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Clinical Spectrum and Outcomes of Congenital and Postnatally Acquired Cytomegalovirus Infections: A Seven-Case Series from the Neonatal Intensive Care Unit of Hassan II University Hospital, Fez, Morocco

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Abstract **Case Series**

Background: Cytomegalovirus (CMV) is the most common congenital viral infection worldwide and a leading cause of sensorineural hearing loss and neurodevelopmental impairment. While postnatally acquired CMV infection is generally asymptomatic or mildly symptomatic in term infants, it can result in significant morbidity, particularly among very preterm and immunocompromised neonates. ***Objective:*** To describe the clinical, laboratory, radiological, therapeutic, and outcome characteristics of CMV infections diagnosed in a neonatal intensive care unit (NICU), with particular emphasis on distinguishing documented congenital infection, symptomatic neonatal disease, and presumed postnatal infection. ***Methods:*** We conducted a two-year retrospective descriptive study in the NICU of Hassan II University Hospital, Fez, Morocco. Seven neonates with detectable CMV DNA by blood polymerase chain reaction (PCR) were included. Perinatal characteristics, neurological, sensory, hematological, hepatic, and respiratory manifestations, imaging findings, antiviral management, and clinical outcomes were reviewed. In the absence of systematic CMV screening at birth, infections diagnosed beyond the neonatal period were classified as presumed postnatal infections. ***Results:*** Gestational age ranged from 29 to 40 weeks, and birth weight from 1,200 to 3,400 g. One term neonate presented with symptomatic congenital CMV infection characterized by microcephaly, neurosensory involvement, chorioretinitis, and severe cerebral abnormalities, including lissencephaly, agenesis of the corpus callosum, and a posterior fossa malformation. Another neonate, born at 34 weeks and 4 days of gestation, developed multisystem disease characterized by jaundice, hepatic involvement, cutaneous manifestations, and a high CMV viral load. Pneumonia was reported in four patients, four required supplemental oxygen, and two required mechanical ventilation. Hematological abnormalities included thrombocytopenia in four patients and anemia in five. Two very-low-birth-weight infants (1,200 and 1,400 g), initially admitted for prematurity, had received breast milk. Three patients had confirmed HIV infection. One neonate with HIV-CMV coinfection died from respiratory failure. ***Conclusion:*** This case series highlights the broad clinical spectrum of CMV infection in the NICU, ranging from severe congenital neurological involvement to hepatic, hematological, and respiratory manifestations in vulnerable neonates. The lack of systematic virological screening at birth limits the reliable distinction between congenital and postnatally acquired infections. Implementing an early diagnostic strategy for at-risk neonates may improve disease classification, guide therapeutic decisions, and optimize long-term follow-up.

Keywords: cytomegalovirus; congenital infection; postnatal infection; prematurity; HIV; ganciclovir.

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INTRODUCTION

Human cytomegalovirus (CMV), a ubiquitous member of the Herpesviridae family, establishes lifelong latent infection. Congenital CMV (cCMV) is the most common congenital viral infection and is a major cause of non-genetic sensorineural hearing loss and neurodevelopmental disability. Clinical presentation is highly variable: most infected infants are asymptomatic at birth, whereas a minority have symptomatic disease characterized by combinations of growth restriction, petechiae, jaundice, hepatosplenomegaly, cytopenias, microcephaly, neuroimaging abnormalities, chorioretinitis, and hearing impairment [1–4].

Distinguishing congenital from postnatally acquired CMV infection depends largely on the timing of virological detection. Recent European recommendations emphasize confirmation within the first three weeks of life, preferably by CMV PCR testing of saliva or urine, to establish congenital infection [1]. When CMV is detected later and no early neonatal specimen is available, attribution to postnatal acquisition remains probabilistic.

In very preterm and very-low-birth-weight infants, postnatal acquisition particularly through breast milk from a CMV-seropositive mother may result in symptomatic disease. Reported manifestations include a sepsis-like syndrome, respiratory distress or pneumonitis, apnea, thrombocytopenia, neutropenia, and hepatic involvement, with greater severity at lower gestational ages [5,6].

Immunodeficiency represents another distinct clinical setting. HIV coinfection may facilitate substantial CMV replication and severe visceral disease, while making causal interpretation difficult because other infections may coexist.

We report seven CMV infections diagnosed in the neonatal intensive care unit of Hassan II University Hospital in Fez, Morocco. Our objective was to characterize their clinical and outcome profiles and to discuss the diagnostic challenges involved in distinguishing congenital disease from presumed postnatal infection.

Patients and Methods

This was a retrospective, single-center descriptive study conducted over a two-year period in the neonatal intensive care unit of Hassan II University Hospital, Fez, Morocco.

We included seven infants in whom CMV infection was confirmed by blood CMV PCR during clinical care. Data were extracted from medical records using a standardized data collection form.

Collected variables included maternal and obstetric information, gestational age, birth weight, sex, age at diagnosis, neurological and sensory manifestations, hematological abnormalities, hepatic involvement, respiratory and gastrointestinal manifestations, CMV viral load when available, imaging findings, antiviral therapy, length of hospitalization, and outcome.

The infant who presented from birth with microcephaly, major neuroimaging abnormalities, and neurosensory involvement was classified as having symptomatic congenital CMV disease. For patients diagnosed later, systematic CMV PCR testing at birth had not been performed; therefore, congenital infection could not be formally excluded. The term “presumed postnatal CMV infection” was used when the clinical context supported postnatal acquisition.

The two very-low-birth-weight preterm infants (1,200 and 1,400 g) had initially been admitted for prematurity, with or without respiratory distress, and had no reported congenital malformation syndrome. Both received breast milk and had undergone multiple transfusions. Infants with confirmed HIV infection were formula-fed or mixed-fed.

Results

General characteristics

The series included five boys and two girls. Gestational age ranged from 29 to 40 weeks and birth weight from 1,200 to 3,400 g. Two infants had a birth weight below 1,500 g. CMV was diagnosed between day 17 of life and 3 months of age. Three patients had confirmed HIV infection by PCR.

Table 1: Individual characteristics of the seven patients

Case	Sex	Gestational age	Birth weight (g)	Age at diagnosis	Classification	Predominant presentation
1	M	34 weeks	1400	34 days	Presumed postnatal	Prematurity; pneumonia; thrombocytopenia and anemia
2	M	34 weeks + 4 days	2300	40 days	Symptomatic neonatal form	Jaundice/hepatic involvement; high viral load; polymorphic skin lesions
3	F	39 weeks	2900	60 days	Presumed postnatal; HIV+	Pneumonia; thrombocytopenia; anemia
4	M	38 weeks	3100	40 days	Presumed postnatal; HIV+	Hypotonia; anemia; pneumonia; ventilation
5	M	40 weeks	3400	3 months	Presumed postnatal; HIV+	Pneumonia; anemia; thrombocytopenia; neutropenia; death
6	M	Term	2300	17 days	Symptomatic congenital	Microcephaly; hypotonia; neurosensory impairment; chorioretinitis; cerebral malformations
7	F	29 weeks	1200	60 days	Presumed postnatal	Very preterm birth; thrombocytopenia and anemia

Clinical and laboratory manifestations

Hematological manifestations were frequent: five patients had anemia and four had thrombocytopenia; neutropenia was documented in one patient. The lowest values recorded on the data-collection form were a hemoglobin level of 6.8 g/dL and a platelet count of 11,000/mm³. Four patients had pneumonia and required oxygen therapy; two required mechanical ventilation.

Neurological involvement was particularly marked in the documented congenital form: microcephaly, hypotonia, psychomotor delay, reported absence of auditory response,

visual impairment, and chorioretinitis. Brain imaging showed lissencephaly associated with agenesis of the corpus callosum and posterior fossa hypoplasia, with intraventricular dilatation and intraparenchymal and periventricular calcifications.



Figure 1: Photographs showing cutaneous lesions associated with CMV infection (Patient 2)

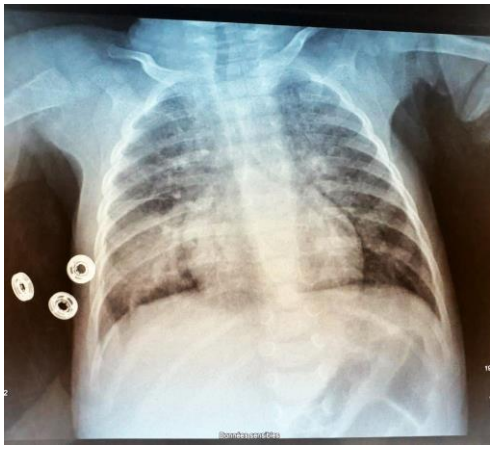


Figure 2: Radiological imaging showing pulmonary involvement (pneumonia) in the setting of CMV infection (Patient 5)

Table 2. Main manifestations observed in the series

Manifestation	Frequency	Details
Anemia	5/7	Cases 1, 3, 4, 5 and 7
Thrombocytopenia	4/7	Cases 1, 3, 5 and 7
Neutropenia	1/7	Case 5
Pneumonia	4/7	Cases 1, 3, 4 and 5
Oxygen therapy	4/7	Cases 1, 3, 4 and 5
Mechanical ventilation	2/7	Cases 4 and 5
Microcephaly	1/7	Case 6
Chorioretinitis	1/7	Case 6
Major cerebral abnormalities	1/7	Case 6: lissencephaly, agenesis of the corpus callosum, posterior fossa hypoplasia with triventricular dilatation and intraparenchymal/periventricular calcifications
Confirmed HIV infection	3/7	Cases 3, 4 and 5
Death	1/7	Case 5: respiratory distress



Figure 3 : CT-Scan image showing intraparenchymal/periventricular calcifications (Patient 6)

Virological data, treatment, and outcomes

CMV PCR was performed on blood in all seven patients. Viral load was available for several cases, with particularly high values in case 2 (approximately 1.36×10^6 according to the data-collection form) and case 5 (approximately 10^6). All patients received ganciclovir-based antiviral therapy, with oral step-down therapy in several cases. Reported treatment durations ranged from 20 days to 6 months. Several patients had a favorable or improving course. One patient was lost to follow-up, and one patient with HIV-CMV coinfection died from respiratory distress.

Table 3: Virological data, treatment, and outcomes

Case	PCR	Viral load	Treatment	Duration	Hospitalization	Outcome
1	Blood	1,910 copies	IV ganciclovir	6 weeks	3 months	Good outcome
2	Blood	1,357,239 IU/mL	IV ganciclovir	4 weeks	2 months	Reported clinical recovery
3	Blood	≈100,007 copies	IV ganciclovir then oral step-down	20 days	1 month	Follow-up ongoing; HIV-CMV
4	Blood	Not reported	Antiviral (record: ganciclovir)	6 months	10 days	Lost to follow-up
5	Blood	≈1,000,000 IU/mL	IV ganciclovir	3 weeks	1 month	Death from respiratory distress
6	Blood	13,574 IU/mL	IV ganciclovir then oral step-down	6 months	20 days	Follow-up ongoing
7	Blood	Not reported	IV ganciclovir then oral step-down	6 months	15 days	Improvement

Notable clinical profiles

Neurological congenital form. Case 6, diagnosed on day 17, was a term newborn with a birth weight of 2,300 g. The infant had microcephaly, hypotonia, psychomotor delay, and sensory impairment. Chorioretinitis was documented. Imaging demonstrated lissencephaly, agenesis of the corpus callosum, and a posterior fossa abnormality. This phenotype is consistent with congenital disease involving the central nervous system.

Multisystem neonatal form. Case 2, born at 34 weeks + 4 days, was described in the detailed clinical observation with an initial gastrointestinal context, followed by cholestatic jaundice and polymorphic skin manifestations. The data-collection form documented substantial hepatic involvement and a high viral load. In the absence of virological sampling during the first three weeks, congenital versus postnatal origin cannot be established retrospectively with certainty.

Prematurity and presumed postnatal CMV. Cases 1 and 7 had birth weights of 1,400 and 1,200 g. They had initially been hospitalized for prematurity, received breast milk, and underwent multiple transfusions. The subsequent occurrence of cytopenias, associated with respiratory involvement in case 1, is compatible with presentations described after postnatal CMV acquisition in preterm infants. No intraventricular hemorrhage was reported in the 1,200-g infant.

HIV-CMV coinfection. Three patients had positive HIV PCR. Presentations were dominated by respiratory and/or hematological involvement. The most severe case combined pneumonia, anemia, thrombocytopenia, neutropenia, and a high CMV viral load, with death from respiratory distress.

Table 4. Comparison of the three clinical profiles observed

Characteristic	Symptomatic congenital CMV	Multisystem neonatal form	Presumed postnatal CMV
Number in series	1	1	5
Predominant setting	Term newborn	Moderate prematurity	Prematurity or HIV immunodeficiency
Time of diagnosis	Day 17	Day 40	Day 34 to 3 months
Predominant involvement	Neurological and sensory	Hepatic / multisystem	Respiratory and hematological
Characteristic signs	Microcephaly, chorioretinitis, cerebral malformations	Jaundice, cytolysis, skin manifestations, high viral load	Pneumonia, anemia, thrombocytopenia; neutropenia in severe disease
Diagnostic issue	Early confirmation compatible with CMV	Congenital versus postnatal origin uncertain	Presumed postnatal due to lack of early neonatal PCR
Outcome	High risk of neurosensory sequelae	Favorable under antiviral therapy according to record	Variable; one death in the HIV-CMV group

DISCUSSION

Our series highlights the marked heterogeneity of CMV infection encountered in a neonatal intensive care setting. Three profiles emerged: severe neurological congenital disease, a multisystem neonatal form with predominant hepatic involvement, and infections diagnosed secondarily in vulnerable patients, mainly preterm infants or patients infected with HIV.

The congenital form in our series illustrates the cerebral tropism of CMV. Neuroimaging abnormalities, microcephaly, ocular involvement, and hearing impairment are among the findings most frequently used to define moderate-to-severe symptomatic congenital disease [1,3,4].

Lissencephaly and agenesis of the corpus callosum reflect early disruption of brain development and expose the infant to a substantial risk of neurodevelopmental sequelae. This profile warrants prolonged neurological, auditory, and ophthalmological follow-up.

Etiological diagnosis should be established early. The 2024 ECCI recommendations advocate testing for CMV in saliva or urine during the first three weeks of life to confirm congenital origin [1]. In our series, the absence of systematic screening at birth represents the main methodological difficulty: in patients diagnosed on days 34, 40, 60, or later, a positive PCR proves infection but cannot by itself date acquisition. Therefore, the term “presumed postnatal” is more rigorous than “confirmed postnatal.”

In preterm infants, postnatal acquisition through breast milk is well documented. Infants with very low birth weight and lower gestational age are most exposed to symptomatic disease. A recent review emphasizes that postnatal CMV may be silent or cause disease ranging from a moderate presentation to a severe, sometimes fatal, syndrome, and that data on long-term sequelae remain heterogeneous [5]. In our series, the two 1,200- and 1,400-g infants received breast milk. This exposure makes breast-milk transmission plausible, although it cannot be demonstrated because the virological status of breast milk was unavailable.

Transfusions represent another potential exposure, but interpretation depends on transfusion practices, particularly leukoreduction and the CMV status of blood products [11]. The two preterm infants had undergone multiple transfusions; in the absence of detailed information on the products used, no transmission route can be favored with certainty.

The respiratory and hematological profile observed is consistent with descriptions of postnatal CMV in preterm infants. In our series, four patients had pneumonia, four required oxygen, two required mechanical ventilation, five had anemia, and four had thrombocytopenia. These abnormalities are not specific to CMV, explaining why PCR was requested in the setting of persistent or unexplained respiratory, hematological, or hepatic presentations.

HIV-CMV coinfection represented another important profile: three of the seven patients had

confirmed HIV infection. Immunodeficiency complicates interpretation of CMV positivity because viral replication may accompany visceral disease or coexist with other infections. The death observed in this subgroup, in the context of respiratory distress and pancytopenia, underscores the potential severity of the association but does not allow mortality to be attributed to CMV alone in a retrospective series [12].

Regarding treatment, the strongest evidence concerns symptomatic congenital disease, particularly when the central nervous system is involved. Valganciclovir administered for six months is the reference strategy in eligible symptomatic forms, with monitoring of the complete blood count and liver function tests [1,2,7]. Indications for and duration of treatment of symptomatic postnatal infection in preterm infants are less standardized; recent literature continues to highlight the lack of robust data defining a universal regimen [5].

In our series, all patients received ganciclovir, with oral step-down therapy in several of them. This therapeutic homogeneity should be interpreted cautiously: the study is descriptive and cannot assess comparative treatment efficacy or establish a causal relationship between antiviral therapy and outcome.

Beyond the clinical description, our series highlights an organizational issue: implementing a targeted early screening strategy in newborns with signs compatible with CMV or belonging to at-risk groups could improve differentiation between congenital infection and postnatal acquisition. Targeted and universal screening programs are currently receiving increasing attention [3,4].

Strengths of the study

This study has several important strengths. It represents the first case series from our center describing neonatal CMV infections in seven newborns and provides a comprehensive characterization of the clinical spectrum of CMV infection in a neonatal intensive care setting. The study includes diverse clinical presentations, ranging from symptomatic congenital infections with severe neurological and sensory involvement to multisystem neonatal forms and presumed postnatal infections in vulnerable populations, including preterm infants and HIV-infected newborns. A major strength is that all included patients had CMV PCR confirmation, ensuring reliable virological diagnosis and strengthening the accuracy of case identification. Furthermore, the integration of clinical, biological, virological, radiological, therapeutic, and outcome data provides a comprehensive real-world assessment of neonatal CMV infection. The cautious interpretation of infection timing, considering the absence of systematic CMV PCR screening during the first three weeks of life, reduces the risk of misclassification and enhances the methodological rigor and relevance of our findings.

Limitations of the study

Several limitations of this study should be acknowledged. First, the relatively small sample size and single-center design may limit the generalizability of our findings. Second, the retrospective design may have resulted in missing data and variability in the investigations performed.

The main limitation concerns the timing of CMV infection. The absence of systematic CMV screening at birth, together with the lack of PCR results obtained during the first three weeks of life in most patients, prevented definitive confirmation of a postnatal origin in cases diagnosed at a later stage.

In addition, CMV testing of breast milk and the virological characteristics of transfused blood products were unavailable, precluding definitive identification of the route of transmission in preterm infants. Finally, long-term auditory and neurodevelopmental follow-up was incomplete, limiting the assessment of potential late sequelae.

CONCLUSION

This seven-case series illustrates the clinical continuum of CMV infections observed in a neonatal intensive care setting, from severe neurological congenital disease to respiratory and hematological presentations diagnosed secondarily in preterm infants and patients infected with HIV. CMV should be considered in the presence of microcephaly or unexplained cerebral abnormalities, but also in persistent pneumonia, cytopenias, or hepatic involvement in a vulnerable newborn. Virological confirmation during the first three weeks of life, when indicated, is essential to distinguish congenital infection from postnatal acquisition. Prospective studies with early screening and standardized neurosensory follow-up are needed to better define the contribution of CMV in neonatal units.

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