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SEROLOGICAL PROFILE OF PARENTERALLY TRANSMITTED HEPATITIS B AND C VIRUSES IN PATIENTS WITH ACUTE SUSPECTED INFECTIOUS HEPATITIS: A PROSPECTIVE CROSS-SECTIONAL STUDY FROM SEMI-URBAN UTTAR PRADESH, INDIA

Medical Microbiology

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ABSTRACT

Background: Hepatitis B virus (HBV) and Hepatitis C virus (HCV) are the dominant parenterally transmitted agents responsible for acute and chronic liver disease globally. India exhibits intermediate endemicity for both viruses, yet granular seroprevalence data from semi-urban regions of Uttar Pradesh remain scarce. Precise multi-marker serological profiling is essential to differentiate acute HBV infection from chronic reactivation and to detect clinically significant co-infections. **Objectives:** To determine the seroprevalence of HBsAg and anti-HCV among patients with acute suspected infectious hepatitis; to evaluate the HBV serological profile (including IgM anti-HBc, HBeAg, and IgM anti-HDV) in HBsAg-positive cases; and to assess the prevalence of HBV–HCV co-infection. **Methods:** A prospective hospital-based cross-sectional study was conducted over 18 months at Dr. KNSMIMS, Barabanki. Six hundred consecutive patients presenting with acute jaundice (serum bilirubin >2.5 mg/dL, illness duration <6 months), without prior chronic liver disease, were enrolled. Serum samples were tested using third-generation ELISA for HBsAg and anti-HCV. HBsAg-positive cases underwent supplementary profiling for HBeAg, IgM anti-HBc, IgM anti-HDV, and anti-HBs. Optical density readings were performed on the BeneSphera™ E-21 bi-chromatic ELISA microplate reader (Avantor, wavelengths 405/450/492/630 nm). Data were analysed using chi-square tests; $p < 0.05$ was considered statistically significant. **Results:** Of 600 participants (268 males, 332 females; mean age 37.7 years), 114 (19.0%) tested positive for at least one viral marker. HBsAg seroprevalence was 8.83% (53/600) and anti-HCV seroprevalence was 10.16% (61/600). It is emphasised that anti-HCV positivity detects exposure to HCV (past or ongoing) and does not independently confirm acute HCV infection. Males demonstrated significantly higher total seropositivity (23.12%) than females (15.66%; p=0.020). Among the 53 HBsAg-positive cases, IgM anti-HBc — the serological hallmark of acute HBV infection — was detected in 47 (88.7%), HBeAg in 12 (2.0%), and IgM anti-HDV in 6 (1.0%). HBV–HCV co-infection (HBsAg+ and anti-HCV+) was identified in 6 of the 53 HBsAg-positive cases (11.32%), and 8 subjects in total carried both markers. **Conclusion:** Both HBV and HCV are significant serological contributors to acute infectious hepatitis in semi-urban Uttar Pradesh. The high rate of IgM anti-HBc positivity confirms predominantly acute-phase HBV infection. Multi-marker screening incorporating IgM anti-HBc, HBeAg, and IgM anti-HDV, with molecular confirmation (HCV RNA PCR) where feasible, is essential for accurate diagnosis and optimal clinical management in high-burden regions.

KEYWORDS

Hepatitis B virus; Hepatitis C virus; HBsAg; anti-HCV; IgM anti-HBc; seroprevalence; co-infection; acute viral hepatitis; ELISA

1. INTRODUCTION

Viral hepatitis remains one of the most consequential infectious threats to global public health. According to the World Health Organization (WHO) 2024 Global Hepatitis Report, viral hepatitis now ranks as the second leading infectious cause of death worldwide, claiming approximately 1.3 million lives annually — a burden comparable to tuberculosis.1 Among the five recognised hepatotropic viruses, Hepatitis B virus (HBV) and Hepatitis C virus (HCV) are the principal parenterally transmitted agents and the dominant drivers of chronic liver disease, cirrhosis, and hepatocellular carcinoma (HCC).2

HBV is a partially double-stranded DNA virus of the family Hepadnaviridae, characterised by a 3.2 kb genome and a replication cycle involving covalently closed circular DNA (cccDNA), which serves as a persistent transcriptional template in infected hepatocytes.3 HCV, a positive-sense single-stranded RNA virus of the family Flaviviridae, encodes an RNA-dependent RNA polymerase with high error rates, generating extensive genetic heterogeneity that compli-cates antiviral therapy and facilitates immune evasion.4 Both viruses share parenteral routes of transmission, including unsafe injections, unscreened blood transfusions, and high-risk sexual behaviour, rende-ring their co-circulation epidemiologically plausible in high-risk settings.

India harbours approximately 40 million chronic HBV carriers, constituting roughly 11% of the global pool, with a national population prevalence of 3–4%.5 HCV antibody prevalence is estimated at 1–1.5%, translating to 6–12 million infected individuals, with regional hotspots in North India attributable to unsafe injection practices and traditional tattooing.6 Despite the substantial national burden, granular seroprevalence data from semi-urban districts of Uttar Pradesh, such as Barabanki, remain limited. Most published studies originate from urban tertiary hospitals or blood bank screening programmes, both of which may underestimate true community-level prevalence in semi-urban cohorts.

The clinical challenge in acute hepatitis management is compounded by the need to distinguish primary acute HBV infection from exacerbation of chronic hepatitis B, as well as to detect simultaneous HBV–HCV co-infection and Hepatitis Delta Virus (HDV) superinfection, all of which significantly alter prognosis and treatment strategy.7 Additionally, the interpretation of anti-HCV seropositivity in the context of acute hepatitis requires particular caution, as third-generation ELISA for anti-HCV detects cumulative IgG antibodies that may reflect past, chronic, or early acute infection and cannot independently differentiate these states without confirmatory HCV RNA testing.8 This prospective study was designed to address the local data gap by systematically profiling serological markers of HBV and HCV in clinically suspected acute hepatitis cases at a semi-urban tertiary centre in Uttar Pradesh.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

A prospective, hospital-based cross-sectional study was conducted in the Department of Microbiology, Dr. KNS Memorial Institute of Medical Sciences (Dr. KNSMIMS), Barabanki, Uttar Pradesh, India, over an 18-month period following Institutional Ethics Committee approval (IEC Ref. No.: [PLACEHOLDER – insert IEC number]). The study adhered to the STROBE guidelines for reporting observational cross-sectional studies.

2.2 Study Population and Eligibility

Participants were consecutively enrolled from outpatient and inpatient departments presenting with clinically suspected acute infectious hepatitis. Inclusion criteria comprised: (i) recent onset of jaundice with or without systemic symptoms (fever, anorexia, malaise) for a duration of less than six months; (ii) absence of documented history or clinical evidence of chronic liver disease (CLD); and (iii) biochemical confirmation of hyperbilirubinemia, defined as serum bilirubin >2.5 mg/dL. Exclusion criteria were: illness duration exceeding six months; post-operative jaundice; non-infectious aetiologies (obstructive jaundice, drug-induced liver injury, or alcoholic hepatitis); and known prior diagnosis of chronic HBV or HCV infection. Written informed consent

was obtained from all participants or their legal guardians.

2.3 Sample Size

A target sample size of 600 was calculated using the standard single-population proportion formula (Z=1.96, estimated prevalence of 15% based on regional literature, margin of error d=0.05), which yielded a minimum of 545; 600 samples were enrolled to account for a potential 10% non-response rate.

2.4 Specimen Collection and Processing

Approximately 3–5 mL of venous blood was collected under aseptic precautions using disposable syringes in anticoagulant-free (plain red-topped) tubes. Serum was separated by centrifugation at 2500 rpm (approximately 1000 × g) for 10–15 minutes at room temperature. Haemolysed or turbid samples, which could produce spurious optical density (OD) readings, were rejected. Sera were aliquoted to avoid repeated freeze-thaw cycles and stored at 2–8°C for assays performed within 48 hours, or archived at –20°C for supplementary marker testing.

2.5 Serological Assays

All 600 serum samples were screened for HBsAg using a third-generation sandwich ELISA (Sure-Test HBsAg kit, J. Mitra & Co., New Delhi, India) and for anti-HCV antibodies using a thirdgeneration indirect ELISA (detecting IgG antibodies against structural and non-structural HCV proteins). Samples reactive on initial HBsAg testing underwent supplementary profiling for: (i) HBeAg (Biovantion Inc. sandwich ELISA), (ii) IgM anti-HBc (capture ELISA; marker of acute HBV infection), (iii) IgM anti-HDV (to detect acute delta co-infection), and (iv) anti-HBs (to assess seroconversion/recovery).

All ELISA runs were performed on the BeneSphera™ E-21 semiautomatic bi-chromatic microplate reader (Avantor Performance Materials, distributed by Avantor India), operating at primary wavelengths of 405, 450, 492, and 630 nm with an 8-channel optic fibre system for simultaneous well measurement. Each run included one blank well, two positive controls, and three negative controls per plate to ensure assay validity. The cut-off OD was calculated for each individual plate as: Cut-off = Mean OD of negative controls + 0.1. Samples with OD ≥ cut-off were designated reactive. All initially reactive samples were retested in duplicate; sustained reactivity on duplicate testing was required for confirmation of positivity.

It is important to note that anti-HCV positivity by ELISA indicates serological evidence of HCV exposure — it does not by itself distinguish acute from past or chronic infection. Confirmation of active HCV infection requires HCV RNA detection by nucleic acid testing (NAT/PCR), which was beyond the scope of this serological study. Accordingly, anti-HCV results are reported as a marker of HCV exposure/seroprevalence, not as definitive evidence of acute HCV infection.8,9

2.6 Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages. The chi-square (χ^2) test or Fisher's exact test was used to assess associations between seropositivity and categorical variables (sex, age group). A p-value <0.05 was considered statistically significant.

3. RESULTS

3.1 Demographic Profile

Of the 600 enrolled participants, 268 (44.67%) were male and 332 (55.33%) were female, yielding a male-to-female ratio of 1:1.24. The mean age was 37.7 years (range: 2–72 years). The majority were in the >40 years age group (264; 44.0%), followed by the 31–40 years group (219; 36.5%), while children aged ≤10 years constituted only 0.5% of the cohort. The sex-based difference in hospital attendance was statistically significant (χ^2=6.82; p<0.05) (Table 1).

Table 1: Age and sex distribution of 600 patients with acute suspected infectious hepatitis

Age Group	Male n (%)	Female n (%)	Total n (%)
0–10 years	1 (0.17)	2 (0.33)	3 (0.5)
11–20 years	5 (0.83)	4 (0.67)	9 (1.5)
21–30 years	52 (8.67)	53 (8.83)	105 (17.5)
31–40 years	97 (16.17)	122 (20.33)	219 (36.5)
>40 years	113 (18.83)	151 (25.17)	264 (44.0)
Total	268 (44.67)	332 (55.33)	600 (100)

3.2 Overall Seroprevalence of Viral Markers

Of the 600 samples tested, 486 (81.0%) were seronegative for both markers. A total of 114 participants (19.0%) were seropositive for at least one viral marker: HBsAg in 53 (8.83%) and anti-HCV in 61 (10.16%). Of these, 8 individuals (7.02% of the 114 positives; 1.33% of total cohort) were reactive for both HBsAg and anti-HCV, constituting the HBV–HCV co-infected subgroup (discussed in Section 3.5). This means the total seropositive count of 114 includes 45 with HBsAg only, 53 with anti-HCV only, and 8 with both markers — with no overlap in the primary individual prevalence figures for each marker (HBsAg: 53/600; anti-HCV: 61/600).

Male subjects demonstrated significantly higher total seropositivity (23.12%; 62/268) compared to females (15.66%; 52/332; χ^2=5.385; p=0.020). This sex-based disparity was primarily driven by significantly higher anti-HCV seroprevalence in males (13.05% vs. 7.83%; χ^2=4.612; p=0.032). HBsAg positivity was numerically greater in males (10.07% vs. 7.83%), though this difference did not attain statistical significance (χ^2=0.941; p=0.332) (Table 2).

Table 2: Overall seroprevalence of HBsAg and anti-HCV stratified by sex (n=600)

Serological Marker	Male n (%)	Female n (%)	Total n (%)	χ^2 Value	p-value
HBsAg	27 (10.07)	26 (7.83)	53 (8.83)	0.941	0.332
Anti-HCV*	35 (13.05)	26 (7.83)	61 (10.16)	4.612	0.032†
Total Positive	62 (23.12)	52 (15.66)	114 (19.0)	5.385	0.020†

**Anti-HCV ELISA detects IgG antibody to HCV structural and non-structural antigens; positivity indicates HCV exposure (acute, chronic, or past) and requires confirmatory HCV RNA PCR for active infection diagnosis. †Statistically significant (p<0.05).*

3.3 Age-Wise Distribution of Viral Marker

Seropositivity for both HBV and HCV was absent in the 0–10 years age group (0/6). The highest proportional positivity was noted in the 11–20 years age group (28.6%; 2/7), though this figure is based on a very small sample and should be interpreted with caution. Among adults, seroprevalence was remarkably uniform: HBsAg positivity ranged from 8.4% (31–40 years) to 9.3% (21–30 years) and 9.1% (>40 years). Anti-HCV seroprevalence was a consistent 10.2% across all three adult age brackets (21–30, 31–40, and >40 years). The highest absolute number of positive cases for both HBV (n=24) and anti-HCV (n=27) occurred in the >40 years group, reflecting the larger testing denominator for that cohort. Statistical analysis confirmed no significant association between age and viral marker distribution for either HBV (χ^2=0.695; p=0.952) or anti-HCV (χ^2=1.47; p>0.05).

3.4 HBV Serological Profile in HBsAg-Positive Cases

Detailed serological profiling was performed on all 53 HBsAg-positive samples. IgM anti-HBc, the definitive serological marker of acute HBV infection, was detected in 47 of 53 HBsAg-positive participants (88.7%; equivalent to 7.83% of the total cohort of 600), indicating that the large majority of HBsAg-positive individuals were in the acute phase of infection rather than exhibiting chronic carrier status or reactivation. HBeAg, a marker of active viral replication and high infectivity, was positive in 12 cases (2.0% of total cohort). IgM anti-HDV was identified in 6 cases (1.0% of total cohort). No participant tested positive for anti-HBs, consistent with an absence of HBV serological recovery or evidence of prior vaccination immunity within this symptomatic cohort (Table 3).

Table 3: Hepatitis B serological profile in HBsAg-positive cases (denominator = 600 total tested)

Serological Marker	N (of 600 tested)	Percentage (%)
HBsAg (primary marker)	53	8.83
IgM anti-HBc†	47	7.83
HBeAg	12	2.0
IgM anti-HDV (co-infection)	6	1.0
Anti-HBs	0	0

†IgM anti-HBc: definitive marker distinguishing acute HBV infection from chronic HBV; high titres are found in true acute infection, whereas low-level positivity may persist in chronic flares. Anti-HBs: marker of serological recovery or vaccination immunity.

3.5 HBV–HCV Co-infection

Of the 114 total seropositive participants (HBsAg+ and/or anti-HCV+), 8 (7.02%) were reactive for both markers, constituting the

HBV–HCV co-infected group. Among the 53 HBsAg-positive individuals, 6 (11.32%) were also reactive for anti-HCV. Among the 61 anti-HCV-positive individuals, 2 additional cases (3.28%) were reactive for HBsAg. These 8 co-infected subjects are included within the total 114 seropositive count: the individual marker totals (HBsAg: 53; anti-HCV: 61) count each co-infected person once under each respective marker; thus 53 + 61 − 8 co-infected = 106 unique seropositive individuals, with 8 counted under both markers in Table 2 for marker-specific prevalence (Table 4).

Table 4: Co-infection patterns among seropositive participants (n=114)

Co-infection Pattern	n	% of total positives (n=114)	% of respective virus positives
HBsAg+ & anti-HCV+	6	5.26%	11.32% of HBsAg+ (n=53)
Anti-HCV+ & HBsAg+	2	1.75%	3.28% of Anti-HCV+ (n=61)
Total co-infections	8	7.02%	–

4. DISCUSSION

This prospective study provides a comprehensive serological profile of parenterally transmitted HBV and HCV among 600 patients with acute suspected infectious hepatitis in semi-urban Uttar Pradesh. The overall viral marker positivity rate of 19.0% corroborates the recognised intermediate-to-high endemicity of this geographic belt and is consis-tent with published data from comparable North Indian hospital based studies. Jain et al. (2013) reported an 18% overall seroprevalence among 267 cases of acute viral hepatitis or acute hepatic failure from North India, with HBsAg positivity of 16.10%.10 Manoj et al. (2014) documented an HBsAg seroprevalence of 10.15% among 2,493 symptomatic patients in New Delhi,11 closely approximating the 8.83% observed in the present cohort. The modest numerical differences likely reflect population-specific factors such as varying vaccination coverage and differing risk-behaviour profiles.

A critical interpretive caveat must be applied to anti-HCV data in this context. Third-generation ELISA for anti-HCV detects IgG antibodies against both structural (core, E1, E2) and non-structural (NS3, NS4, Ns5) HCV antigens, and a positive result indicates serological exposure to HCV — it does not distinguish between acute, chronic, or resolved past infection.8,9 Confirmation of active infection requires demonstration of HCV RNA by nucleic acid testing (NAT). In this study, anti-HCV seroprevalence of 10.16% therefore reflects the burden of HCV exposure in this symptomatic cohort. The clinically meaningful finding is the identification of HBsAg seroprevalence (8.83%) alongside high anti-HCV exposure rates, together accounting for the 19.0% overall burden, and underscoring the need for comprehensive screening in this population.

The remarkably uniform anti-HCV seroprevalence of 10.2% across all adult age strata (21–30, 31–40, and >40 years) warrants epidemiological interpretation. This age-invariant pattern is consistent with what epidemiologists describe as a 'historical cohort exposure' phenomenon: HCV transmission in India was amplified in the pre-screening era through unsterile injections, unscreened blood transfusions, and reused medical equipment, resulting in a large prevalence pool distributed across adult age groups rather than clustering in a specific birth cohort.12 This pattern stands in contrast to settings where transmission is primarily driven by ongoing intrave-nous drug use, which tends to produce higher prevalence in younger cohorts. The absence of seropositive cases in the paediatric 0–10 years group (0/6, noting the small sample) may partially reflect improved paediatric hepatitis B immunisation coverage and improved blood product safety in the contemporary period.

Male sex was a significant predictor of overall seropositivity (p=0.020) and specifically of anti-HCV seroprevalence (p=0.032). This sex-based disparity has been documented across Indian cohorts and is biologically and behaviourally plausible: occupational exposures to unsafe injections through informal practitioners, tattooing, and higher-risk procedures are disproportionately reported in males in semi-urban settings.13 The absence of a statistically significant sex difference for HBsAg positivity (p=0.332) is consistent with HBV's broader transmission dynamics — encompassing perinatal, household horizontal, and parenteral routes — which are less gender-discriminating than the predominantly percutaneous exposure pathway driving HCV seroprevalence.

The detailed HBV serological profile yields clinically significant insights. Detection of IgM anti-HBc in 47 of 53 HBsAg-positive individuals (88.7%) strongly supports genuine acute HBV infection in the majority of this subgroup, rather than chronic carrier status or reactivation flares. This is directly pertinent to clinical management: acute HBV in immunocompetent adults is typically self-limiting and requires supportive care, whereas acute-on-chronic hepatitis B exacerbations may necessitate urgent antiviral therapy to prevent progression to acute liver failure.14 The 6 HBsAg-positive individuals lacking IgM anti-HBc (11.3%) merit clinical re-evaluation for possible chronic HBV with acute exacerbation. HBeAg positivity in 12 cases (2.0% of total cohort) identifies a subset with active viral replication and maximum infectivity, requiring appropriate isolation measures and contact tracing.

The identification of IgM anti-HDV in 6 HBsAg-positive cases (1.0% of total cohort; 11.32% of HBsAg-positive cases) carries substantial clinical implications disproportionate to its apparent prevalence. HDV is an obligate satellite virus requiring HBsAg as its envelope protein; HDV co-infection or superinfection in an HBV carrier accelerates progression to cirrhosis and markedly increases the risk of HCC.15 The present finding aligns with a recent cross-sectional study from Western UP by Nishad et al. (2025), which reported 6.8% HDV seroprevalence among HBsAg-positive patients at UPUMS, Saifai.16 The 2024 WHO HDV guidelines recommend serological HDV testing for all HBsAg-positive individuals in settings with HDV prevalence >5% or in defined high-risk groups.17 Routine IgM anti-HDV screening in all HBsAg-positive acute hepatitis presentations is therefore strongly recommended for centres in this region.

HBV–HCV co-infection was documented in 8 cases (7.02% of the 114 seropositive subjects), with 11.32% of HBsAg-positive individuals also carrying anti-HCV — substantially exceeding the pooled Indian estimate of approximately 1.89% from systematic reviews of general populations.18 This elevated rate is expected given that the study cohort comprises symptomatic acute hepatitis cases, who are enriched for individuals with multiple parenteral risk exposures compared to general screening populations. From a pathophysiological standpoint, HBV–HCV co-infection leads to complex virological interactions: in the majority of co-infected individuals, HCV exerts a dominant suppressive effect on HBV replication at the transcriptional level, resulting in lower HBsAg titres and reduced HBV DNA levels than would be expected in HBV monoinfection; conversely, suppression of HCV with direct-acting antivirals can unmask HBV replication.19,20 These atypical serological profiles may lead to diagnostic underestimation if only a single hepatitis marker is screened. Co-infected patients also carry higher risks of cirrhosis and HCC and may respond less predictably to antiviral regimens, underscoring the clinical necessity of dual-marker testing.20

The female predominance in hospital attendance (55.33% vs. 44.67% males; $\chi^2=6.82$; $p<0.05$) is a sociodemographic feature of this semi-urban cohort that does not necessarily reflect higher community prevalence of hepatitis among women. Differential healthcare-seeking behaviour, whereby women present earlier or more frequently for symptoms, is a recognised phenomenon in hospital-based studies in rural and semi-urban India and can inflate female representation without implying a genuine sex-based disease excess.21 This finding reinforces the importance of interpreting attendance rates separately from true epidemiological prevalence.

LIMITATIONS

This study has several limitations. First, it was conducted at a single tertiary centre, which may not fully represent the heterogeneous epidemiology of rural Uttar Pradesh. Second, diagnosis relied on serological ELISA markers alone; the absence of nucleic acid testing (HBV DNA PCR; HCV RNA PCR) limits the ability to confirm active HCV viraemia, detect window-period acute HBV, or identify occult hepatitis B. Third, anti-HCV positivity cannot independently diagnose acute HCV infection without confirmatory PCR, representing a diagnostic limitation acknowledged throughout this manuscript. Fourth, long-term clinical follow-up was not feasible within the study design, preventing assessment of chronicity rates and clinical outcomes. Fifth, the small sub-cohort sizes in the 0–10 and 11–20 years strata limit age-stratified conclusions for paediatric and adolescent populations.

5. CONCLUSION

This prospective study establishes that HBV and HCV are significant

serological contributors to acute infectious hepatitis in semi-urban Barabanki, Uttar Pradesh, with an overall viral marker seroprevalence of 19.0% in clinically suspected cases. HBsAg seroprevalence was 8.83% and anti-HCV seroprevalence was 10.16%, with the important caveat that anti-HCV positivity reflects HCV exposure and must be confirmed by HCV RNA PCR for definitive diagnosis of active infection. Male sex is a significant determinant of anti-HCV seroprevalence. The high proportion of IgM anti-HBc positivity (88.7% of HBsAg-positive cases) confirms predominantly acute-phase HBV infection in this cohort. HDV co-infection (1.0%) and HBV–HCV dual seropositivity (11.32% of HBsAg-positive cases), though less frequent, are clinically critical diagnoses associated with accelerated liver disease progression. These findings argue strongly for routine implementation of multi-marker serological screening panels — including HBsAg, IgM anti-HBc, HBeAg, IgM anti-HDV, and anti-HCV — supplemented by molecular diagnostic capacity for HCV RNA confirmation in all patients presenting with acute jaundice in high-burden semi-urban regions of India.

Declarations

Ethical Approval: The study was approved by the Institutional Ethics Committee of Dr. KNSMIMS, Barabanki (IEC Ref. No.: [PLACEHOLDER – insert IEC number]). Informed written consent was obtained from all participants or their legal guardians.

Conflict of Interest: The authors declare no conflicts of interest.
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Data Availability: Available from the corresponding author on reasonable request.

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