

Clinical Insights and Social Vulnerability in Infantile Hemangiomas: A 10-Year Retrospective Cohort in Puerto Rico

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Abstract

Infantile hemangiomas (IHs) are the most common benign vascular tumors in childhood. While most occurrences regress spontaneously, some require treatment due to ulceration, functional impairment, or cosmetic concerns. In Puerto Rico, social determinants of health potentially affect both access to healthcare and clinical outcomes. The Social Vulnerability Index (SVI), a composite metric encompassing socioeconomic status, household composition, transportation, and other social factors, is commonly used to evaluate community-level vulnerability. This study sought to describe lesion location and treatment modalities, and evaluate whether the SVI is associated with treatment outcomes in IH patients in Puerto Rico as measured by lesion size at presentation, follow-up, and change in lesion size following treatment. Records of pediatric patients diagnosed with hemangiomas between 2015 and 2025 were retrospectively reviewed. A total of 30 patients were identified, and extracted data included patient demographics, treatment modality, lesion location, and lesion size, as measured via a flexible measuring tape, before and after treatment. SVI was calculated and assigned according to patients' residential Zone Improvement Plan (ZIP) code and matched to the corresponding SVI values obtained from the Agency for Toxic Substances and Disease Registry (ATSDR) database at the time of presentation. Most patients (43.3%) were treated with propranolol, followed by surgical resection or embolization (20%). The most affected areas were the limbs (50%) and the head and neck (30%) region. Using Spearman's rank correlation, the study found that a higher SVI was associated with a larger hemangioma lesion size at presentation (Spearman's $\rho = 0.681$, $p = 0.005$) and remained correlated with a larger post-treatment lesion size (Spearman's $\rho = 0.634$, $p = 0.011$). However, the SVI was not associated with the magnitude of size reduction following treatment (Spearman's $\rho = 0.09$, $p = 0.742$), meaning that the disparities in lesion size are likely due to differences in disease severity at the time of diagnosis rather than differences due to treatment response. Meanwhile, results also suggest that since more than half of patients were managed with pharmacologic therapy, treatment compliance may play an important role in influencing outcomes. Ultimately, SVIs could be used to flag patients who may present later or with larger lesions in order to enable earlier monitoring and management.

Categories: Public Health, Pediatrics, Dermatology

Keywords: infantile hemangioma, pediatrics, social vulnerability index, tumor, vascular

Introduction

Infantile hemangiomas (IHs) are the most common tumors in pediatric populations. They commonly arise as benign, vascular tumors originating from abnormal proliferation of endothelial cells that form aberrant vascular channels [1]. IHs occur due to dysregulated vasculogenesis and angiogenesis, which is thought to be triggered by hypoxia-induced activation of the hypoxia-inducible factor-1 α (HIF-1 α) pathway. This, in turn, leads to overexpression of the vascular endothelial growth factor (VEGF), with simultaneous clonal proliferation of vascular endothelial cells [2]. As a result of hypoxic stress, HIF-1 α accumulates in the nucleus and activates transcription of VEGF. In addition, the concomitant decrease in vascular endothelial growth factor receptor-1 (VEGFR1) has a direct effect on the increased VEGF, as this acts as a decoy receptor that normally sequesters VEGF. Additional contributing mechanisms are believed to promote IH development and progression, including the renin-angiotensin system, estrogen signaling, and inflammatory mediators [1]. Hypoxia has been considered the initiating factor in hemangiogenesis, resulting in a vascular overgrowth that attempts to restore normal oxygen tension [3]. This generates a cascade resulting in stem cells expressing markers such as CD133, which are able to differentiate into the endothelial cells, pericytes, and adipocytes that constitute the final lesion [3]. Moreover, the fact that hemangioma endothelial cells express erythrocyte-type glucose transporter GLUT1, a placenta-associated marker that is seldom present in normal skin, leads to the placental origin hypothesis, in which placental endothelial progenitor cells are believed to enter the developing fetus and manifest in the postpartum period [4]. It is also believed that the renin-angiotensin aldosterone system can further explain the development of these lesions, given that renin is known to promote the proliferation of hemangioma stem

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cells through the increase in angiotensin II. Moreover, renin is known to be increased early in infancy [3]. Hormonal signaling via estrogen and progesterone has also been considered as a simultaneous pathway due to the presence of receptors for both of these hormones within IHs, along with the fact that estradiol stimulates hemangioma VEGF production and subsequent endothelial cell proliferation [3]. All of these factors developing within a pro-angiogenic microenvironment populated by pericytes and M2-polarized macrophages promote vessel formation and growth [3].

IHs have an estimated global, pooled prevalence of 2.8%, with multiple studies reporting prevalences ranging 2%-10% [5]. Though IH prevalence data specific to Puerto Rico and Latino populations remain largely unexplored, one prospective, US-cohort study found Hispanics to comprise 14.4% of IH patients [6]. Several multifactorial risk factors have been found to increase the likelihood of developing IH. In particular, female sex increases the likelihood of IH by twice as much as in males [7,8]. Many studies also cite low birth weight and prematurity as strong, independent risk factors for IH [8,9]. However, some studies in Asian populations have reported contradictory or conflicting findings, which may suggest variation by ethnic background [10]. White, non-Hispanic race and previous family history of hemangioma have also been associated with increased likelihood of IH [5,6]. Additional maternal and placental independent risk factors for IH have been identified, including history of miscarriage, vaginal bleeding, placenta previa and other placental abnormalities, preterm premature rupture of membranes, preterm rupture of membranes, anemia during pregnancy, and maternal progesterone use before or early in pregnancy [5,8,10].

At birth, the clinical presentation of IHs is often subtle or not visible, but may present as faint stains with a blanched halo [11]. IHs typically undergo a proliferative phase of rapid growth one to three months following birth, with 80% of lesions reaching their final size and distribution by five months [12]. At around nine to 12 months of age, IH lesions enter an involution phase, fading in color and flattening; of these regressing lesions, 50% are fully involuted by age 5, and 95% by age 10-12 [13]. Superficial IHs appear as bright red, protuberant plaques, while deeper lesions appear as more bluish, dome-shaped nodules with irregular borders [14]. Approximately 47% of all lesions grow around the face and neck, with 73.8% of these appearing along the vertical midline or ocular axis, and on the tip of the nose and perioral region [5,15].

While nearly all IH lesions spontaneously involute over time, approximately 10%-15% of these lesions require specialized treatment to manage or avoid complications [14]. Complications of untreated, non-regressing IHs commonly include ulceration, bleeding, infection, anatomic distortion, and functional impairment [14,16,17]. IHs located on the eyelids may result in visual obstruction, which may evolve into proptosis, difficulty in eyelid closure, or damage to the optic nerve [14]. Other functional impairments may include obstruction of the oral cavity [17]. One study found that 55%-69% of non-regressing IH lesions result in permanent disfigurement [18]. Concerningly, some cases may even have life-threatening complications, including profuse bleeding from ulcers, airway obstruction by subglottic hemangiomas, and cardiac failure due to macrovascular shunting, as in the case of IHs on the liver [17].

Nonetheless, given the self-resolving nature of these tumors, they often require no treatment. However, since approximately 10 to 15% can cause functional impairment, ulceration, disfigurement, or life-threatening complications, close and active intervention is oftentimes necessary [16]. Because of this broad range of presentations, treatment is tailored utilizing a risk-based approach in which treatment is determined based on size, depth, location, and growth trajectory of the lesion, rather than because of its mere presence [19]. These distinctions are what make these lesions time-sensitive, since intervention during the proliferative phase is associated with higher response rates and better outcomes. First-line treatment is pharmacological therapy with propranolol at a dose of 2-3mg/kg/day for at least six months. With this intervention, there is an expected clearance rate of 95% [18]. Propranolol is believed to promote regression via early vasoconstriction of the feeding vessels, downregulation of VEGF, and fibroblast growth factor that suppresses angiogenesis, leading to subsequent apoptosis of the developing and proliferating endothelium [3]. Since it is a nonselective beta-blocker, its use must be monitored given the potential risk of hypotension, bradycardia, hypoglycemia, bronchospasm, and sleep disturbances. Atenolol has been utilized as a second-line treatment when propranolol is poorly tolerated due to its lower risk of bronchospasm and hypoglycemia [4]. Timolol can be used as well for small, thin, and superficial IH [18]. Sirolimus or systemic corticosteroids can be considered for IHs that are unresponsive to first-line treatment [20]. Pulsed dye laser (PDL) therapy is another alternative for smaller IHs or even remaining telangiectasias after initial treatment. Surgical interventions are generally avoided in IHs given the risks that can be associated with undergoing anesthesia and the potential for inferior cosmetic outcomes [18]. These are generally reserved for cases that have failed first-line treatment and obstruct or deform vital structures [18]. Treatment length and modality require clinical judgement given that between 10 and 25% of lesions recur after propranolol is discontinued. IHs require early detection and close follow-up by a multidisciplinary team in order to achieve successful outcomes [18].

The Centers for Disease Control and Prevention and Agency for Toxic Substances and Disease Registry Social Vulnerability Index (CDC/ATDR SVI) is a place-based measure of a community's vulnerability to harm [21]. The CDC/ATDR SVI functions as a tool to identify which communities are most vulnerable using geographic units such as Zone Improvement Plan (ZIP) codes to rank areas based on 15 social variables assigned by the CDC, assigning them a percentile ranking from 0 to 1, in which a higher value indicates a greater degree of vulnerability [22]. The variables evaluated fall into the following domains: socioeconomic status, household

composition and disability, racial and ethnic minority status and language, and housing type and transportation [22]. Since its introduction in 2011, the SVI has been used in various studies to evaluate its association with health disparities, including those in pediatric patients. In one cohort study assessing pediatric postoperative outcomes, higher SVI scores were associated with worse surgical outcomes [23]. Another study found that pediatric patients with inflammatory bowel disease from more vulnerable neighborhoods were more likely to be hospitalized at diagnosis and within the first year and have active disease at the six-month mark [24]. However, the association between SVI and pediatric dermatological conditions remains understudied. To our knowledge, this is the first study to evaluate how the CDC/ATDR SVI may be associated with disparities in presentation and outcomes of IH. This article was previously presented as a poster presentation at the 2026 SID Annual Meeting on May 14, 2026.

Materials And Methods

A retrospective cohort study was conducted at Hospital Universitario de Adultos (located at the University of Puerto Rico, Medical Sciences Campus) from January 2015 to January 2025. All patients admitted for hemangioma lesions under 18 years old were included for review. Exclusion criteria consisted of patients with co-existing, non-hemangioma vascular malformations. A total of 30 pediatric hemangioma patients were identified. Collected data included age, sex, ZIP code, lesion location, treatment modality, and lesion size before and after treatment. Duration of treatment and follow-up timing were not recorded in patient data. SVI scores were assigned to each patient based on their residential ZIP code at the time of presentation. ZIP codes were mapped to corresponding SVI values obtained from the ATSDR database from the CDC. Specifically, the 2022 CDC/ATSDR SVI dataset for Puerto Rico was used, which provided both ZIP Code Tabulation Area (ZCTA) identifiers and their corresponding SVI values.

The data were analyzed using IBM SPSS Statistics version 32.0 (IBM Corp., Armonk, NY), and figures were generated using Microsoft Excel (Microsoft® Corp., Redmond, WA). Categorical variables such as sex, lesion location, and treatment modality were summarized as frequencies and percentages. For quantitative variables, we tested the normality of the distribution using the Shapiro-Wilk test, which revealed that lesion-size variables were not normally distributed. These were reported as both mean with standard deviation and median with interquartile range (IQR). Lesion size was recorded as the maximum linear dimension before and after treatment, and the change in lesion size was calculated as the post-treatment maximum linear dimension minus the pre-treatment maximum linear dimension. Correlation analyses were limited to 15 patients with both pre- and post-treatment measurements documented in their medical records. Patients lacking either one or both of these measurements were excluded from analysis. The association between lesion size and SVI was evaluated using Spearman's rank and Pearson correlation coefficients. All tests were two-tailed, and $p < 0.05$ was considered statistically significant.

This study was approved by the Institutional Review Board of the University of Puerto Rico, Medical Sciences Campus, School of Medicine (approval no. 2506429796, 07/09/2025).

Results

We analyzed a total of 30 patients under 18 years old diagnosed with IH. Sex distribution demonstrated that 19 patients (63.3%) were male and 11 (36.7%) were female. Hemangioma localization was classified into five main anatomical regions: head and neck (30%), trunk (10%), upper limb (30%), lower limb (20%), and external genitalia (10%) (Table 1).

Location	Number of subjects
Head and neck	
Scalp	2
Facies	4
Eyelid	3
Trunk	
Anterior chest	1
Back	1
Buttock	1
Upper limb	
Left arm and forearm	3
Right arm and forearm	6
Lower limb	
Left leg and thigh	3
Right leg and thigh	3
External genitalia	
Scrotum	2
Labia majora	1

TABLE 1: Anatomical distribution of infantile hemangiomas (N = 30)

Types of interventions and treatments varied across the cohort (Figure 1). However, pharmacologic monotherapy was the most common approach, which accounted for 17 patients (56.7%). Specifically, propranolol was the most frequently used agent and was administered to 13 patients (43.3%). An additional three patients (10%) received sirolimus, and one patient (3.3%) received intralesional triamcinolone. Procedural interventions were performed on six patients (20%), which were divided into embolizations (three patients, 10%) and surgical resections (three patients, 10%). A combination of two or more modalities was used in two patients (6.6%). Specifically, one patient was treated with a combination of sirolimus and embolization, while the other was treated with cefadroxil and mupirocin. The remaining five patients (16.7%) received no active treatment and were solely observed.

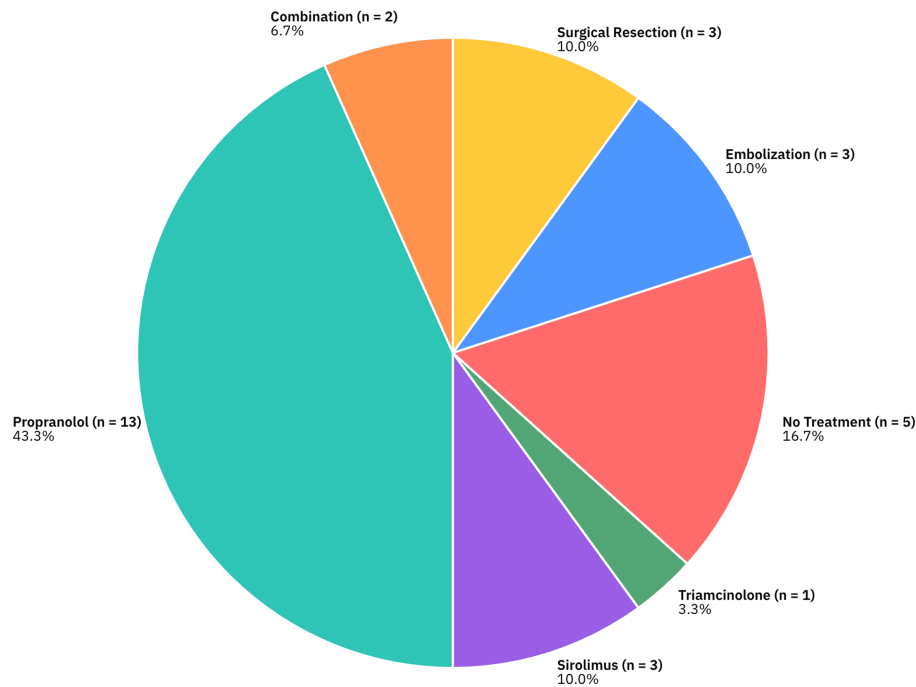


FIGURE 1: Treatment classification for infantile hemangiomas (N = 30)

Additionally, we examined whether social vulnerability was associated with lesion severity and treatment response. Pearson correlation analyses were conducted to assess the relationship between the SVI score and hemangioma lesion size, measured as linear maximum dimension, before and after treatment. These analyses included only 15 out of 30 patients, as the remainder of the cohort lacked recorded lesion measurements and were therefore excluded. As shown in Table 2, higher SVI scores were associated with larger lesions at both presentation and follow-up, but not with the degree of change following treatment.

Variable	Mean	SD	Median (IQR)	Spearman's rho (p)	p-value
SVI	0.39	0.19	0.40	-	-
Pre-treatment maximum linear dimension (cm)	4.61	3.39	3.50 (2.50-4.75)	0.681	0.005
Post-treatment maximum linear dimension (cm)	3.65	3.53	2.50 (1.25-5.00)	0.634	0.011
Change in lesion size (cm)	0.96	1.42	0.00 (0.00-2.00)	0.093	0.742

TABLE 2: Descriptive statistics and correlations of hemangioma lesion size with the social vulnerability index (SVI) (n = 15)

Notes: Because all lesion-size variables were non-normally distributed (Shapiro-Wilk p < 0.05), correlations with SVI are reported as Spearman's rank coefficients (p). Pearson correlation analyses yielded results consistent with the Spearman analyses (pre-treatment r = 0.754, p = 0.001; post-treatment r = 0.734, p = 0.002; change r = -0.026, p = 0.928).

SVI: Social Vulnerability Index; SD: standard deviation; p: Spearman's rank correlation coefficient

Discussion

In this cohort study, IHS were distributed across five main anatomical regions, with head and neck and upper limb portions accounting for 30% of lesions each, followed by lower limb (20%), trunk and external genitalia (10% each). This distribution differs slightly from that of larger prospective cohorts, in which head and neck lesions account for approximately 40-60% of cases and are generally attributed to the region's surface area and vascular density during embryogenesis [24]. The comparatively high proportion of extremity lesions in this study may reflect the relatively small sample size (n = 30). Additionally, the study observed a male predominance (63.3%), starkly contrasting with a well-established 2:1 female-to-male ratio [6]. While this

difference is likely associated with the size of the cohort, it further reinforces the need for a larger cohort study in this historically understudied population.

The allocation of treatment in this cohort was consistent with current literature and guidelines. A total of 40% of patients were treated with propranolol, the gold-standard, first-line agent for IHs requiring intervention [18]. Interestingly, only five patients (17%) were managed with observation alone, a much smaller proportion relative to the ~90% of IHs that undergo spontaneous regression without therapy [17]. The remaining patients received second-line or procedural therapies, including sirolimus, embolization, surgical resection, triamcinolone, or combination regimens, which are generally reserved for lesions that are unresponsive to first-line treatment, threaten vital structures, or pose significant cosmetic concerns [20,25].

Upon non-parametric correlational analysis, it was found that a higher SVI was associated with larger hemangioma dimensions at both pre-treatment (Spearman's $\rho = 0.681$, $p = 0.005$) and post-treatment (Spearman's $\rho = 0.634$, $p = 0.011$), but there was no significant relationship with the magnitude of size reduction. The SVI aggregates census-tract measures of socioeconomic status, household composition, minority status, and housing and transportation into a composite score that serves as a proxy for the structural barriers a patient's community faces [26]. Although subject to the limitations discussed below, the positive correlation between a patient's SVI and lesion size at presentation observed in this cohort is consistent with a growing amount of literature that supports the notion that socially disadvantaged children experience a delayed diagnosis and present with a more advanced disease at initial evaluation. For example, in a retrospective cohort of 804 children, those of lower socioeconomic status were more likely to present for initial evaluation of an IH after three months, and children evaluated after six months carried higher hemangioma severity scores [27]. Similarly, national inpatient data showed that infants from the lowest income quartiles present for initial evaluation at older ages and undergo more hemangioma-related procedures [28]. Because IHs proliferate most rapidly between five and eight weeks of age, delayed access to specialty care among more vulnerable families likely allows lesions to reach a larger maximum dimension before they are treated, an interpretation that is consistent with recognized disparities in pediatric dermatologic care [29].

As mentioned above, it is important to note that SVI was not associated with the magnitude of size reduction following treatment (Spearman's $\rho = 0.09$, $p = 0.742$). It is possible that once patients had access to care, the degree of treatment response was comparable across the SVI spectrum. In other words, we hypothesize that this disparity in lesion size reflects differences in disease severity at the time of diagnosis rather than differences in treatment effectiveness, meaning that since these patients from vulnerable communities presented with larger lesions, they were more likely to experience larger post-treatment lesions. A potential approach to reduce the observed disparity is to focus on earlier detection and timely referrals within more socially vulnerable populations, rather than on treatment protocols alone. Ultimately, the SVI may have potential as a tool to flag higher-risk patients who could benefit from earlier monitoring and intervention, although its clinical utility should be validated in larger prospective studies.

This study has some limitations. First, the statistical power was limited by the relatively small sample size. Also, because the cohort was obtained from a tertiary referral center, the study's sample may overrepresent higher-risk lesions, which could explain the atypical anatomical and sex distributions, as well as the low rate of observation-only management. Meanwhile, lesion measurements were documented for only 15 patients, so if measurements from the remaining 15 patients were missing in a non-random pattern (e.g., larger lesions were more likely to be recorded), then selection bias would be an important limitation in this subsample. Also, the timing of post-treatment measurements was not standardized, as the interval between treatment and follow-up measurement was not documented; change in maximum linear dimension is therefore a limited measure of comparative treatment response. Additionally, the study did not directly evaluate treatment adherence. Moreover, the SVI is an ecological measure based on an individual's ZIP code, which may not be the most accurate reflection of a family's unique circumstances [30]. Larger, multicenter studies with standardized lesion measurements and individual-level social data are needed to validate these findings.

Conclusions

IHs are treatable when identified early, with timely diagnosis and referral being key for achieving favorable outcomes in affected children. To our knowledge, this retrospective cohort is the first study to correlate the CDC/ATSDR SVI with IHs outcomes. Moreover, it is the first to characterize IH presentation, distribution, and treatment patterns within the Puerto Rican pediatric population. In this cohort, lesions were predominantly distributed across the head, neck, and limbs, with a male predominance. Most were managed with pharmacologic monotherapy using propranolol. Higher SVI was significantly associated with larger lesion size both at presentation and follow-up, but not with the magnitude of size reduction achieved through treatment. We hypothesize that the disparity in lesion burden reflects more advanced disease at diagnosis rather than a difference in treatment response. Nevertheless, the cohort's small sample size, single tertiary-center design, and correlational analyses restricted to only the subset of patients who had completed pre- and post-treatment measurements limit the generalizability of these conclusions. Future studies with larger, multi-site prospective cohorts are needed to confirm these associations and explore whether SVI-guided monitoring can meaningfully reduce disparities in the care of IH patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. University of Puerto Rico, Medical Sciences Campus Institutional Review Board issued approval 2506429796. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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