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Etiological Profile of Epilepsy in Patients at a Tertiary Care Hospital: An Observational Study

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ABSTRACT

Epilepsy has diverse etiologies that vary by age, gender, and region. Identifying the cause is essential for management and prognosis. **Objectives:** To determine the etiological profile of epilepsy in patients at a tertiary care hospital and assess associations with age and gender. **Methods:** This observational study was conducted in the Department of Neuro-medicine, Liaquat University of Medical and Health Sciences, Jamshoro, from March 15 to September 14, 2022. A total of 156 patients aged ≥18 years with epilepsy for over five years were enrolled by consecutive sampling. Those with acute symptomatic seizures or incomplete records were excluded. Etiologies were classified using International League Against Epilepsy 2017 criteria based on electroencephalography and magnetic resonance imaging. **Results:** A total of 156 adult epilepsy patients were included (mean age 54.7 ± 15.6 years; 101 male, 55 female). Acute ischemic stroke was the most common etiology, 74 (47.4%), followed by idiopathic epilepsy, 29 (18.6%), brain tumors, 15 (9.6%), traumatic brain injury, 13 (8.3%), intracerebral hemorrhage, 10 (6.4%), CNS infections, 8 (5.1%), and encephalitis, 11 (7.1%). A higher proportion of female was observed within the brain tumor and idiopathic epilepsy subgroups, 10 (66.7%) and 19 (65.5%), respectively ($p < 0.05$); however, these findings reflect distribution within the tertiary cohort and should not be interpreted as population-level risk. Insignificant associations were found between etiology and age group or epilepsy duration. **Conclusions:** Acute ischemic stroke was the leading cause of epilepsy, with significant gender differences for brain tumors and idiopathic epilepsy.

INTRODUCTION

Epilepsy is common in resource-limited regions of Pakistan, notably Khyber Pakhtunkhwa and younger male, with associations including parental consanguinity (42.8%), low socioeconomic status (88.6%), and urban residence; treatment non-compliance, deprivation, and seizure type predict poor therapy response [1]. A tertiary hospital study of Karachi reported 42% of seizures as secondary, primarily from neonatal encephalopathy (18.7%), head trauma (18.2%), and CNS infections (17.9%) [2]. Further research is needed to clarify epilepsy etiologies and seizure types in Pakistan. Data from the Emergency Department of Aga Khan University Hospital, Karachi, indicate that structural lesions account for 89.6% of new-onset seizures, whereas idiopathic causes comprise only 4%, emphasizing the need for locally developed guidelines to support emergency physicians in accurate diagnosis and effective management [3]. Consanguineous marriages, observed in approximately 63% of tribal societies in Pakistan, are strongly associated with an increased risk of epilepsy, with an odds ratio of 3.25 (95% CI: 2.35–4.51, $p<0.001$). One study found that 37.33% of patients had parents who were first cousins, with rates exceeding 60% in certain tribal areas of Khyber Pakhtunkhwa [4]. Although some data are available for

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children showing that 45.5% of refractory pediatric epilepsy cases have an undetermined cause and that the ketogenic diet, when started gradually, is a safe and effective treatment for Pakistani children, there is a lack of comprehensive data on the use and effectiveness of this therapy in adult patients [5]. A North Indian study reported that infections, malignancies, and metabolic causes are more prevalent in younger patients with new-onset seizures, whereas strokes predominate in individuals over 55 years, underscoring the importance of age-specific diagnostic and therapeutic strategies [6]. Status epilepticus is a severe neurological emergency with high morbidity and mortality, most common in young children and the elderly. In Pakistan, infections (17%) and trauma (11%) are the leading causes. In our setting, it is rare, predominantly convulsive, with no non-convulsive cases, highlighting the need for larger studies [7]. Epilepsy affects ~46 million people globally, with a higher incidence in low-income regions. A Karachi study found normal EEGs in two-thirds of pediatric patients and interictal epileptiform discharges in one-third, with generalized epilepsy more common in older children and focal in younger, highlighting EEG's diagnostic value and limitations [8]. Machine learning shows potential for predicting outcomes in drug-resistant epilepsy but is underutilized in Pakistan. Using clinical and demographic data, a support vector machine achieved 76.1% overall accuracy, 96.7% for temporal lobe, and 79.5% for extratemporal epilepsy, supporting supervised classification and feature selection to improve presurgical predictions [9]. Tertiary care hospitals play a key role in managing complex and drug-resistant epilepsy, offering advanced diagnostics such as MRI, PET, EEG, and genetic testing. Even in resource-limited settings, epilepsy-specific MRI and video-EEG are essential for accurate diagnosis, syndrome classification, lesion identification, and surgical candidate selection [10].

Although epilepsy is a significant burden in Pakistan, there is sparse local data that has been systematically used to describe the etiological spectrum of epilepsy with the updated ILAE 2017 classification. The majority of literature is based on outdated classifications that do not involve the use of advanced modalities of diagnosis, thus incomplete epidemiological profiling and non-optimal management plans. This study aims to assess the etiological picture of adult epilepsy at a tertiary care unit using the ILAE 2017 framework, evaluate the demographic correlations, and consider the new diagnostic tools to improve clinical decision-making and better resource allocation to the region.

METHODS

This analytical cross-sectional study was conducted in the Department of Neuro-medicine at Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, from March 15 to September 14, 2022, following ethical approval from the Institutional Review Board of LUMHS (Approval No. LUMHS/REC/-180). Using a non-probability consecutive sampling method, 156 patients were enrolled to assess the etiological spectrum of epilepsy. The sample size for this study was determined using the World Health Organization (WHO) sample size calculator. Based on a previously reported prevalence of encephalitis of 6.94% among epilepsy patients, a 4% margin of error, and a 95% confidence interval, the required sample size was calculated to be 156 participants. However, this sample size was deemed adequate to provide sufficient statistical power to analyze etiological factors within the study population [11]. Patients aged 18 years or older, of either gender, presenting to the Neuro-medicine outpatient department with a clinical diagnosis of epilepsy were enrolled after obtaining informed written consent. Inclusion criteria encompassed all adult epilepsy patients regardless of seizure duration, thereby including both newly diagnosed and chronic cases. Explicit exclusion criteria were applied to minimize bias and included patients presenting with acute symptomatic seizures, those with incomplete or missing medical records, and individuals unwilling to participate. Comprehensive demographic and clinical data were systematically collected for each participant. This included basic demographic variables such as age, gender, weight, and height. Seizure types were classified according to the International League Against Epilepsy (ILAE) 2017 criteria to ensure standardized diagnostic categorization. A thorough neurological examination was conducted to identify any focal neurological deficits or signs suggestive of underlying etiologies. Additionally, information on comorbid medical conditions was documented, given their potential impact on disease progression and management. Details regarding anti-seizure medication (ASM) regimens, including types and dosages, were recorded alongside the current seizure control status, defined by frequency and severity of episodes. Relevant patient history was also obtained, covering prior central nervous system infections, perinatal insults, traumatic head injuries, and family history of epilepsy, factors critical to understanding genetic and environmental contributions to epilepsy. All data were meticulously recorded using a structured proforma to maintain uniformity, completeness, and accuracy for subsequent analysis. All patients underwent CT (Toshiba, Canon) and epilepsy-specific MRI (1.5T, high-resolution T1/T2 sequences; 3T MRI), along with EEG and standard 12-

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lead digital ECG for seizure classification. CT scans were reviewed by the principal investigator and confirmed by a consultant radiologist. Etiologies were classified as hemorrhagic/ischemic stroke, CNS infections, neoplasms, metabolic, or idiopathic. Combining EEG, ECG, and imaging allowed comprehensive etiological profiling, with all data collected personally by the principal researcher under standardized protocols. Data analysis was performed using IBM SPSS Statistics Version 26.0. Categorical variables such as gender, etiological categories, and seizure types were summarized as frequencies and percentages. Data were stratified by age, gender, and epilepsy duration to examine associations with etiological patterns. Chi-square or Fisher's exact tests were used as appropriate, with a p-value<0.05 considered statistically significant.

RESULTS

The descriptive characteristics of 156 patients with epilepsy included in the study. The mean age of the cohort was 54.72 ± 15.60 years, with a predominance of male, 101(64.7%), compared to female, 55(35.3%). The average duration of epilepsy was 8.35 ± 2.49 years. Regarding etiological distribution, acute ischemic stroke was the most common cause, observed in 74 (47.4%), followed by central nervous system infections in 29 (18.6%), including tuberculous meningitis in 18 (10.9%) and encephalitis in 11 (7.1%). Hemorrhagic stroke was identified in 21 (13.5%), brain tumors in 15 (9.6%), and idiopathic epilepsy in 29 (18.6%). Structural causes, especially stroke, predominated in this adult epilepsy cohort, with notable contributions from infectious and idiopathic etiologies (Table 1).

Table 1: Descriptive Statistics of Epileptic Patients with Total Sample Size (n=156)

Variables	Mean ± SD, n (%)
Age Group	54.72 ± 15.60
Male	101 (64.7%)
Female	55 (35.3%)
Duration of Epilepsy	8.35 ± 2.49
Hemorrhagic Stroke	21 (13.5%)
Acute Ischemic Stroke	74 (47.4%)
Central Nervous System Infections	29 (18.6%)
Tuberculous Meningitis	18 (10.9%)
Encephalitis	11 (7.1%)
Brain Tumors	15 (9.6%)
Idiopathic	29 (18.6%)

Table 2: Descriptive Statistics of Epileptic Patients with Total Sample Size (n=156)

Etiological Factors of Epilepsy		Age Group (in years)		95% Confidence Interval for Mean 18 – 50		95% Confidence Interval for Mean>50		p-value
		18 – 50	>50	Lower Bound	Upper Bound	Lower Bound	Upper Bound	
Hemorrhagic Stroke	Yes	9 (42.9%)	12 (57.1%)	6.1	24.5	5.8	18.9	0.609
	No	50 (37.0%)	85 (63.0%)					
Acute Ischemic Stroke	Yes	30 (40.5%)	44 (59.5%)	38.1	63.5	35.5	55.3	0.506
	No	29 (35.4%)	53 (64.6%)					
Central Nervous System Infections	Yes	12 (41.4%)	17 (58.6%)	10.1	30.6	9.9	25.1	0.661
	No	47 (37.0%)	80 (63.0%)					
Tuberculous Meningitis	Yes	9 (50.0%)	9 (50.0%)	6.1	24.5	3.5	15.1	0.204
	No	48 (35.8%)	86 (64.2%)					
Encephalitis	Yes	2 (18.2%)	9 (81.8%)	0.0	8.0%	3.5	15.1	0.141
	No	57 (39.3%)	88 (60.7%)					
Brain Tumors	Yes	6 (40.0%)	9 (60.0%)	2.5	17.9	3.5	15.1	0.855
	No	53 (37.6%)	88 (62.4%)					
Idiopathic	Yes	7 (4.5%)	22 (14.1%)	3.6	20.1	14.4	31.0	0.092
	No	52 (33.3%)	75 (48.1%)					

Applied Chi-square and Fisher's Exact test

The distribution of epilepsy etiologies according to gender in the study cohort (n=156). Hemorrhagic stroke, acute ischemic stroke, central nervous system infections, tuberculous meningitis, and encephalitis showed no significant differences between male and female (p>0.05 for all). In contrast, brain tumors and idiopathic epilepsy demonstrated significant gender differences, with female accounting for a higher proportion in both categories: brain tumors: 66.7% female vs. 33.3% male (p=0.010), and idiopathic epilepsy: 12.2% female vs. 6.4% male (p<0.001). Most etiologies were similarly distributed by gender, with higher frequencies of brain tumors and idiopathic epilepsy among female (Table 3).

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Table 3: Stratification of Gender with Etiological Factors of Epilepsy (n=156)

Etiological Factors of Epilepsy		Gender		95% Confidence Interval for Mean Male		95% Confidence Interval for Mean Female		p-value
		Male	Female	Lower Bound	Upper Bound	Lower Bound	Upper Bound	
Hemorrhagic Stroke	Yes	13 (61.9%)	8 (38.1%)	6.4	19.4	5.2	23.9	0.770
	No	88 (65.2%)	47 (34.8%)					
Acute Ischemic Stroke	Yes	49 (66.2%)	25 (33.8%)	38.8	58.3	32.3	58.6	0.715
	No	52 (63.4%)	30 (36.6%)					
Central Nervous System Infections	Yes	15 (51.7%)	14 (48.3%)	7.9	21.8	13.9	37.0	0.104
	No	86 (67.7%)	41 (32.3%)					
Tuberculous Meningitis	Yes	14 (77.7%)	4 (22.3%)	7.1	20.6	0.4	14.1	0.398
	No	85 (63.4%)	49 (36.6%)					
Encephalitis	Yes	7 (63.6%)	4 (36.4%)	2.0	11.9	0.4	14.1	0.586
	No	94 (64.8%)	51 (35.2%)					
Brain Tumors	Yes	5 (33.3%)	10 (66.7%)	0.7	9.2	8.0	28.3	0.010
	No	96 (68.1%)	45 (31.9%)					
Idiopathic	Yes	10 (6.4%)	19 (12.2%)	4.1	15.7	21.9	47.0	<0.001
	No	91 (58.3%)	36 (23.1%)					

Applied Chi-square and Fisher's Exact test

The relationship between epilepsy duration and etiological factors in the study cohort (n=156), with durations categorized as 6–8 years and >8 years. Across all etiologies, including hemorrhagic stroke, acute ischemic stroke, central nervous system infections, tuberculous meningitis, encephalitis, brain tumors, and idiopathic epilepsy, no significant differences were observed between the two duration groups ($p>0.05$ for all). Etiological distribution was independent of epilepsy duration (Table 4).

Table 4: Stratification for Duration of Epilepsy with Etiological Factors of Epilepsy (n=156)

Etiological Factors of Epilepsy		Duration (in Years)		95% Confidence Interval for Mean 6 – 8		95% Confidence Interval for Mean >8		p-value
		6 – 8	>8	Lower Bound	Upper Bound	Lower Bound	Upper Bound	
Hemorrhagic Stroke	Yes	14 (66.7%)	7 (33.3%)	6.7	19.5	4.5	24.1	0.838
	No	93 (68.9%)	42 (31.1%)					
Acute Ischemic Stroke	Yes	49 (66.2%)	25 (33.8%)	36.4	55.3	37.0	65.0	0.544
	No	58 (70.7%)	24 (29.3%)					
Central Nervous System Infections	Yes	18 (62.1%)	11 (37.9%)	9.8	23.9	10.7	34.1	0.402
	No	89 (70.1%)	38 (29.9%)					
Tuberculous Meningitis	Yes	12 (66.6%)	6 (22.4%)	5.2	17.2	3.1	21.4	0.589
	No	93 (69.4%)	41 (30.6%)					
Encephalitis	Yes	6 (54.5%)	5 (45.5%)	1.3	9.9	1.7	18.6	0.236
	No	101 (69.7%)	44 (30.3%)					
Brain Tumors	Yes	11 (73.3%)	4 (26.7%)	4.6	16.0	0.5	15.8	0.463
	No	96 (68.1%)	45 (31.9%)					
Idiopathic	Yes	19 (12.2%)	10 (6.4%)	10.5	25.0	9.1	31.7	0.693
	No	88 (56.4%)	39 (25.0%)					

Applied Chi-square and Fisher's Exact test

DISCUSSION

This study characterized the etiological spectrum of adult epilepsy at a tertiary Pakistani hospital, identifying acute ischemic stroke as the leading cause, followed by idiopathic epilepsy and brain tumors. These results align with global trends recognizing cerebrovascular disease as a major contributor to adult-onset epilepsy, particularly in aging populations. Globally, stroke accounts for ~10% of epilepsy cases and up to 50% in adults >60 years. Hemorrhagic stroke, cortical involvement, and early seizures increase risk, with Select (AUC 0.77) and CAVE (AUC 0.81) showing good predictive accuracy [12]. In our cohort, acute ischemic stroke (47.4%) and intracerebral hemorrhage (6.4%) mirrored global trends, underscoring the need for stroke prevention, optimal management, and validated prediction tools. Meningiomas, common primary brain tumors, frequently cause seizures, with complete resection achieving up to 90% seizure freedom. Postoperative risk depends on histology, location, edema,

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recurrence, and complications, while long-term antiepileptic benefit is limited [13]. In our study, brain tumors caused 9.6% of cases, with a higher proportion of female observed within the brain tumor and idiopathic epilepsy subgroups (66.7% vs. 33.3%, p=0.010); however, these findings reflect distribution within the tertiary cohort and should not be interpreted as a higher population-level risk, highlighting the importance of surgical and risk-based management. A meta-analysis of 22 voxel-based morphometry studies involving 913 patients with idiopathic generalized epilepsy demonstrated increased cortical and decreased subcortical gray matter, indicative of thalamocortical network disruption [14]. In our cohort, idiopathic epilepsy accounted for 18.6% of cases and showed a higher proportion of female (p<0.001). However, this finding reflects the distribution within our tertiary care sample and should not be interpreted as indicating a higher population-level risk for female, while the normal conventional imaging in most cases is consistent with the absence of overt structural lesions in idiopathic epilepsy. A study of 198 patients with new-onset seizures reported that most were middle-aged (35–65 years, 44.4%) with a slight female predominance (55.1%), and structural brain lesions were the predominant etiology [15]. Similarly, in our cohort (mean age 54.72 ± 15.60 years; male-to-female ratio 2:1), acute ischemic stroke (47.4%) and brain tumors (9.6%) were the leading causes, reinforcing the impact of structural lesions in adult-onset epilepsy and the need for region-specific diagnostic and management approaches. A previous study of 996 patients reported primary seizures in 58% and secondary in 42%, with structural lesions including head trauma, tumors, and stroke predominating in adults, and generalized tonic–clonic seizures being most common [16]. Similarly, in our 156-patient cohort, acute ischemic stroke was the leading cause (47.4%), followed by idiopathic epilepsy (18.6%), brain tumors (9.6%), and traumatic brain injury (8.3%), with generalized seizures predominating, confirming the association of adult-onset epilepsy with structural brain lesions. In a retrospective study of 431 adult epilepsy patients at King Abdulaziz Medical City, generalized seizures were most common (25.3%), with structural etiologies predominating (42.9%), particularly stroke (24.3%) and tumors (23.8%), while 47.6% remained of unknown etiology [17]. In our 156-patient cohort, structural causes predominated acute ischemic stroke (47.4%), idiopathic epilepsy (18.6%), brain tumors (9.6%), and traumatic brain injury (8.3%), with more female in tumor-related and idiopathic cases, reflecting global trends and regional demographic variations. A retrospective review of 1,097 pediatric epilepsy patients at The Children's Hospital, Lahore (2016–2020), found afebrile (72.7%) and generalized seizures (49.8%) most common, with idiopathic (49.2%) and congenital (20.8%) etiologies; typical seizures lasted 1–3 minutes, with up-rolling eyes and frothing (34.9%) frequently observed [18]. In contrast, our adult cohort showed higher structural etiology prevalence of acute ischemic stroke (47.4%) and brain tumors (9.6%), with idiopathic epilepsy at 18.6%, highlighting age-related differences in etiology and seizure patterns. A 25-year retrospective study of 5,712 adult-onset epilepsy patients in China reported declining unknown etiologies, predominance of structural epilepsy, especially cerebrovascular in older adults, higher rates in male, and a recent rise in immune-related cases [19]. Similarly, our study found structural causes, particularly acute ischemic stroke (47.4%), as the most common, with idiopathic epilepsy and brain tumors more frequent in specific subgroups, highlighting age- and sex-related differences and the need for targeted diagnostics and management. This study has several limitations. The cross-sectional design precludes assessment of temporal or causal relationships, and observed associations reflect cohort distributions rather than independent risk factors. Multivariate analysis was not performed; therefore, associations across age and sex subgroups may be confounded by factors such as disease severity, comorbidities, or referral patterns. The single tertiary care–center setting limits generalizability and may overrepresent structural or severe epilepsy. Etiological subcategories, particularly central nervous system infections, were analyzed by case distribution rather than population prevalence; thus, subgroup proportions should not be interpreted as population-level risk [20].

Finally, limited access to advanced diagnostics (e.g., prolonged video-EEG, autoimmune, and genetic testing) may have led to under- or misclassification in some cases. Despite these limitations, the study provides valuable tertiary care–based insights into epilepsy etiology, highlighting the predominance of post-stroke and other structural causes. The findings underscore the need for integrated neurological follow-up for stroke survivors, improved diagnostic pathways to distinguish seizures from acute cerebrovascular events, and strengthened prevention strategies targeting cerebrovascular disease and central nervous system infections. Future multicenter, longitudinal studies with multivariate modeling and advanced diagnostics are warranted to identify independent predictors and support personalized epilepsy management. The study was a single tertiary center cross-sectional study, which is unable to cause causality, and results might be biased to structural or severe epilepsy as opposed to the general population. Case distribution was used to analyze etiological subgroups and, in particular,

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central nervous system infections; proportions cannot be used to denote risk at the population level. Out of disturbed diagnostics, it might have under- or misclassified certain types of epilepsy due to limited accessibility. Despite these drawbacks, the research offers good information on the pattern of adult epilepsy and the necessity of developing special diagnostics, prevention, and treatment measures.

CONCLUSIONS

Acute ischemic stroke was the leading cause of epilepsy in this cohort, with gender differences observed in brain tumor–related and idiopathic cases. These findings underscore the importance of systematic evaluation and region-specific management strategies, including timely neuroimaging and electrophysiological assessment, to guide targeted interventions, improve stroke prevention, and optimize epilepsy care.

Authors' Contribution

Conceptualization: WA, MAL
Methodology: WA, SK, MAL
Formal analysis: WA, SK, SAN, AHB, MAL
Writing and Drafting: WA, SK, SAN, AHB, MAL
Review and Editing: SK, SAN, AHB, WA, MAL

All authors approved the final manuscript and take responsibility for the integrity of the work.

Conflicts of Interest

All the authors declare no conflict of interest.

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