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Review Article

The Parochial Meddle-Endolymphatic Sac Tumour

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Abstract

Endolymphatic sac tumour is an exceptional, benign neoplasm arising from epithelium of endolymphatic duct or sac. Endolymphatic sac tumour is associated with von Hippel –Lindau disease in around 11% to 30% of subjects. The gradually progressive endolymphatic sac tumour manifests with acute or progressive deafness, tinnitus, episodic vertigo, otalgia, otorrhoea, disequilibrium, aural fullness, occipital headache, neurological deficits, facial paresis or hypoglossus muscle paralysis. The papillary neoplasm is layered with simple, cuboidal to columnar epithelial cells with minimal nuclear atypia, infrequent mitotic activity and absence of tumour necrosis. Endolymphatic sac tumour requires a segregation from neoplasms such as middle ear adenoma, middle ear carcinoma, choroid plexus papilloma, paraganglioma, papillary ependymoma, metastatic renal cell carcinoma, metastatic thyroid papillary carcinoma, jugulotympanic paraganglioma, glomus jugulare tumour, enlarged vestibular aqueduct, petrous apicitis, cholesterol granuloma, meningioma or secondary metastasis within bone. Upon computerized tomography, erosion of petrous bone displays an infiltrative or "moth-eaten" configuration along with centric calcified spicules, intra-tumour and posterior rim calcification and intense, homogeneous enhancement of the tumefaction. Comprehensive surgical excision of the neoplasm is an optimal treatment strategy.

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Keywords

Endolymphatic Duct; Tumour; Neoplasm; Haemorrhage; Endoplasmic Reticulum

Abbreviations

WHO: World Health Organization; ER: Endoplasmic Reticulum; EMA: Epithelial Membrane Antigen; NSE: Neuron Specific Enolase; GFAP: Glial Fibrillary Acidic Protein

Introduction

Endolymphatic sac tumour is an extremely exceptional, benign neoplasm of the endolymphatic sac arising from epithelium of endolymphatic duct or sac. Endolymphatic sac tumour demonstrates a potential for localized tumour infiltration and bone destruction, indicative of clinically invasive biological behaviour. Nevertheless, distant tumour metastasis is absent. Endolymphatic sac tumour was initially described by Heffner DK in 1989 and the neoplasm was previously misinterpreted as choroid plexus tumour and adenoma or adenocarcinoma of posterior fossa or cerebellopontine angle [1]. Notwithstanding, World Health Organization (WHO) has segregated aggressive papillary tumour from endolymphatic sac tumour in 2017. Endolymphatic sac tumour is additionally designated as papillary adenomatous tumour, papillary adenoma of endolymphatic sac, middle ear adenocarcinoma, papillary tumour of temporal bone, adenocarcinoma of endolymphatic sac, adenocarcinoma of temporal bone or mastoid, low grade adenocarcinoma of probable endolymphatic sac origin, Heffner tumour or aggressive papillary middle ear tumour. Preliminary tumour discernment is crucial as antecedent surgical intervention may circumvent progression of hearing loss.

Disease Characteristics

Endolymphatic sac tumour originates from intraosseous or intra-temporal segment of endolymphatic sac which appears during neuro-ectodermal embryogenesis [2,3]. Endolymphatic sac tumour is associated with von Hippel-Lindau disease in around 11% to 30% of subjects. Nearly 30% neoplasms occurring in concurrence with von Hippel-Lindau disease are bilateral. Precursor lesions may be discerned in individuals with von Hippel- Lindau disease. Tumour cells may demonstrate somatic mutations of von Hippel-Lindau gene, especially in individuals devoid of the entity [2,3]. Upon imaging, roughly ~60% subjects with von Hippel-Lindau disease and vestibulocochlear symptoms demonstrate an absence of endolymphatic sac tumour [2,3]. The neoplasm can arise in concurrence with tumours of

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female adnexa, possibly of wolffian origin. Endolymphatic sac tumour may occur as a singular, independent entity [2,3]. Typically, endolymphatic sac tumour emerges in young individuals with a mean age of 22 years or a median age at 30 years although the neoplasm is commonly discerned within 11 years to 71 years. Mean age of emergence of endolymphatic sac tumour in concurrence with von Hippel-Lindau disease is 31.3 years, in contrast to independent neoplasms appearing at 52.5 years. An equivalent gender predilection is observed [2,3].

Clinical Elucidation

Clinical representation appears with progressive hearing loss in combination with or in the absence of diverse cranial neuropathies. Clinical symptoms are contingent to location and extent of incriminated brain tissue [3,4]. Ubiquitous clinical symptoms emerge as gradual or acute, unilateral sensi-neural deafness which occurs due to tumour progression and induced pressure upon adjoining auditory structures or the emergence of endolymphatic hydrops [3,4]. Typically, endolymphatic sac tumour is gradually progressive and manifests clinical symptoms as acute or progressive deafness, tinnitus, episodic vertigo, otalgia, otorrhoea, disequilibrium, aural fullness, occipital headache, neurological deficits, facial paresis or hypoglossus muscle paralysis [3,4].

Cranial neuropathies are comprised of cranial nerve paralysis, jugular foramen syndrome associated with glossopharyngeal neuralgia, paralysis of XII nerve and motor deficit related to the X nerve along with cerebellopontine angle syndrome demonstrating hearing loss, facial paralysis and dizziness [3,4]. History of trauma or surgical intervention may be elicited in concurrence with emergence of the neoplasm [3,4].

Histological Elucidation

Endolymphatic sac tumour is a low-grade, vascular, poorly circumscribed neoplasm associated with extensive, localized destruction of soft tissue and bone [4,5]. Grossly, the neoplasm represents as a soft, reddish-blue, polypoid lesion [4,5]. The tumefaction demonstrates a papillary configuration wherein papillary articulations are layered with simple, cuboidal to columnar epithelial cells with minimal nuclear atypia. Mitotic activity is infrequent and tumour necrosis is absent [4,5]. Upon cytological assessment, few epithelial cell aggregates and papillary articulations are discerned which are intermingled with foamy macrophages. Tumour cells depict a distinctive cellular perimeter and are incorporated with an eosinophilic, focally vacuolated cytoplasm with bland, uniform nuclei. Few cells demonstrate accumulation of pigmented granules, simulating hemosiderin pigment deposits [4,5]. Upon microscopy, tumefaction is composed of simple papillary articulations layered with singular layer of columnar to cuboidal epithelial cells with minimal cellular and nuclear pleomorphism.

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Morphologically, the neoplasm resembles a choroid plexus papilloma. A distinctive myoepithelial cell layer arising from flattened stroma is discernible. Epithelial cells demonstrate pale to clear cytoplasm, uniform centric or luminal nuclei and distinct cellular outlines [4,5]. Granulation tissue can be admixed with papillary articulations, composed of miniature vascular spaces intermingled with an acute and chronic inflammatory cell infiltrate. Occasionally, thyroid- like, hyper-cellular zones of cystic, glandular spaces imbued with colloid- like substance are observed. Foci of recent haemorrhage and cholesterol clefts are delineated. Mitotic figures are exceptional and foci of tumour necrosis are absent [4,5]. Upon microscopic examination, endolymphatic sac tumour exhibits

- A papillary variant which depicts papillary articulations layered by neoplastic cuboidal or columnar epithelial cells
- A follicular variant which enunciates cystic cavities imbued with colloid protein [5,6]. Certain neoplasms demonstrate an admixture of papillary and follicular patterns Additionally, endolymphatic sac tumour may demonstrate distinct histological subcategories denominated
- Mixed subtype which is preponderantly exhibited in confined neoplasms Papillary adenomatous subtype which is constituted of aggressive lesions demonstrating localized infiltration of abutting bone [5,6]

Upon ultrastructural examination, tumour cells display distinctive features as intercellular junctional complexes, microvilli, a definitive basement membrane, rough Endoplasmic Reticulum (ER), glycogen molecules and secretory granules [5,6]. Upon immunohistochemistry, tumour cells are diffusely immune reactive to pan-cytokeratin, cytokeratin 7, vascular endothelial growth factor, SOX10, vimentin, 5-lipoxygenase, Oligo-2 or iron and variably immune reactive to Epithelial Membrane Antigen (EMA), Neuron Specific Enolase (NSE), Glial Fibrillary Acidic Protein (GFAP), S100 protein, synaptophysin or Leu7/CD57. Tumour cells can be stained with Periodic Acid Schiff's (PAS) stain [5,6]. Ki-67 proliferation index is around 2%. p53 is reactive in roughly 80% tumour cells [5,6]. Tumour cells are immune non-reactive to mucin, cytokeratin 20(CK20), Carcinoembryonic Antigen (CEA), chromogranin, transthyretin, thyroglobulin and inhibin A (Fig. 1-8) [5,6].

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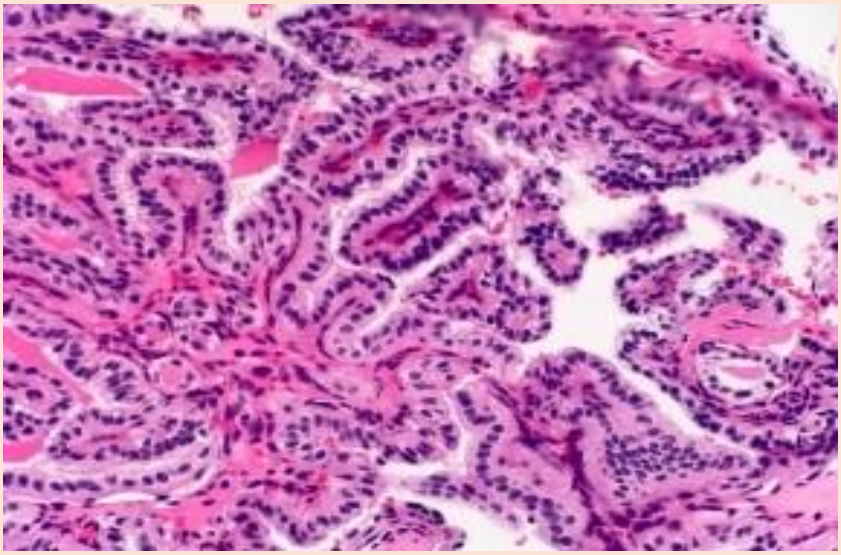

Figure 1: Endolymphatic sac tumour depicting papillary articulations lined by cuboidal to columnar epithelium with uniform nuclei and focal haemorrhage.

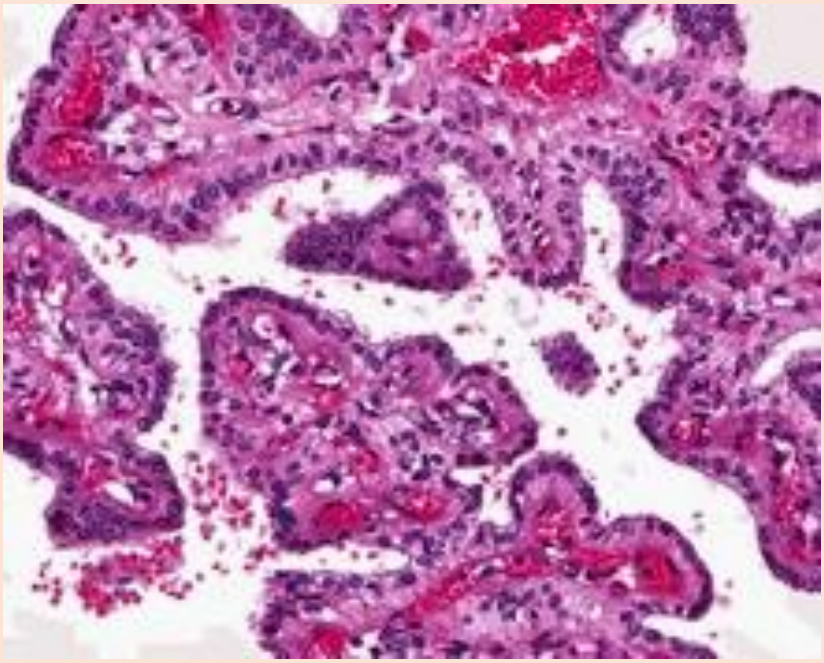

Figure 2: Endolymphatic sac tumour enunciating papillary configurations lined by columnar epithelium with uniform nuclei and pale cytoplasm. Focal haemorrhage is observed.

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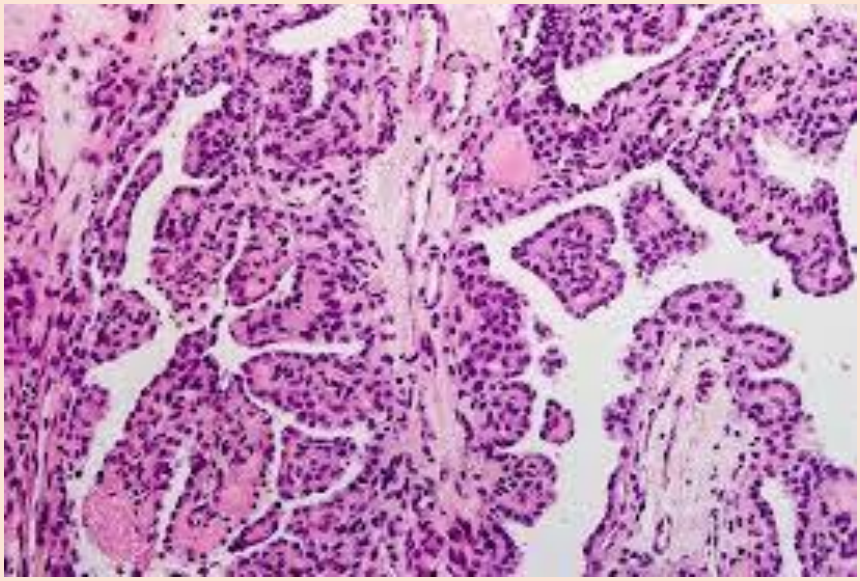

Figure 3: Endolymphatic sac tumour exemplifying papillary structures lined by columnar epithelium, uniform nuclei and intervening fibro-vascular stroma.

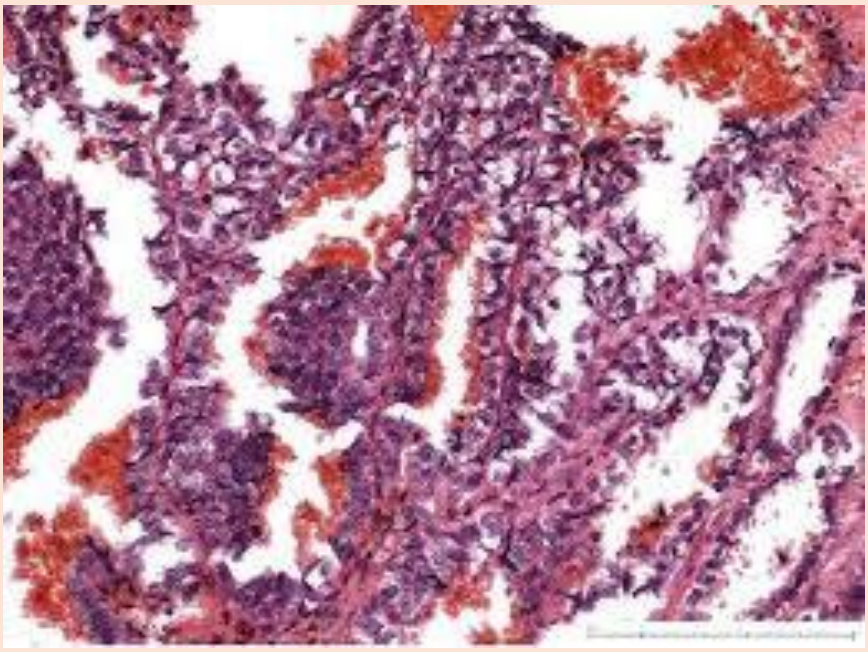

Figure 4: Endolymphatic sac tumour displaying papillary architecture with lining columnar epithelium and hemosiderin pigment deposits.

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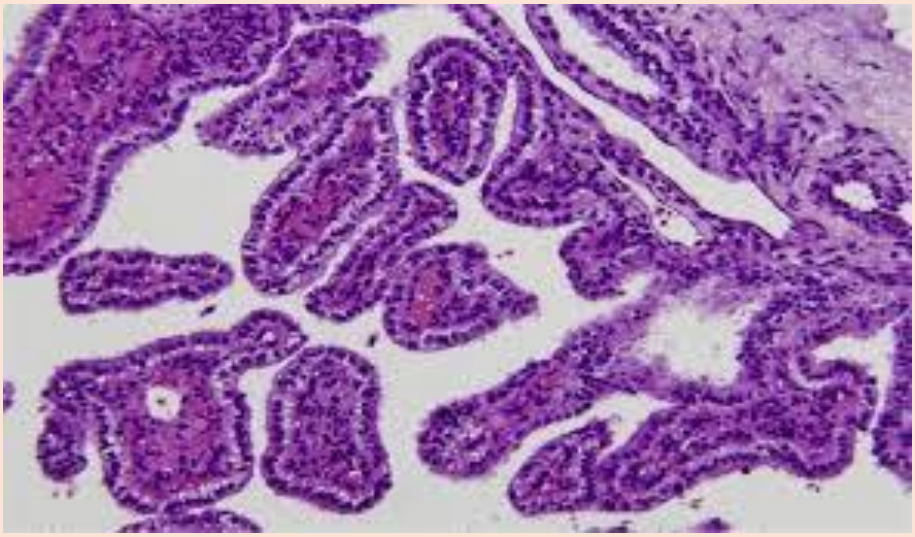

Figure 5: Endolymphatic sac tumour delineating papillary articulations layered with uniform columnar epithelium, fibrotic stroma and focal haemorrhage.

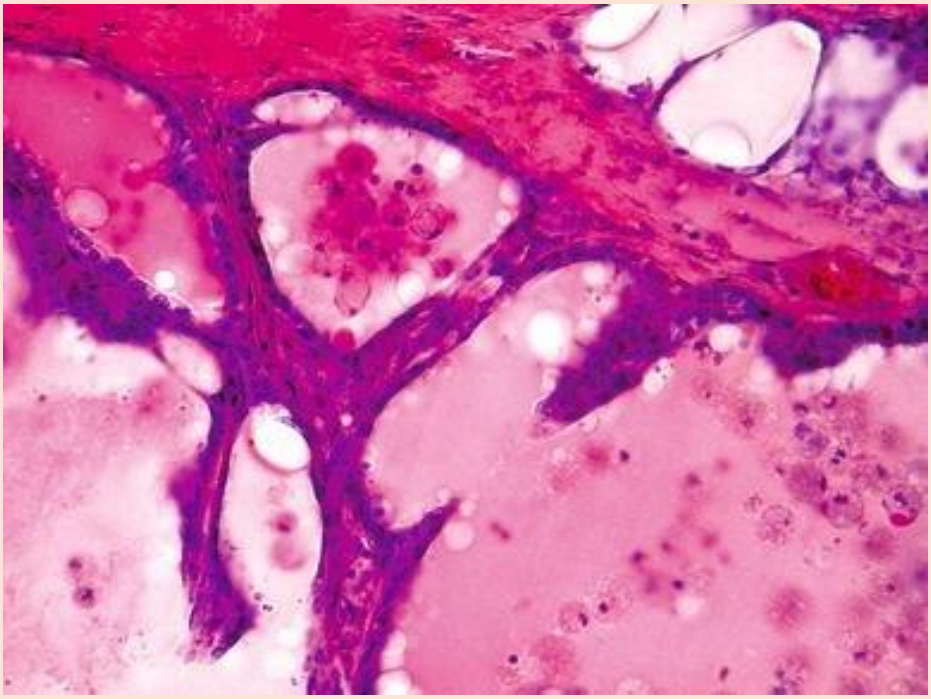

Figure 6: Endolymphatic sac tumour exhibiting papillary projections lined by columnar epithelium and abutting bony fragments.

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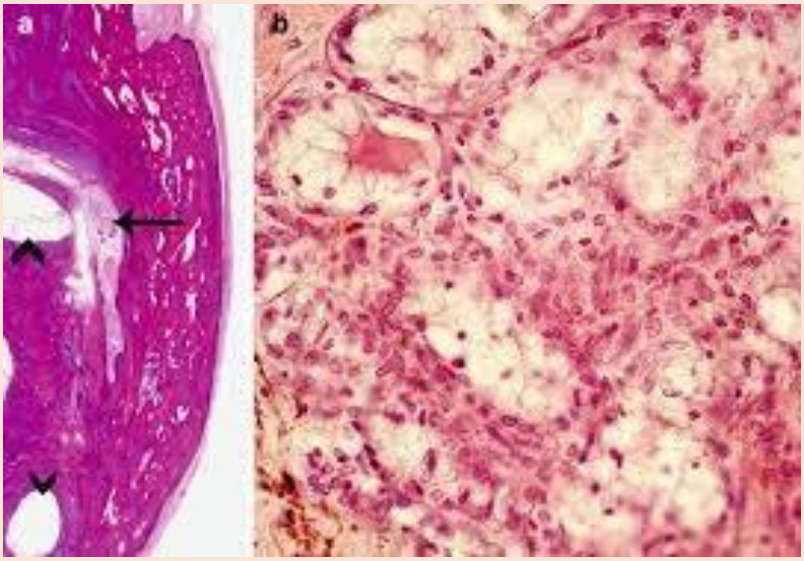

Figure 7: Endolymphatic sac tumour enunciating papillary architecture lined by cuboidal epithelium with regular nuclei and surrounding fibro-vascular stroma.

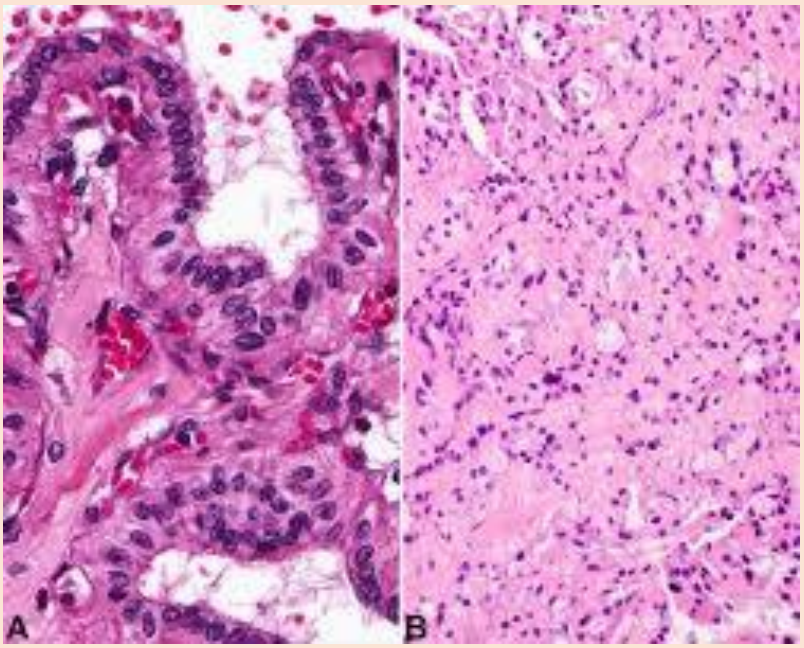

Figure 8: Endolymphatic sac tumour depicting papillary articulations lined by cuboidal epithelium with pale cytoplasm and uniform nuclei with encompassing fibrotic stroma.

Differential Diagnosis

Endolymphatic sac tumour requires a histological segregation from neoplasms such as middle ear carcinoma, choroid plexus papilloma, para-ganglioma, papillary ependymoma, metastatic

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renal cell carcinoma and metastatic thyroid papillary carcinoma [7,8]. Middle ear adenoma is a neoplasm demonstrating variable neoplastic configurations such as cellular sheets, solid areas, trabeculae, cystic articulations, cribriform pattern or glandular structures. Generally, papillary architecture is absent. Tumour glands or tubules are coated by uniform, singular layer of cuboidal or columnar epithelial cells incorporated with variable quantities of eosinophilic cytoplasm and spherical to elliptical, hyperchromatic nuclei with eccentric nucleoli. Neoplastic cells may exhibit significant pleomorphism. The circumscribing stroma is sparse, fibrotic or myxoid. Mitotic activity is minimal to absent. Tumour necrosis is absent. The neoplasm lacks infiltration or destruction of circumscribing bone [7,8]. Middle ear carcinoma is constituted of tumour cells displaying significant cellular and nuclear pleomorphism, nuclear anaplasia, frequent mitotic activity, foci of tumour necrosis and infiltration of adjoining bone or soft tissue. Neoplastic glands depict an absence of dual cell layer and a singular layer of luminal epithelial cells may be observed. Well differentiated neoplasms may simulate an adenoma although may delineate an invasive pattern of tumour evolution. The neoplasm can represent as adenoid cystic carcinoma or muco-epidermoid carcinoma. Tumefaction expands within the meso-tympanum and is devoid of bony infiltration and destruction [7,8].

- Choroid plexus papilloma-carcinoma originates within the brain ventricles. Choroid plexus papilloma exhibits papillary architecture with a singular layer of monomorphic, cuboidal to columnar epithelial cells coating the papillae. Mild nuclear pleomorphism, exceptional mitotic activity and absence of tumour necrosis is observed. Choroid plexus carcinoma is a cellular, malignant neoplasm enunciating nuclear pleomorphism with hyperchromatic nuclei. Papillary architecture is diminished with emergence of solid areas. Mitotic figures are frequent and focal tumour necrosis is exemplified. Tumour cells are immune reactive to S100 protein [7,8]
- Jugulotympanic paraganglioma is a vascular neoplasm which demonstrates tumour cells nests or an organoid pattern with the configuration of “zellballen”. However, papillary-cystic articulations are absent. Classic, organoid or nesting tumour cells depict centrally placed spherical or elliptical chief cells imbued with abundant, eosinophilic, granular or vacuolated cytoplasm and uniform nuclei with dispersed chromatin. Spindle-shaped, basophilic, peripheral sustentacular cells are disseminated which are immune reactive to S100 protein. Tumour cell nests are segregated by prominent fibro-vascular stroma. Occasionally, dense fibrous stroma circumscribes tumour cell aggregates. An infiltrative pattern of tumour evolution is encountered. Cellular and nuclear pleomorphism is enunciated although mitotic figures or foci of necrosis are exceptional. Glandular or alveolar differentiation is absent. Tumour cells are immune non-reactive to keratin [7,8]
- Metastatic renal cell carcinoma arises from diverse primary neoplasms such as breast, gastric mucosa, well differentiated neuroendocrine carcinoma or urothelial carcinoma wherein metastatic tumour cell aggregates are confined to the renal parenchyma. The morphological representation is diverse, may simulate urothelial carcinoma with divergent

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differentiation or unusual, primary renal neoplasms. Tumour cells arising from various primary neoplasms are immune non-reactive to S100 protein, glial fibrillary acidic protein (GFAP) or synaptophysin. Metastatic renal cell carcinoma can be appropriately delineated upon Computerized Tomography (CT) [7,8]

- Metastatic thyroid carcinoma demonstrates characteristic nuclear features. Tumour deposits may appear within multiple areas of thyroid gland parenchyma. Deposits can be miniature and occur within lympho-vascular spaces or emerge as enlarged tumour masses. Generally, tumour metastasis appear as moderately differentiated or poorly differentiated adenocarcinoma. Tumour cells are immune reactive to thyroglobulin [7,8]
- Upon cogent imaging, endolymphatic sac tumour requires a segregation from glomus jugulare tumour wherein centric segment of the lesion appears at the jugular bulb rather than vestibular aqueduct. The neoplasm enunciates a classic, organoid pattern with configuration of “zellballen” or nesting architecture. Tumour cell aggregates appear as centric, spherical to elliptical chief cells imbued with abundant, eosinophilic, granular or vacuolated cytoplasm and uniform nuclei with dispersed chromatin. Spindle-shaped, basophilic, peripheral sustentacular cells circumscribe the cellular nests which are segregated by prominent, fibro-vascular stroma. Occasionally, dense fibrous stroma may circumscribe tumour cell clusters. Foci of tumour cell infiltration are observed. Mitotic activity or focal tumour necrosis is exceptional. Glandular or alveolar differentiation is usually absent [7,8]
- Enlarged vestibular aqueduct is associated with expansion of aqueduct depicting a smooth perimeter. Also, normal configuration of the aqueduct may be preserved [7,8]
- Petrous apicitis is a condition arising within petrous air cells. Besides, bone circumscribing the aqueduct is un-aerated. The condition may represent as osteomyelitis within non-pneumatized bone or as an osteitis engendered from infected and obstructed air cells constituting pneumatized apex of petrous bone. Approximately 30% individuals delineate an asymmetric, pneumatized petrous apex [7,8]
- Cholesterol granuloma is commonly located within the petrous apex rather than the vestibular aqueduct. The non-encapsulated lesion demonstrates fibrosis, foreign body giant cell reaction, aggregates of chronic inflammatory cells, necrosis of mature adipose tissue, focal calcification, hemosiderin pigment deposits and cholesterol clefts comprised of needle-like, vacant spaces [7,8]
- Meningioma exhibits hyper-ostotic foci of subjacent bone. Tumefaction is composed of spherical, elliptical or spindle-shaped cells configuring whorls, nests or lobular pattern. Tumour cells enunciate indistinct cell borders and are imbued with pale cytoplasm, punched out nuclei and intra-nuclear cytoplasmic inclusions. Psammoma bodies may be discerned along with microscopic foci of bone infiltration. Tumour cells are immune reactive to Epithelial Membrane Antigen (EMA) and vimentin [7,8]

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Secondary metastasis within bone exemplifies tumour cells with diverse morphological configurations pertaining to the primary neoplasm. Epithelial neoplasia articulate tumour acini, solid sheets, columns and cords of epithelial cells along with a papillary pattern. Tumour cells exhibit morphological and functional similarity to the parent neoplasm and enunciate features of malignant metamorphosis such as loss of polarity, cellular and nuclear pleomorphism, enhanced nucleo-cytoplasmic ratio, anisonucleosis, anisocytosis, hyperchromasia and significant mitotic activity with atypical mitosis. Multinucleated tumour giant cells or enlarged tumour cells with singular, bizarre nuclei are observed [7,8].

Investigative Assay

Endolymphatic sac tumour commonly arises within the vestibular aqueduct and incriminates endolymphatic sac or duct. Thus, the lesion is contemplated to be situated within the posterior retro-labyrinthine petrous bone [9,10]. Upon cogent physical examination, unilateral deafness can be observed. Upon plain radiography, tumefaction appears to arise from posterior medial petrous ridge of temporal bone, the site of endolymphatic sac. Features of bone destruction are discerned upon imaging [9,10]. Upon computerized tomography, erosion of petrous bone displays an infiltrative or "moth-eaten" configuration. Centric calcified spicules, intra-tumour and posterior rim calcification is frequently observed. Intense, homogeneous enhancement of tumefaction is common [9,10]. Computerized tomography (CT) of the cerebrum demonstrates a bulky, enhancing soft tissue mass of variable magnitude confined to cranial fossae or temporo-occipital region. Bony erosion of base of skull and middle or posterior cranial fossa may be observed. Linear, dense spicules of calcification and residual bone are delineated following localized tumour invasion and destruction of bone. Compression of ventricles may engender hydrocephalu [9,10]. Computerized Tomographic Angiography (CTA) of cerebral and cervical vascular articulations demonstrate a hyper-vascular lesion which is predominantly perfused by external carotid artery and branches of subclavian artery [9,10]. Angiographic evaluation is pertinent for discerning the neoplasm and assessing interventional or intravascular embolism. Vascular perfusion of the hyper-vascular endolymphatic sac tumour is obtained by external carotid artery, branches of ascending pharyngeal artery or occipital artery. Also, internal carotid artery may perfuse the neoplasm [9,10]. Preoperative embolization contributes to decimated intraoperative haemorrhage [9,10]. Magnetic Resonance Imaging (MRI) can enunciate a patchy, massive, irregular, heterogeneous, lobulated neoplasm confined to cranial fossae or temporo-occipital region. The neoplasm is hypo-intense upon T1 weighted imaging and demonstrates a heterogeneous or mixed signal intensity upon T2 weighted imaging [9,10]. Alternatively, upon T1 weighted imaging hyper-intense tumour foci are observed. Upon T1 weighted imaging with gadolinium contrast, heterogeneous tumour enhancement is exemplified, especially of non-cystic neoplastic component [9,10]. MRI displays a heterogeneous signal intensity upon T1 weighted imaging and T2 weighted imaging.

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Haemorrhagic foci appear markedly hyper-intense upon T1 weighted imaging and T2 weighted imaging. Hypo-intense foci are attributed to zonal tumour necrosis, calcification or residual bone. Enhanced MRI scan depicts a heterogeneous intensity upon T1 weighted imaging and flow voids upon T2 weighted imaging. Magnetic resonance Diffusion-Weighted Imaging (DWI) represents hypo-intense signal intensity. A heterogeneous tumour enhancement is observed with administration of contrast medium [9,10].

Therapeutic Options

Comprehensive surgical excision of the neoplasm is an optimal treatment strategy. Cogent surgical approach with transmastoid, posterior fossa or retrolabyrinthine-transdural access is commonly utilized. Surgical procedures such as mastoidectomy or temporal bone resection with probable excision of cranial nerves may be necessitated [9,10]. With comprehensive surgical extermination of the neoplasm tumour reoccurrence is absent whereas subtotal resection may be accompanied by localized tumour reoccurrence. However, surgical extermination of late-stage neoplasm may be challenging on account of anatomic complexity and distinctive tumour extension. The infiltrative, destructive neoplasm is accompanied by significant haemorrhage during surgical procedures and tumour-associated mortality may ensue [9,10]. Adjuvant radiotherapy can be optimally employed to treat repetitive or mammoth neoplasms unamenable to surgical resection. Nevertheless, advantageous adoption of manoeuvers such as radiotherapy or gamma knife surgery for appropriate tumour alleviation remains debatable [9,10]. An estimated duration of 10 years of monitoring of the neoplasm is considered appropriate [9,10]. Genetic evaluation is contemplated to be a pertinent investigation for assessing impending neoplasms. An annual MRI is beneficial for cogent tumour assessment [9,10].

Conflicts of Interests

The authors declare that have no competing interest and not any conflict of interest.

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