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MAGNETIC RESONANCE IMAGING

Evaluation of symmetrical increased echogenicity of bilateral caudothalamic grooves detected on cranial ultrasonography by comparing with susceptibility-weighted imaging

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Abstract

Objective To assess symmetrical increased echogenicity of bilateral caudothalamic grooves (SIEBCG) detected on newborn cranial ultrasonography (CUS) using magnetic resonance susceptibility-weighted imaging (SWI).

Materials and methods A total of 14 newborns (8 girls; 12 premature with mean gestational age of 30 weeks and 5 days, 2 mature) who were detected to have SIEBCG on routine serial CUS and underwent cranial magnetic resonance imaging (MRI) were recruited for the study. The cranial MRI examinations including SWI acquired on the same day of SIEBCG detection and serial CUS to assess the progress of SIEBCG lesions in the following 6 month period were retrospectively evaluated and compared for the presence of germinal matrix hemorrhage.

Results On SWI, solely one patient (7, 1%) had signal alteration on caudothalamic groove compatible with grade 1 germinal matrix hemorrhage. Two patients (14, 2%) had parenchymal (on cerebellar and parietal white matter) millimetric hemorrhagic foci. Seven patients (50%) had signs of presumptive hypoxic insult including hyperintense dots on centrum semiovale and periventricular white matter in five, and increased signal intensity on the globus pallidi in two, on T1-weighted images. Four patients (28, 6%) had normal findings. Of these, 10 patients became normal on follow-up CUS at postterm-equivalent age, whereas four were missing.

Conclusion Symmetrical increased echogenicity of bilateral caudothalamic grooves seen on newborn CUS may be the indicator of other pathologies as ischemic insult or focal parenchymal hemorrhage. In the presence of SIEBCG, further examination with SWI should be performed.

Keywords Germinal matrix hemorrhage · Ultrasound · Magnetic resonance imaging · Susceptibility-weighted imaging

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Introduction

Germinal matrix hemorrhage (GMH) is one of the most crucial complications encountered in preterm neonates that usually occurs during the first week of the life and tends to be unilateral [1–3]. The primary diagnostic tool to assess GMH is cranial ultrasonography (CUS) [1, 2, 4]. It is well known that GMH is initially detected on the caudothalamic groove and this region must be scanned thoroughly on CUS examinations [2]. Increased echogenicity limited to the caudothalamic groove is the manifestation of subependymal hemorrhage and graded as grade 1 GMH, according to Papile's classification [5]. However, increased echogenicity of the caudothalamic groove detected following first week of the life has been suggested to be an isolated finding or associated with non-hemorrhagic factors, as infection, prematurity, intrauterine growth retardation, and asphyxia. These lesions were described as bilaterally symmetrical teardrop-shaped echogenicities [4, 6–8]. However, the data of non-hemorrhagic origin have not been studied on a wide study group.

Magnetic resonance imaging (MRI) is considered as the gold-standard imaging method for evaluation of prematurity associated cranial pathologies. Furthermore, detectability of small intracranial hemorrhages has been improved with the implementation of susceptibility-weighted imaging (SWI) [2, 9]. To our knowledge solely in one article, addressing this issue, four infants who were detected to have such lesions were examined by MRI including T2*-weighted gradient-echo sequence and subependymal hemorrhage was excluded [8].

Though seldom, we came across symmetrical increased echogenicity of bilateral caudothalamic grooves (SIEBCG) on CUS examinations performed in our neonatal intensive care unit (NICU). In our institute, the neonates diagnosed with any parenchymal lesion on CUS usually undergo MRI with the exception of ventilator dependence or unstable clinical conditions. We aimed to retrospectively investigate whether SIEBCG is of hemorrhagic origin or associated with other intracranial pathologies by evaluating SWI with the contribution of other MRI sequences.

Materials and methods

The current study was institutional review board approved and compliant with the Declaration of Helsinki. Among 701 newborns examined with CUS in our NICU from July 2013 to January 2015, 20 newborns were retrospectively detected to have SIEBCG according to radiological database review. Six of these patients were excluded due to lack of additional MRI scans. Eventually, a total of 14 newborns [8 girls, 12 premature with gestational age between 26 to 35 weeks and 3 days (mean 30 weeks and 5 days), 2 mature with gestational age of 37 weeks and 6 days, 39 weeks] with the overall mean birth weight of 1532 g (range 800–2650 g) who were detected to have SIEBCG on CUS and underwent MRI on the same day of SIEBCG detection were included in the study.

In our NICU as a part of routine practice, serial CUSs were performed beginning from the first week of life until discharge. Follow-up CUSs after discharge were performed at around term equivalent or postterm-equivalent age. All CUSs in this patient group were performed by pediatric radiologists with one of two ultrasonography scanners (Acuson Antares; Siemens Medical Solutions, Mountain View, CA, USA or GE LOGIQ S7 Expert, GE Healthcare, WI, USA) using 4–10 MHz sector and 5–13 MHz linear transducers for the former and 3.6–10 MHz sector and 5–15 MHz linear matrix array transducers for the latter scanner. As the routine CUS protocol, sequential images were obtained on both sagittal and coronal planes via the anterior fontanel by using both of aforementioned sector and linear transducers.

Routine cranial MRI examinations were carried out on 1.5 Tesla MRI device (Siemens Aera; Siemens Medical Systems, Germany). The routine neonatal cranial MR imaging protocol consisted of turbo spin-echo T2-weighted imaging on axial and coronal planes [TR = 3800 ms, TE = 104 ms, number of slice (NS) = 20, slice thickness (ST) = 4 mm, FOV: 180 × 180 mm, resolution: 256 × 256], spin-echo T1-weighted imaging (TR = 800 ms, TE = 20 ms, ST = 4 mm, FOV: 150 × 200 mm, resolution: 192 × 256) on axial and sagittal planes, diffusion weighted imaging performed with single-shot spin-echo echoplanar sequence (TR = 3600 ms, TE = 83 ms, NS = 22, ST = 5 mm, FOV: 229 × 229 mm, resolution: 192 × 192, with two *b* values; 0, and 1000 s/mm^2), and SWI (TR = 49 ms, TE = 40 ms, ST = 3 mm, FOV: 162 × 200 mm, resolution: 364 × 448).

Both of CUS and MRI images for each patient were assessed by two pediatric radiologists (M.S.D. and S.D. with 5 and 10 years of postresidency experience, respectively) in consensus. Bilateral, teardrop-shaped, smoothly bounded, hyperechogenic lesions at caudothalamic grooves on CUS examinations were defined as SIEBCG (Fig. 1). Following the confirmation of SIEBCG lesions on CUS images, MRI examinations obtained on the same day of SIEBCG detection were evaluated. Hemorrhagic lesions on SWI and other accompanying pathological signal alterations on routine MRI sequences were noted. If discordance occurred in the interpretation of imaging findings, the investigators reassessed the examinations and reached a consensus. The detailed data about clinical progress of newborns in NICU were provided by two neonatologists (A.O. and L.K) depending on medical records review.

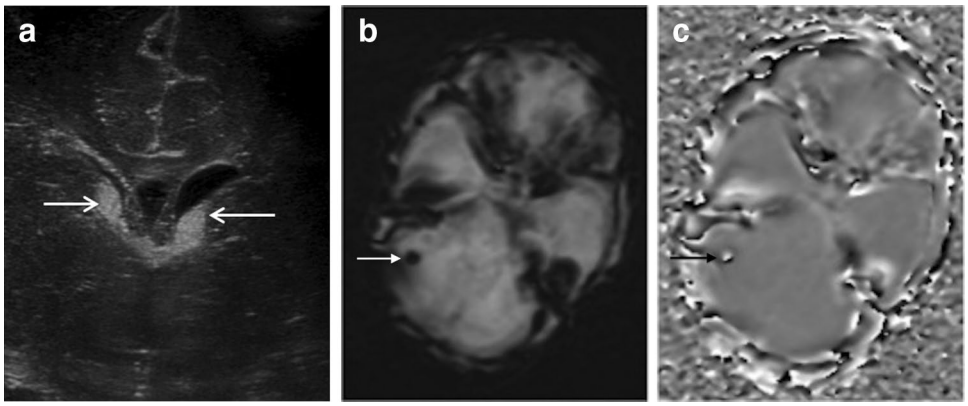

Fig. 1 Coronal image of SIEBCG (**a**, arrows) detected by CUS on 21th day of life in patient two. Images of SWI (**b**) and phase map (**c**) revealed right cerebellar millimetric hemorrhagic focus (arrows)

Results

12 newborns out of 14 were admitted to the NICU due to prematurity and associated clinical problems. These problems are listed as follows: Respiratory distress ($n = 7$, respiratory distress syndrome (RDS) in 6, transient tachypnea of the newborn and pneumothorax in 1 newborn), retinopathy of prematurity (ROP, $n = 6$), patent ductus arteriosus (PDA, $n = 5$), sepsis ($n = 4$), pneumonia ($n = 2$), necrotizing enterocolitis (NEC, $n = 2$), seizure ($n = 2$), patent foramen ovale (PFO, $n = 2$), coarctation of aorta ($n = 1$), ventricular septal defect (VSD, $n = 1$), supraventricular tachycardia ($n = 1$), disseminated intravascular coagulation (DIC, $n = 1$), and duodenal web ($n = 1$). Remaining two mature neonates were referred to the NICU with the diagnosis of meconium aspiration syndrome (MAS), small for gestational age (SGA), and hypoglycemic seizure in one and congenital pneumonia and pneumothorax in other neonate. In the course of hospitalization, 10 of the newborns needed mechanical ventilator support for up to 13 days. Five newborns had surgical operation due to duodenal web ($n = 1$), PDA ($n = 1$), aorta coarctation ($n = 1$), and ROP ($n = 3$).

Symmetrical increased echogenicity of bilateral caudothalamic grooves was detected by CUS on 2nd to 65th days of life, with a mean age of 28, 6 days, and 36th gestational weeks. In two patients, SIEBCG was detected on 2nd and in one patient on 9th days of life. SIEBCG was determined on more than 20 days following birth in the remaining 11 patients. Of these, five had no previous CUS examination in our clinic, whereas in six patients, previously performed CUS scans were reported to be normal. On SWI, solely one patient (7, 1%) had signal alteration at the region of caudothalamic groove consistent with grade 1 germinal matrix hemorrhage (Fig. 2), while on remaining 13, no pathological signal alteration was detected at the region of caudothalamic groove on any of the MRI sequences (Figs. 3 and 4). Two patients (14, 2%) had parenchymal [on cerebellar (Fig. 1) and parietal (Fig. 3) white matter] millimetric hemorrhagic foci. Seven patients (50%) had signs of probable hypoxic insult including hyperintense dots on centrum semiovale (Fig. 4) and periventricular white matter in five, and subtle increased signal intensity on the globus pallidi in two on T1-weighted images. Four patients (28, 6%) had normal MRI findings. In five patients, several microcysts within SIEBCG lesions were also noted on CUS examinations (Figs. 3 and 4). 10 out of 14 patients included in the study

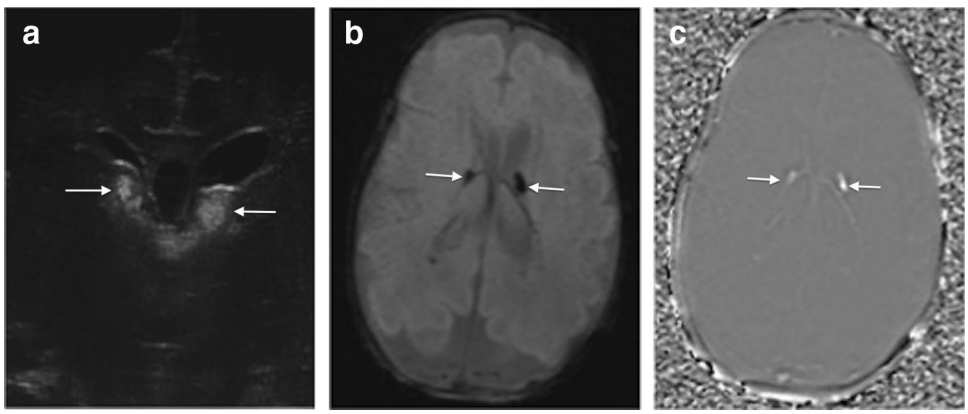

Fig. 2 Demonstration of SIEBCG detected by CUS (**a**, arrows) on 16th day of life in patient one. SWI (**b**) and phase map (**c**) showed bilateral hemorrhage on caudothalamic grooves (arrows)

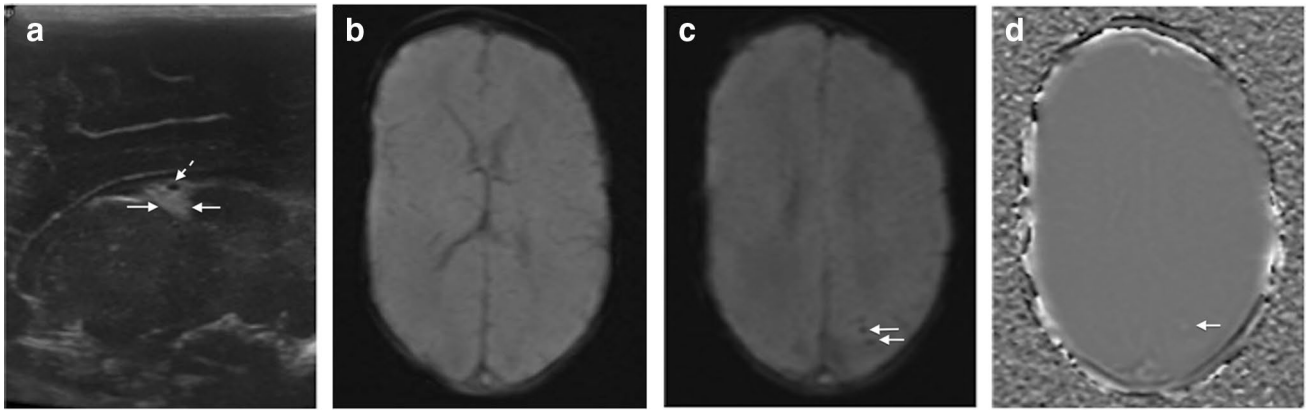

Fig. 3 SIEBCG (arrows) with a millimetric cyst (dashed arrow) was shown on 31th day of life in patient eleven on right parasagittal image (**a**). No evidence of hemorrhage was seen at the region of caudothalamic grooves on SWI (**b**), whereas focal millimetric areas of hemorrhage detected on left parietal white matter (**c**, **d**, arrows) on SWI and phase map images

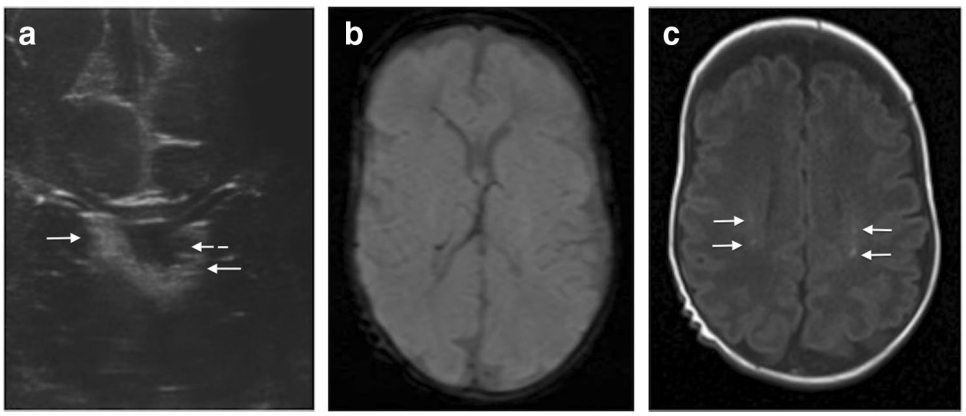

Fig. 4 SIEBCG was seen on coronal CUS (arrows) in premature newborn (patient five); microcyst was also noted on left caudothalamic groove (**a**, dashed arrow). No signal alteration indicating hemorrhage was detected on SWI (**b**). There were millimetric hyperintense foci on centrum semiovale on T1-weighted image (**c**, arrows)

had normal CUS scans on follow-up at postterm-equivalent age, whereas four were missing. Detailed clinical characteristics and imaging findings are given in Table 1.

Discussion

Our results showed that SIEBCGs detected on CUS in all patients, but one was not compatible with GMH on SWI. However, implementation of MRI including SWI sequence may further help for the demonstration of accompanying findings of probable ischemic insult and parenchymal hemorrhage. In the literature, there have been several studies addressing SIEBCG with different nomenclature as hyperechoic caudate nuclei, late germinal matrix hemorrhage-like lesions, and non-hemorrhagic germinal matrix echogenicity [6–8]. They were generally defined as bilateral, symmetrical, teardrop-shaped hyperechogenicities detected on caudothalamic grooves beyond the first week of life. Our findings were correlated with the literature in terms of SIEBCG definition. However, we detected SIEBCG lesions on the second day of life in two patients (patient 10 and patient 14) with the gestational age of 32 weeks and 2 days; 39 weeks and 2 days; respectively. Schlesinger et al. [6] and van Baalen et al. [8] reported SIEBCG in two of nine and one of five patients, respectively, in the first week of life. The detection time of these lesions according to gestation was stated as term, 30 weeks and 6 days and 34 weeks, respectively. In the literature, the overall mean detection time of the lesions was reported as near-term-equivalent period rather than early preterm period in terms of corrected gestational age [6–8]. In our study, SIEBCG was detected with a mean age of 28, 6 days (range 2–65 days) and 36th gestational weeks (range 31 weeks and 2 days–41 weeks). Due to broader range of detection time of SIEBCG reported in the current study, proposing a definitive detection time would not be feasible. The incidence of GMH is inversely related to gestational age in contrast to SIEBCG [1].

Smets et al. [10] reported the evolution steps of late neonatal onset subependymal echogenicities in the order of hyperechogenic stage, cyst formation, and disappearance on the follow-up CUS examinations. Unilateral microcysts

Table 1 Clinical characteristics, detection time of SIEBCG, and MRI findings

Patient	Gestational age at birth	Diagnosis	Detection time of SIEBCG on CUS	MRI findings
1	29 w + 2 d	PM, PDA, SVT, sepsis	16 d (31 w + 2 d)	Bilateral hemorrhage at caudothalamic grooves on SWI
2	29 w + 6 d	PM, PDA, PFO, grade 1 ROP, sepsis, DIC	21 d (32 w + 6 d)	Hemorrhagic focus at right cerebellar hemisphere on SWI
3	30 w	Duodenal web, grade 1 ROP, sepsis	65 d (39 w + 2 d)	Normal
4	37 w + 6 d	Congenital pneumonia, pneumothorax	22 d (41 w)	Hyperintense dots on right centrum semiovale and periventricular white matter on T1
5	27 w	PM, grade 3 ROP	53 d (34 w + 4 d)	Hyperintense dots on bilateral centrum semiovale on T1
6	26 w	PM, RDS, PDA, pneumonia, grade 3 ROP	59 d (34 w + 3 d)	Hyperintense dots on right centrum semiovale on T1
7	35 w	PM, CoA, PDA, RDS, NEC, grade 2 ROP	31 d (39 w + 3 d)	Hyperintense dots on right periventricular white matter on T1
8	30 w	PM, RDS, grade 1 ROP, seizure	30 d (34 w + 2 d)	Normal
9	35 w + 3 d	PM, transient tachypnea, pneumothorax, seizure	25 d (39 w)	Normal
10	32 w	PM, RDS, NEC	2 d (32 w + 2 d)	Bilateral hyperintensity at globus pallidi on T1
11	32 w	PM, RDS, sepsis	31 d (36 w + 3 d)	Millimetric hemorrhagic foci on left parietal white matter on SWI
12	33 w + 4 d	PM, PFO, VSD	9 d (34 w + 6 d)	Bilateral hyperintensity on globus pallidi on T1
13	28 w	PM, RDS, PDA	35 d (33 w)	Normal
14	39 w	MAS, SGA, hypoglycemic seizure	2 d (39 w + 2 d)	Hyperintense dots on bilateral centrum semiovale and periventricular white matter on T1

RDS respiratory distress syndrome, *ROP* retinopathy of prematurity, *PDA* patent ductus arteriosus, *NEC* necrotizing enterocolitis, *PFO* patent foramen ovale, *CoA* coarctation of aorta, *VSD* ventricular septal defect, *SVT* supraventricular tachycardia, *DIC* disseminated intravascular coagulation, *MAS* meconium aspiration syndrome, *SGA* small for gestational age, *w* weeks, *d* days

in two and bilateral microcysts in one patient of nine patients were noted in the study of Schlesinger et al. [6]. Van Baalen et al. [8] defined the term 'pseudocystic germinolysis' for such cyst formations before they disappeared during the next months. In our study group, we encountered several microcysts in SIEBCG lesions of five patients. On follow-up CUS of ten patients at postterm-equivalent age, SIEBCG lesions disappeared consistent with the evolution steps in the literature.

The etiology of the SIEBCG lesions still remains unclear. Smets et al. [10] reported the association with chronic lung disease. Schlesinger et al. [6] considered that these lesions could be either normal variant of the appearance of the caudate nuclei or possible manifestation of ischemia. Van Baalen et al. [8] speculated that the etiology of subventricular echogenicities is more likely ischemic rather than hemorrhagic origin that led to germinolysis and gliosis.

Magnetic resonance imaging was reported to be more sensitive than CUS to detect white matter injury in preterm newborns [11, 12]. We encountered accompanied hyperintense dots on centrum semiovale and periventricular white matter in five patients and subtle increased signal intensity on the globus pallidi in two patients on T1-weighted MR images. In preterm neonates, periventricular leucomalacia (PVL) is one of the characteristic manifestations of mild asphyxia apart from GMH. Periventricular foci of T1 shortening are accepted as findings of early white matter injury in PVL [13]. Increased signal of globus pallidi on T1-weighted images may be associated with kernicterus, hypoglycemia, liver disease, and total parenteral nutrition [14]. However, there was no evidence of such problems in both patients with increased signal intensity on the globus pallidi; nevertheless, RDS and NEC in one, and VSD in the other one were noted as the clinical evidence of probable ischemia. Medical history of our patients revealed clinical evidence of possible ischemia such as RDS, sepsis, seizures, NEC, DIC, PDA, VSD, SGA, and MAS, and mechanic ventilator support. Therefore, in the presence of clinical signs of hypoxia, those signal alterations may be attributed to presumptive ischemic insult.

To our knowledge, this study evaluates the largest group of newborns with SIEBCG who were investigated by MRI

and is the first study to investigate SIEBCG by using SWI. SWI is an advanced MRI technique, established based on a high-resolution, three-dimensional, gradient-echo sequence, utilizing both magnitude and phase images. It is very sensitive to magnetic susceptibility changes and differentiate blood products and calcifications. It has been stated that SWI is more sensitive than computed tomography and gradient-echo MRI sequence in detecting small-sized cerebral hemorrhages [1, 15]. In addition to the conventional MRI sequences, SWI has been shown to have the potential of providing further precious information about parenchymal microhemorrhages, GMH, intraventricular hemorrhage, and dilated prominent intramedullary veins [16]. In our study group, SWI was also beneficial in detection of cerebellar and parietal microhemorrhages in two patients in addition to the exclusion of subependymal hemorrhages. In four patients, no pathological finding was found on neither conventional MRI sequences nor SWI. However, there exists debate whether these neonates should be considered as normal in this retrospective study without the knowledge of long-term neurological outcome. The impact of SIEBCG alone on neurodevelopmental outcome is not well known. The study [17] comparing two groups of neonates with and without subependymal lesions, matched for gestational age, with chronic lung disease, concluded that these lesions have no major impact on neurodevelopmental outcome. Conversely, association with neurodevelopmental outcome was revealed in the study by Horsch et al. [7].

We acknowledge the retrospective design and lack of the knowledge of neurological outcome on long-term follow-up as limitations of this study. Prospectively designed studies with larger study groups and long-term follow-up should be performed to determine interval imaging findings and clinical outcome of SIEBCG in the future.

Conclusion

We indicate that SIEBCG detected on newborn CUS is generally not a manifestation of GMH by using SWI. However, it may be related with other pathologies as presumptive ischemic insult or focal parenchymal hemorrhage. Knowledge about characteristic imaging features of SIEBCG is important in daily CUS practice, and if it exists, further examination with SWI should be performed.

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Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments. All authors have approved the manuscript and have significantly contributed to it. This article does not contain any studies with animals performed by any of the authors.

Informed consent Informed consent was waived by the institutional ethical committee due to the retrospective nature of this study.

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