq/L)	117	-	70-105
Urea nitrogen (mEq/L)	64.9	63	7-18
Cr (mEq/L)	4.05	4.64	0.42-1.09
TP[a] (s)	-	12.5	12.1-14.5
TPT[b] (s)	-	43.2	33.6-43.8
Fibrinogen (mg/dL)		131	180-350
TGO[c] (U/L)	247	286	10-a-42
TGP[d] U/L	50	62	10-a-40
DHL[e] (U/L)	1.417	2.085	91a-180
Albumin (g/dL)	-	2.41	3.5-5.0
PCR[f] (mg/L)	-	93.59	0.00-1.00
Pregnancy test	Negativa	-	-
Rheumatoid test	Negativa	-	-
HIV	Negativa	-	-

Prothrombin time b. Partial thromboplastin time c. Glutamic oxaloacetic transaminase d. Glutamic pyruvic transaminase e. Lactate dehydrogenase f. C-reactive protein

second day of hospitalization, arterial blood gases showed improvement, due to which bicarbonate was withdrawn and fluid correction was performed with 5 % dextrose serum, 500 ml bidistilled water, 20 % sodium chloride, 15 % potassium chloride, 50 % magnesium chloride, 10 % glucose by infusion pump at 81. 6 ml/hour. She required an increase in the dose of adrenaline to 0. 15 mg/kg/min because she started with hypotension and oliguria (Table 2). On the fifth day, mechanical ventilation was suspended, oxygen management was started through a mask with a reservoir, and ultrasonography was performed, which reported no alterations in the kidneys.

On the seventh day of hospitalization, she continued with little verbal response; somnolent, four days later, she was stable. Brain MRI showed hyperintense signal foci at the level of the basal ganglia in T2 and FLAIR sequences with mild atrophic changes, permeable ventricular system, and no abnormalities. Thalamus, hypothalamus, pons, cerebellum, medulla oblongata, craniocervical junction, corpus callosum, and pituitary gland without abnormalities. The eyeballs were seen without abnormalities, and there

Table 2. Laboratory tests performed during hospital stay

Laboratory data (Unit)	Days of hospital evolution														Normal value
	0	2	3	4	5	9	11	12	14	15	18	21	26	30	
Hematocrit (%)	23.5	25.9	34.0	32.5	30.7	37.0	26.1	26.9	24.0	26.3	23.4	32.0	30.0	20.7	33-36
Hemoglobin (g/dL)	9.0	10.1	12.1	11.8	10.9	12.5	-	8.8	8.2	8.8	8.7	11.0	11.3	7.8	10.5-12.0
Leukocytes (x10^3/pl)	6	13	24.82	23.46	7.57	18	7.36	6.69	5.6	7.08	2.77	5.13	4.31	6.44	6-17
Lymphocytes (%)	82	86	81	86	68	73	87.9	75	59	68.6	79.1	65	-	76	55-65
Platelets (%)	12	9	14	13.5	24	19	8.6	15	31	19.8	16.6	2	-	18	20-40
Platelets (x10^3/mL)	135.7	38	55	89	115	189	322	148	160	154	200	86	167	250	150-350
Na (mEq/L)	142	125	138	128	134	152	132	134	-	129	128	135	133	123	135-145
K (mEq/L)	2.53	12.1	3.68	7.4	4.84	5.34	4.19	3.39	-	4.39	2.7	2.82	4.09	3.8	3.50-5.50
Glucose (mEq/L)	101	314	241	284	163	-	147	120	-	-	95	-	-	92	70-105
NU (mEq/L)	68.9	64.9	77.4	81.70	80.40	-	32.40	26.8	16.1	-	-	-	-	-	7-18
Cr (mEq/L)	3.7	3.32	3.3	2.9	2.6	1.79	1.28	1.08	0.9	-	0.8	-	-	0.9	0.42-1.09
TP[a] (s)	12.9	9.6	8.5	8.7	8.8	8.3	11.6	-	8.4	-	9.6	-	-	10.4	12.1-14.5
TPT[b] (s)	24.8	23.2	20.5	19.5	22	22.5	23	-	20.2	-	25.9	-	-	28	33.6-43.8
Fibrinogen (mg/dL)	85	138	164	128	-	489	574	427	380	-	597	-	-	250	180-350
TGO[c] (U/L)	237	98	-	67	-	51	44	33	42	-	17	-	-	-	10-42
TGP[d] U/L	58.0	39	-	32	-	25	22	17	27	-	8	-	-	-	10-40
DHL[e] (U/L)	1426	-	-	-	-	-	-	-	-	-	-	-	-	-	91-180
Total protein (g/dL)	5.37	-	-	-	-	-	-	-	-	-	-	-	-	-	6.0-8.30
Albumin (g/dL)	2.3	-	-	2.8	3.1	3.2	3.1	3	2.5	-	2.1	-	-	2.3	3.5-5.0
Ferritin (x10^{3}ng/mL)	1.5	-	-	-	-	-	-	1,5	-	-	-	-	-	-	11.0-306.8
D-dimer (mg/L)	-	-	16	-	-	-	-	-	-	-	-	-	-	-	0-0.5
PCR[f] (mg/L)	12.02	7.07	-	7.8	-	-	7.05	2.36	1.06	-	28.9	-	-	10.4	0.00-1.00
Lipase (UI/L)	-	-	-	-	-	-	-	-	-	-	-	-	-	56	13-60
Amylase (UI/L)	-	-	-	-	-	-	-	-	-	-	-	-	-	21	28-100
Troponin I (ng/mL)	-	-	-	-	-	-	-	-	-	-	-	-	-	7948.3	0-0.4
Urine protein (mg/dL)	30	10	-	-	-	-	-	-	-	-	20	-	-	-	Absent

were inflammatory signs in the ethmoidal and sphenoidal areas.

They left frontal sinuses and inflammatory signs at the left mastoid level in addition to a symmetrical hyperintense signal of the basal ganglia, possibly related to encephalitis and ethmoidal sinusitis (Figure 2).

On the twelfth day of the hospital stay, she was evaluated by the nephrology specialist, who diagnosed renal failure secondary to salmonellosis. They indicated the suspension of treatment with amines, a decision that responded to the resolution of shock and hemodynamic stabilization. On the fourteenth day, she was afebrile, drained bilious fluid through a nasogastric tube, her abdomen was soft and depressible, and she was receiving oxygen through a nasal cannula at a rate of 3 L/min

On the twentieth day of the hospital stay, she was draining green-colored liquid through the nasogastric tube. In addition, she started with clonic movements of the right upper limb; due to this, she was administered 500 mg of phenytoin every 12 hours (two doses) and 5 mg of midazolam by IV; improvement was evidenced by mild reaction to stimuli, after six days of improvement. On the 27th day of hospitalization, the patient had a nasogastric tube draining green gastric contents, with diuresis of 0. 25 cc/hour and HR of 155 bpm. Due to this, she was ma-

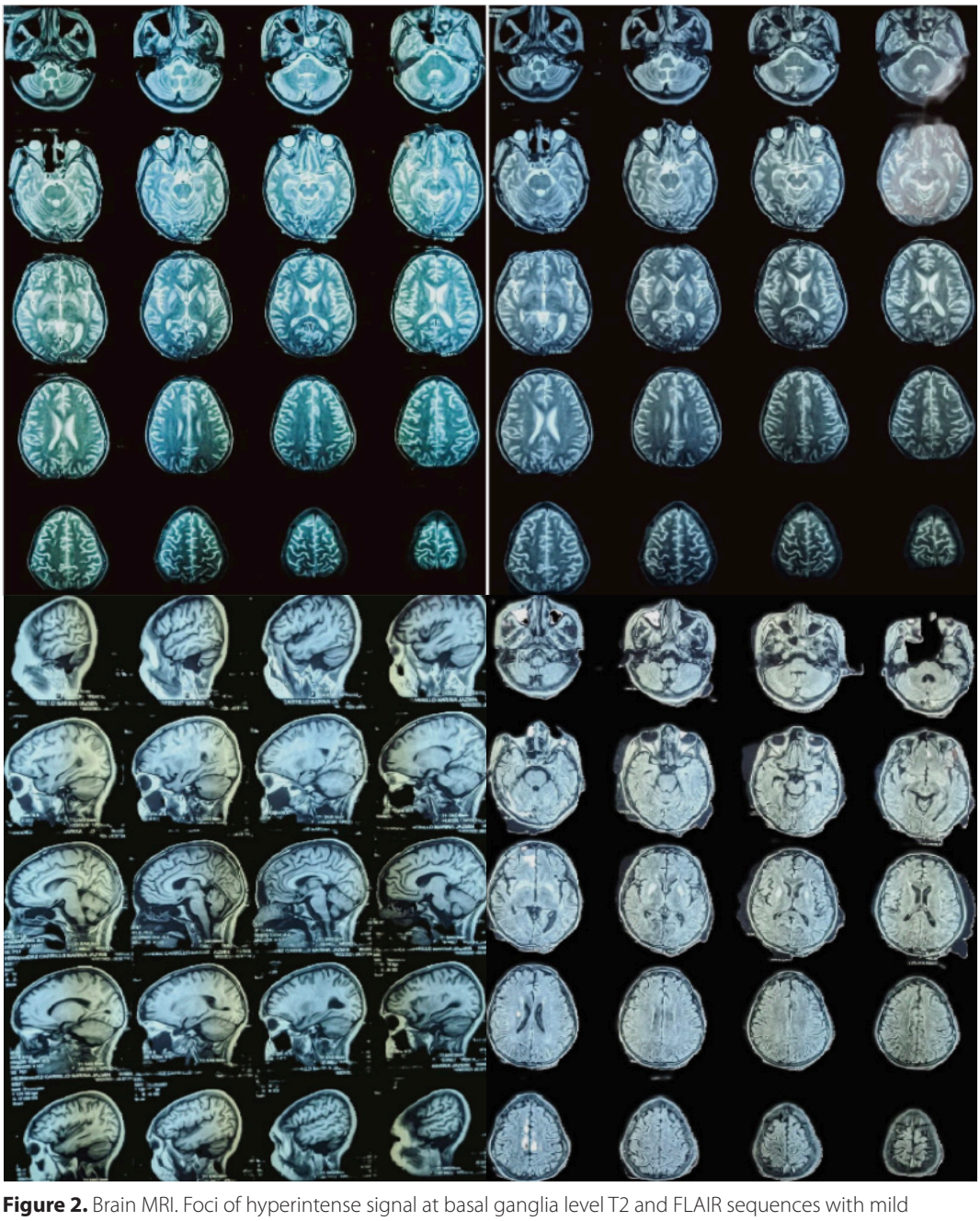

Figure 2. Brain MRI. Foci of hyperintense signal at basal ganglia level T2 and FLAIR sequences with mild atrophic changes, ventricular system permeable and without abnormalities. Inflammatory signs in ethmoidal, sphenoidal, and left frontal sinuses and inflammatory signs at the left mastoid level in addition to a symmetrical hyperintense signal of basal ganglia.

naged with IV hydration. The following day, she evolved with a globular abdomen.

On her thirtieth day of hospitalization, the patient was diagnosed with typhic encephalitis with a positive blood culture for *Salmonella typhi* and sepsis due to acinobacter baumani. In addition, she started with dyspnea and was draining abundant yellowish fluid through the nasogastric tube. She then presented further neurological deterioration deterioration with a GCS of eight, abdominal distension, and oliguria.

Consequently, invasive mechanical ventilation was started again, a central vein catheter was placed, and she was transferred to the intensive care unit. A cerebral computed axial tomography was performed, which reported an ischemic event in the lenticular area.

Physical examination showed periumbilical ecchymosis in the abdominal region, ecchymosis in the extremities, abdominal distension, decreased peristalsis, and edema of the lower limbs. The patient was administered packed red blood cells a unit every eight hours, for 12 units, and seven units of platelets every eight hours, for a total of 21. The results of laboratory tests indicated decreased clotting times and elevated fibrinogen and troponin I (Table 2).

On the thirty-first day of the hospital stay, the patient continued to require mechanical ventilation and amines. On the thirty-second day, the patient died from an infection of infective myocarditis.

Diagnosis

Salmonellosis by blood culture positive for *Salmonella typhi*. Sepsis with positive blood culture to multidrug-resistant *Acinetobacter baumannii*; the patient died of TF, disseminated vascular coagulation.

Discussion

TF is associated with socioeconomic factors,[iii] such as limited access to drinking water, poor sanitation, overcrowding, and malnutrition, which increase vulnerability to infection. The patient came from a rural area of El Salvador with a low socioeconomic level; she lived in a collective dwelling with intermittent water service, where the latrine was located next to the bedroom and less than one meter away from the kitchen, which had precarious hygienic conditions. These factors favored his exposure to the disease.[x,xi]

TF, without timely treatment, can trigger an exaggerated immune response with the formation of immunocomplexes, which are deposited in the glomeruli, causing glomerulonephritis, loss of protein in the urine (nephritic or nephrotic syndrome), and eventually, renal failure. Systemic inflammation can damage podocytes, favoring the massive proteinuria characteristic of nephrotic syndrome and contributing to renal deterioration. In this case, the patient developed renal failure secondary to TF; the tests showed hypoalbuminemia, proteinuria, creatinine elevations (Table 2), and lower limb edema. A similar case occurred with a ten-year-old boy who developed hypoalbuminemia and proteinuria and was diagnosed with typhoid encephalopathy with secondary nephrotic syndrome. TF with renal involvement is rare but may present as acute kidney injury, which may occur in 2-4 % of patients with TF.[xii]

This patient had had hematologic complications with plateletopenia since her initial presentation (Table 1), in addition to confusion, cardiovascular symptoms and hypotension. In a similar study, a patient presented cerebellitis as a complication of TF, in addition to plateletopenia of 133 000 and Hb of 5. 6 g/dl.[xiii]

With early diagnosis and appropriate treatment, TF is mild without complications with changes in coagulation tests, fibrinogen, including Dimero D (Table 2).

Cases have been described with complications, such as the patient with disseminated vascular coagulation. In another case, the patient presented from the beginning alterations in fibrinogen and platelets abnormalities of less than 75 000 10/ml[xiv], and died on the fourteenth day of hospital stay due to disseminated intravascular coagulation. In this disease, we must work with the population and continue to strengthen prevention and hygiene measures such as hand washing to avoid this disease.[xv]

TF, although predominant, affects the gastrointestinal system and can cause neurological complications such as encephalopathy, confusion, and seizures. In this case, the patient had an ischemic event in the lenticular area with neurological involvement, probably secondary to cerebral ischemia or inflammation. In addition, the inflammatory signs in paranasal sinuses and mastoids reinforce the hypothesis of a multifocal inflammatory process linked to the severity of the infection.

From the beginning, the patient under study was drowsy, with neurological alterations, neutrophilia, and plateletopenia (Table 1). A similar case occurred in a 34-year-old woman of low socioeconomic status and rural area, who presented with transaminase elevations, somnolence, seizures,

hypertensive lesion that did not enhance on T2-weighted and T2-FLAIR imaging, with substantial diffusion restriction on diffusion-weighted imaging sequences, involving the splenium of the corpus callosum.[xv]

Another male patient with a positive blood culture for *Salmonella typhi* experienced seizures, somnolence and disorientation, and no comorbidities. Brain MRI showed T2 and Flair[xv] hypertensive areas. *Salmonella typhi*-associated encephalopathy is not case-specific and may include cerebral edema, ischemia, and demyelination.[xvi]

The neurological changes in the patient under study were present from her presentation at emergency, whose brain MRI showed an ischemic hypoxic event, as well as T2 and Flair changes. Similar results were found in the reported case of a 75-year-old female patient, in whom brain MRI revealed confluent areas of hyperintense T2 and FLAIR signal.[xvii]

Early diagnosis of TF is crucial to prevent serious complications, such as irreversible brain damage or multi-organ failure. Rapid identification of unusual neurological signs, such as confusion, seizures, or focal deficits, allows timely intervention, especially in settings where systemic infections can have rare but severe neurological effects.

Strategies for detecting encephalitis include intensive clinical surveillance for neurological symptoms in patients with TF and the use of imaging tools such as MRI and CT scanning to determine brain ischemia or inflammation in early stages. In addition, monitoring of inflammatory markers and evaluation of renal and liver function may provide clues to systemic complications that could involve the central nervous system.

Treatment requires the administration of appropriate antibiotics to control the underlying infection, in addition to aggressive management of complications such as sepsis or encephalopathy. In cases of cerebral ischemic or inflammatory events, specific therapies such as anticoagulants are crucial for limiting damage. Timely treatment is essential for this infection; the patient arrived at the hospital center after ten days, which can significantly impact the efficacy of treatment and increase the risk of death in patients with this disease.

Ethical aspects

Consent and authorization of the clinical case were obtained from the mother responsible for the minor, respecting the Helsinki Declaration and international ethical guidelines for health-related research involving human beings.

Funding

No external funds were received for this work.

References

i. Kumate Rodríguez J, Gutiérrez Trujillo G, Santos Preciado JI, Muñoz Hernández O, Miranda Novales MaG, Solórzano Santos F. Infectología clínica Kumate-Gutiérrez. México. 18th Edition Méndez Editores; 2016.

ii. Heymann DL. 19 th Edition. El control de las enfermedades trasmisibles. Organización Panamericana de la Salud; (2017).

iii. Zhang J, Jolly , Nguyen T, Taha M, Lee, Corbeil A, *et al.* Locally acquired Typhoid fever outbreak linked to chronic carriage in Ottawa, Canada, 2018-2022. Can Commun Dis Rep. 2024 Nov 7;50 (11):412-418, DOI: 10.14745/ccdr.v50i11a05.

iv. Bhandari J, Thada PK, DeVos E.Typhoid Fever. In: StatPearls. StatPearls Publishing 2022. Available at: https://www.ncbi.nlm.nih.gov/books/NBK557513

v. Esther Bandres de Blas, Celia Buñuel, María Sangrós Tolosa.Fiebre tifoidea y otras salmonelosis. Revisión bibliográfica. RSI - Revista Sanitaria de Investigación. 2023. Consulted on August 5, 2023. Available at: https://revistasanitariadeinvestigacion.com/fiebre-tifoidea-y-otras-salmonelosis-revision-bibliografica/

vi. Birkhold M, Coulibaly Y, Coulibaly O, Dembélé P, Kim DS, Sow S, *et al.* Morbidity and Mortality of Typhoid Intestinal Perforation Among Children in Sub-Saharan Africa 1995-2019: A Scoping Review. World J Surg. 2020;44(9):2892-2902. DOI: 10.1007/s00268-020-05567-2

vii. Perforation Among Children in Sub-Saharan Africa 1995-2019: A Scoping Review. World J Surg. 2020;44(9):2892-2902. DOI: 10.1007/s00268-020-05567-2

viii. Revista Sanitaria de Investigacion.Fiebre Tifoidea.Consulted on October 8, 2024. Available at: https://revistasanitariadeinvestigacion.com/fiebre-tifoidea-articulo-monografico/

ix. Kgosiore M, Kelatlhegile G. Modelado de la trasmisison de la fiebre tifoidea con respuesta a las recaidas del tratamiento:control optimo y analisis de la relacion coste efectividad.Math Models Comput SiMUL, 2021; DOI: 10.1016/j.rinp.2023.107120.

x. Cortés JA, Romero-Moreno LF, Aguirre-León CA, Pinzón-Lozano L, Cuervo SI, Cortés JA, *et al.* Enfoque clínico del síndrome febril agudo en Colombia. Infectio. 2017;21(1):39-50. DOI: 10.22354/in.v21i1.640

xi. Khalaf YJ, Alagha R. Fatal Complications of Extensive Drug-Resistant Typhoid Fever:

A Case Report. Cureus. 15(6): e40672. DOI: 10.7759/cureus.40672

xii. Seratin F, Widiasta A, Adrizain R, Hilmanto D. An evaluation of kidney dysfunction as a common symptom of typhoid infection in an endemic country: A rare case study. IDCases. 2022;29: e01580. DOI: 10.1016/j.idcr.2022.e01580

xiii. Kumwenda M, Iroh Tam P-Y. An adolescent with multi-organ involvement from typhoid fever. Malawi Med J. 2019;31(2):159-160. DOI: 10.4314/mmj. v31i2.10

xiv. Nurnaningsih, William V, Rusmawatiningtyas D, Makrufardi F, Kumara IF. Sepsis and disseminated intravascular coagulation are rare complications of typhoid fever: A case report. Annals of Medicine and Surgery. 2022;73. DOI: 10.1016/j.amsu.2021.103226

xv. Azhar AB, Khalid A, Shah S, Azhar AB, Khalid A, Shah S. The Implications of Extensive Drug-resistant Typhoid Fever: A Case Report. Cureus. 2019;11(6). DOI: 10.7759/cureus.5032

xvi. Ghosh R, Moreno-García S, Roy D, Dubey S, Benito-León J. Typhoid fever presenting with central and peripheral nervous system involvement. Neurologia. 2023;38(2):134-136. DOI 10.1016/j.nrleng.2022.03.005

xvii. Ahmed M, Sureka J, Mathew V, Jakkani R, Abhilash K. Magnetic resonance imaging findings in a fatal case of Salmonella typhi-associated encephalopathy: a case report and literature review. Neurol India. 2011;59(2):270-272. DOI: 10.4103/0028-3886.81598