Case Series

Radiologist as the Crux of Management in Incidentally Detected Gorlin-Goltz Syndrome: Case Series

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A B S T R A C T

Introduction: Gorlin-Goltz Syndrome (GGS) is characterised by a constellation of features involving multiple organ systems, of which, Odontogenic keratocysts (OKC), Basal cell carcinoma (BCC), dense Falx calcifications and rib anomolies are pathognomonic. Among these, BCC poses a threat due to its local aggressiveness and malignant potential. OKCs also have a risk of malignant transformation into ameloblastoma or squamous cell carcinoma. The purpose of this case series is to emphasise the role of a radiologist in the diagnosis and management of incidental GGS whilst providing a brief review of literature.

Case report: Two middle aged female patients and a 9 year old female child with jaw swelling, headache and fever respectively were referred to the department of radiology ESIC Superspeciality Hospital, Sanathnagar, Hyderabad, India for further evaluation. Multiple imaging findings in these patients triggered the suspicion of GGS. Clinical examination revealed the presence of indolent skin lesions in 2 of them, which were biopsied. No relevant family history was noted in the adults. Skin lesions were biopsied and proven to be BCCs. With the presence of multiple major and minor criteria being detected, a diagnosis of GGS was made.

Conclusion: By suspecting GGS in the presence of triggering imaging findings, a radiologist can alert the clinician for evaluation of the skin lesions. Thus safe-gaurding the patient from morbidity and mortality caused by BCC, which is a hazardous entity amongst all the other syndromic components.

Keywords: Gorlin-Goltz Syndrome; Basal Cell Carcinoma; Nevus; Odontogenic Keratocysts; Falx Calcifications; Bifid Ribs

INTRODUCTION

Gorlin-Goltz syndrome a.k.a Gorlin syndrome has numerous synonyms like basal cell nevus syndrome, 5th phakomatosis, bifid-rib basal cell nevus syndrome, hereditary cutaneo-mandibular polyoncosis etc. Tissues of multiple organ systems are affected but the triad of basal cell carcinoma(BCC), odontogenic keratocysts (OKC), and rib abnormalities is pathognomonic. Additionally, dense falx calcifications, palmar/ plantar pits and positive family history form the major criteria. More than 100 minor criteria have been described in the literature.[1] (Table 1). Presence of atleast two major criteria or one major criterion with two minor criteria will suffice for the diagnosis of GGS(Table 2). The syndrome has been relatively under-reported in Indian Radiology literature[2] . OKC, BCC, and medulloblastoma are the usual presenting symptoms, which trigger the suspicion of GGS in the clinician (dentist, dermatologist and paediatrician respectively) and further imaging is performed to confirm the diagnosis and determine the disease extent. OKCs being the consistent and most common presenting feature, dental literature has published a major proportion of the GGS cases in India.[3]

However, the syndrome being suspected and confirmed primarily in the department of Radiodiagnosis based on ancillary imaging findings is a rarity and this article illustrates case reports of that scenario.

CASE REPORT 1

A 30-year-old female patient, with complaints of pain and swelling in the left jaw since two months, consulted a dentist with the onset of purulent discharge from the lower left 2nd molar tooth. Orthopantomogram revealed multiple bilateral mandibular oval radiolucent lesions. The patient was then referred to our department for a CT of the mandible and paranasal sinuses to assess the extent of the disease.

On examination, mild ocular hypertelorism was noted. CT revealed four well defined expansile lytic lesions involving bilateral rami, angles, and posterior body of the mandible, with impaction of the unerupted 3rd molars (Figure 1). Medial cortical breach and intra-lesional air foci were noted suggesting infection. Similar lesions were noted bilaterally in the maxilla, with impacted upper 3rd molars and superior extension with bowing into the maxillary sinus antrum. Findings were consistent with the imaging features of

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odontogenic keratocysts or dentigerous cysts.

Diffuse discrete thick laminar dural calcifications were noted in the falx cerebri (Figure 2), along with scattered calcifications in the tentorium cerebelli. Calcified diaphragma sellae was noted bilaterally causing lateral sellar bridging. Further imaging revealed a bifid left 4th rib (Figure 3), spina bifida occulta of 7th cervical and 1st dorsal vertebrae on CT (Figure 4) and bilateral ovarian solid hypoechoic masses with calcifications and perilesional vascularity with normal RI values on ultrasound, which were suggestive of ovarian fibromata (Figure 5).

As the imaging findings triggered a suspicion of GGS, the patient was further inquired for any family history or the presence of any cutaneous lesions. She denied the former but revealed the presence of a slow growing painless lesion on the right lower back. On examination, a 1 x 1 cm hyperpigmented maculopapular lesion with regular margins was noted on the right side at the level of the lumbar fossa (Figure 6). Excision biopsy was performed by a dermatologist, considering the possibility of the lesion being a melanoma or a dysplastic nevus. Histopathology revealed trabeculae and islands of basaloid cells with peripheral palisading, ill-defined cytoplasm and mitosis suggestive of a Basal cell carcinoma, with the tumor reaching one edge of the biopsy margin and the underlying subcutis appearing free.

The dental cysts were enucleated and histopathologically confirmed to be odontogenic keratocysts. With the presence of 4 major criteria (odontogenic keratocysts, laminar dural calcifications, Basal cell carcinoma, rib anomolies) and 4 minor criteria (ovarian fibroma, lateral sellar bridging, vertebral anomolies, hypertelorism), a diagnosis of Gorlin-Goltz Syndrome was made.

Due to financial and personal constraints, the patient could not undergo genetic analysis.

CASE REPORT 2

A 35-year-old female patient was referred to our department for a CT brain for evaluation of chronic headache. On clinical examination, the patient was of a tall (173cms) stature (Marfanoid built), had bilateral post-axial polydactyly of the feet with hallux valgus (Figure 7) few palmar pits (Figure 8), and disseminated maculopapular hyperpigmented well marginated cutaneous lesions predominantly on the face, trunk and calves which were suspicious for dysplastic nevi/ basal cell carcinoma (Figure 9).

Thick bilaminar diffuse dural calcifications involving the falx cerebri, tentorium cerebelli and petro-clinoid ligaments (Figure 10) were noted on CT, with calcified diaphragma sellae causing bilateral lateral sellar bridging. A well-defined hypodense lytic lesion (measuring 1.6 x 1.2 cms) was seen in the left hemi-maxilla adjacent to the 2nd molar tooth, with margins bowing into the maxillary sinus antrum- suggestive of an odontogenic keratocyst or a dentigerous cyst (Figure 11). Spina bifida occulta of c6 vertebral body was noted.

Organ/System	Features in GGS
SKIN	Pigmented BCCS, Palmar/plantar pits, Milia, Comedones, Epithelial cysts, Dyskeratosis
RIBS	Splaying, Bifid, Synostosis, Cervical
SPINE	Hemivertebrae, Block, Spina bifida occulta, Kyphoscoliosis
CRANIOFACIAL	Bilamellar falx and tentorial calcifications, lateral bridging of sella, Frontal and parietal bossing, Macrocrania, Coarse face, Cleft lip and palate
BRAIN	Medulloblastoma, Meningioma, Agenesis of corpus callosum, Congenital hydrocephalus, Mental retardation, asymmetry of lateral ventricles
EYES	Hypertelorism, Strabismus, Dystopia canthorum, Congenital blindness
SKELETAL	Shortening of 4th metacarpal, Polydactyly, Syndactyly, Hallux valgus, Pectus excavatum/carinatum, Sprengel shoulder, Marfanoid build, Flame shaped lucencies in tubular bones and phalanges
CARDIOVASCULAR	Cardiac fibromas
GENITOURINARY	Calcifying ovarian and uterine fibromata (females); Cryptorchidism and hypogonadism (males); Minor renal anomalies; Lymphomesenteric cysts
OTHER NEOPLASMS	Melanoma, Neurofibroma, Rhabdomyosarcoma, leiomyoma

Table 1: Spectrum of Findings in Gorlin-Goltz Syndrome[5].

Criteria			Patient 1	Patient 2	Patient 3
Major	Multiple BCC's or 1 BCC less than 20 years		✓	✓	×
	Rib Anomalies (bifid, splayed, fused)		✓	✓	✓
	OKC's		✓	✓	✓
	Palmar &/or Plantar pits		×	✓	×
	Falx cerebri & cerebelli bilamellar calcifications		✓	✓	✓
	1st degree relative with NBCCS		×	×	✓
Minor	Macrocephaly (height adjusted)		×	×	×
	Congenital malformations (cleft lip / palate, frontal bossing, coarse phase, moderate to severe hypertelorism)		✓	×	×
	Skeletal abnormalities (syndactyly, polydactyly, Sprengel's shoulder, pectus deformity)		×	✓	×
	Radiological abnormalities	Bridging of Sella	✓	✓	×
		Vertebral anomalies (hemivertebrae, fusion, elongation)	✓	✓	×
		Modelling defects of hands and feet	×	×	×
		Flame shaped hands and feet	×	×	×
	Ovarian Fibromata		✓	×	×
	Medulloblastoma		×	×	×
	Others (hyperdense renal cysts, marfanoid build, intracranial anomalies- callosal or septal anomalies)		×	✓	✓

Table 2: Major and Minor criteria of GGS in our patients.

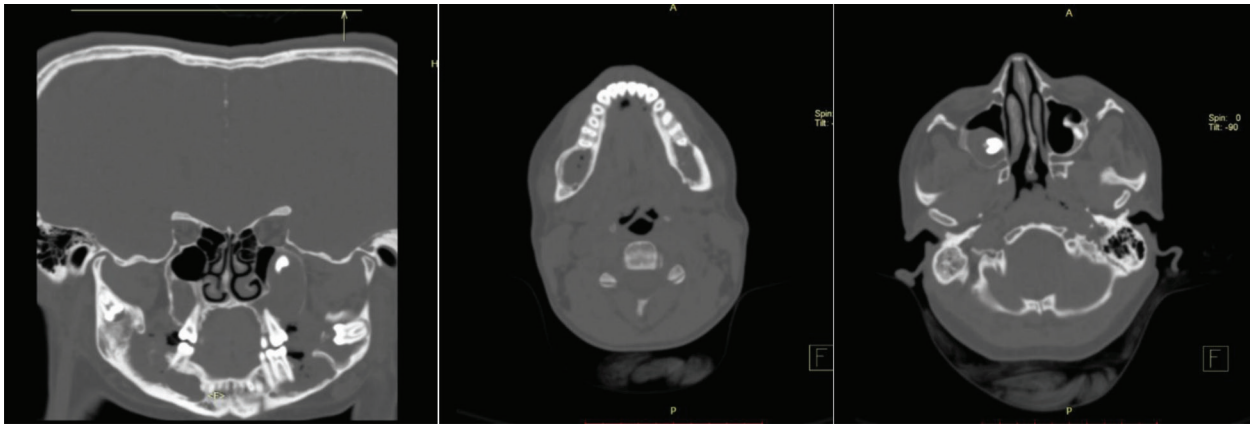

Figure-1: CT Curved MPR, Axial bone window images demonstrating maxillary and mandibular odontogenic keratocysts, with trapped unerupted molars, intralesional air foci and medial cortical breach.

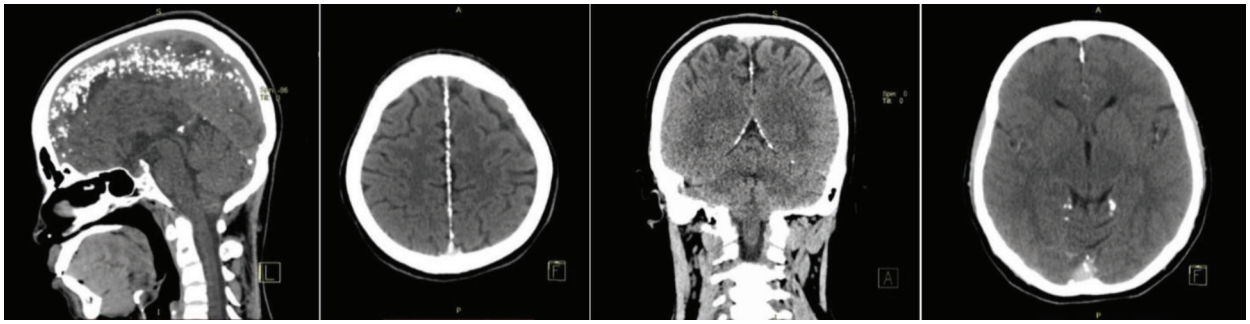

Figure-2: CT (brain window) images demonstrating thick bilamellar falx cerebri and tentorium cerebelli calcifications.

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Screening chest topogram revealed splaying of anterior ends of bilateral 1st ribs (Figure 12). Ultrasound and CT revealed the presence of a complex left ovarian cyst (Figure 13) and multiple hyperdense renal cysts (Figure 14).

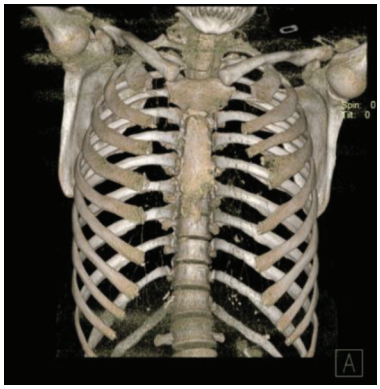


Figure-3: Chest VRT image revealing bifid left 4th rib.

An 11-site wide local excision was performed for the larger cutaneous lesions on the face, trunk, and lower extremity under sterile aseptic precautions by the dermatologist. Histopathology confirmed the diagnosis of MULTIFOCAL SUPERFICIAL BASAL CELL CARCINOMA (Figure 15) with infiltrative lesions arising from the basal layer of epidermis, arranged in the form of nests extending into dermis. The nests demonstrated palisading polygonal cells with hyperchromatic nuclei, atypical mitosis, peritumoral clefts and melanin laden macrophages between them. The maxillary lytic lesion was not biopsied as the patient was asymptomatic and the lesion was solitary.

With the presence of four major criteria (multiple BCCs, Falx calcifications, palmar pits, splayed ribs), one probable

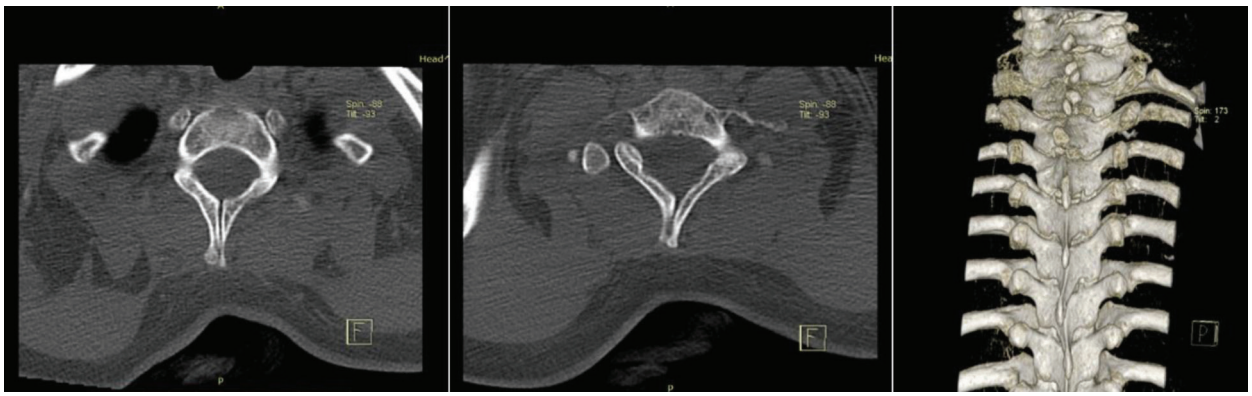


Figure-4: CT axial bone window and VRT images demonstrating spina bifida occulta of 7th cervical and 1st dorsal vertebrae.

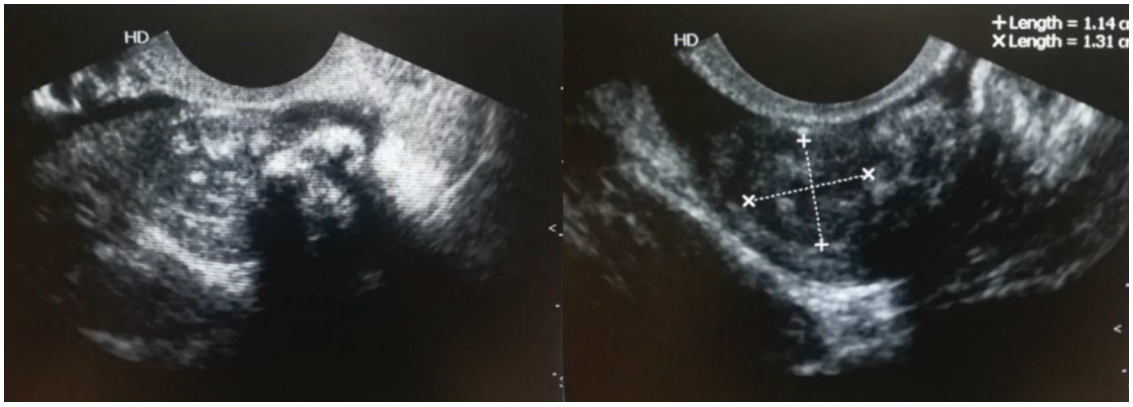


Figure-5: ovarian fibromata on Grey scale Ultrasound

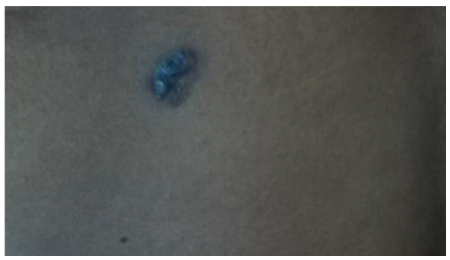


Figure-6: Indolent BCC in patient 1.

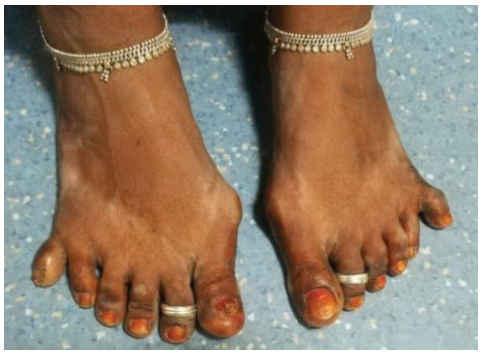


Figure-7: Bilateral post-axial polydactyly with Hallux valgus in patient 2.

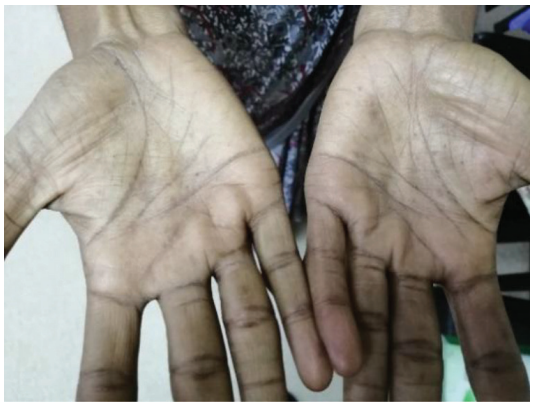


Figure-8: Numerous palmar pits.

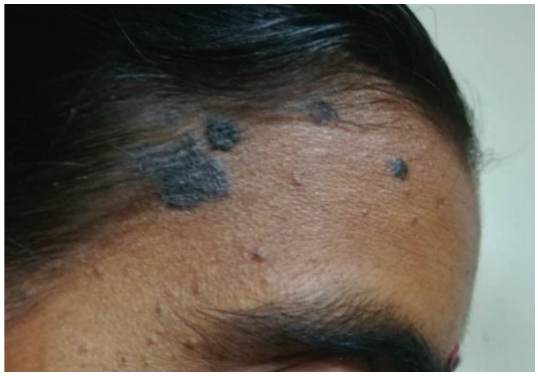
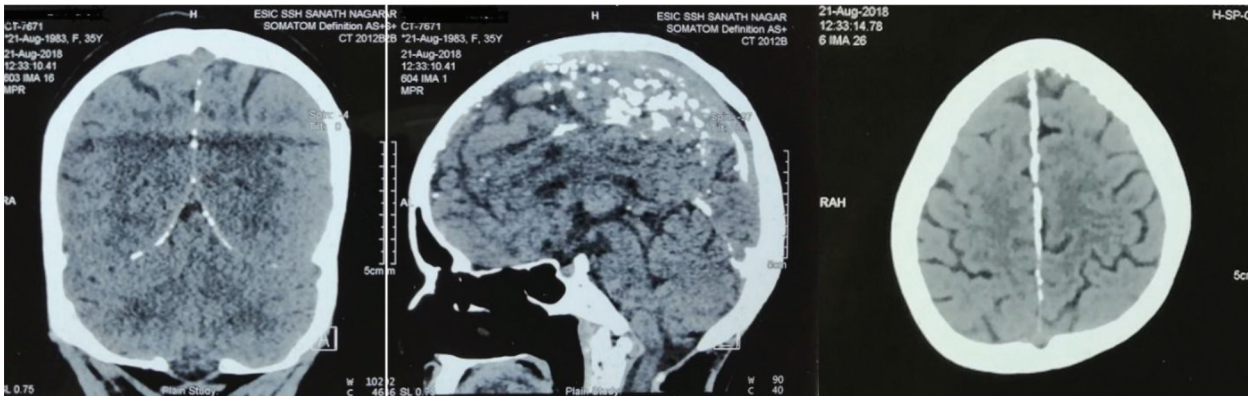


Figure-9: Disseminated BCC.

major criterion (OKC in left hemimaxilla) and five minor criteria (polydactyly with hallux valgus, lateral sellar bridging, spina bifida, marfanoid built and hemorrhagic renal cysts), a diagnosis of GORLIN-GOLTZ SYNDROME was made. Molecular genetic testing for PTCHI/SUFU gene mutation was deferred due to financial constraints. The patient is now on regular surgical oncology follow up and was advised limited sun-exposure. There was no family history, and the patients first degree relatives were advised pre-symptomatic targeted mutation analysis.

CASE REPORT 3

A nine-year old daughter of patient 1, presented to the department of paediatrics with intermittent fever for the past three months. Routine haematological and urine investigations were inconclusive. Chest X-ray demonstrated the presence of Bifid 3rd right and left 5th ribs (Figure 16). As two major criteria were met (1st degree relative with GGS, Rib anolomies) the possibility of Gorlin-goltz syndrome was considered. Further investigations for detecting other minor and major criteria revealed few discrete Falx cerebri and Tentorium cerebelli calcifications (Figure 17), lytic lesion in left hemimaxilla around unerupted 2nd molar tooth (likely a dentigerous cyst/odontogenic keratocyst) (Figure 18), Absent septum pellucidum in the brain and tonsillar ectopia resulting in a crowded foramen Magnum (Figures 19,20). No significant abnormality was detected on ultrasound



Figures-10: Coronal, Sagittal and Axial CT Brain images revealing bilamellar coarse Falx cerebri and Tentorium cerebelli calcifications.

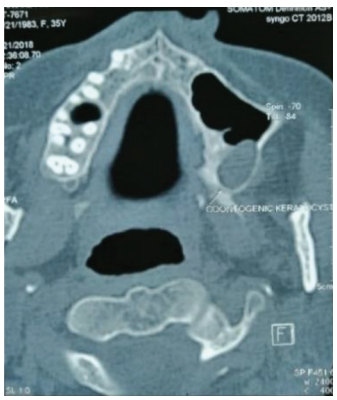


Figure-11: Lytic lesion in left hemi-maxilla around unerupted molar tooth and bulging into maxillary sinus antrum.

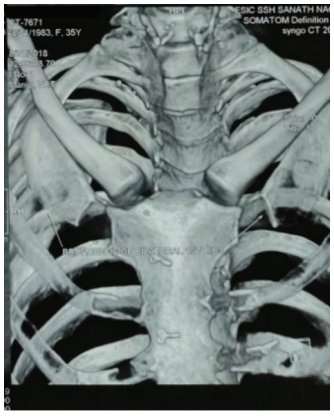
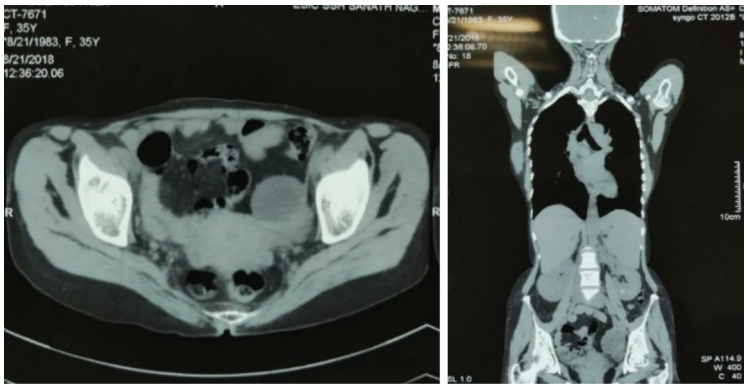


Figure-12: Chest VRT image demonstrating splaying of anterior ends of bilateral first ribs.



Figures-13, 14: Axial and Coronal CT images demonstrating left complex ovarian cyst with papillary wall projections, Hyperdense left renal lesions.

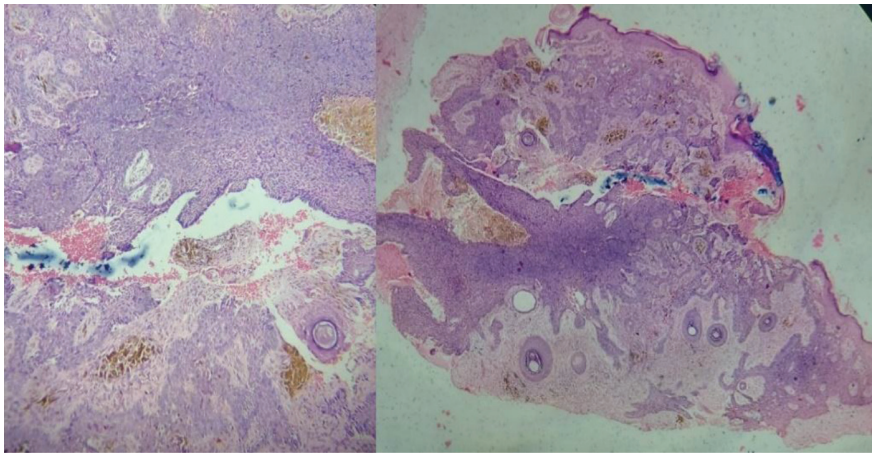


Figure-15: Histopathological images of the cutaneous lesions demonstrating features of BCC.

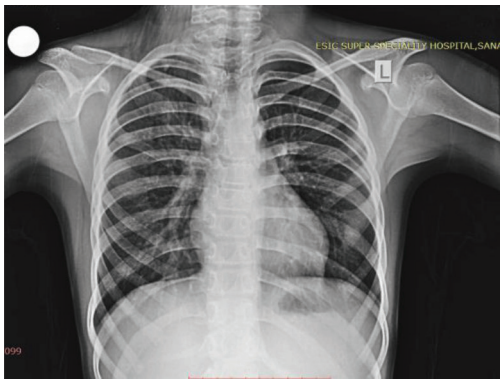


Figure-16: Chest Xray PA view in patient 3 showing Bifid right 3rd and left 5th ribs.

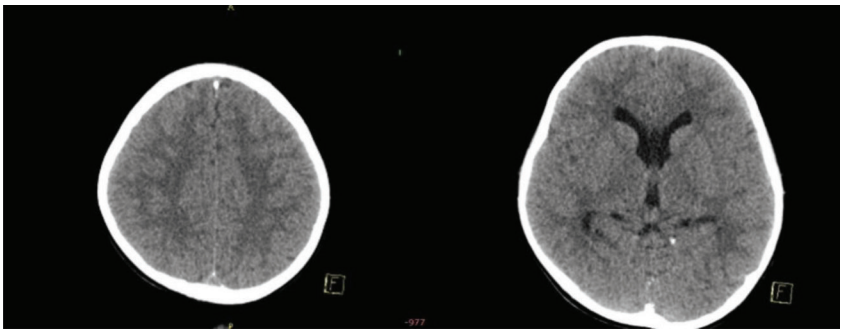


Figure-17: Axial CT Brain images show early falx cerebri and tentorium cerebelli calcifications.

abdomen. Thus, with two definitive major criteria, two possible major criteria and one minor criterion, a diagnosis of GGS was made.

DISCUSSION

GGS is a rare multisystem disorder with myriad benign and malignant entities. The incidence is variable with a general consensus of 1 in 56000 to 164000[4] and no specific gender predilection. GGS is an inherited autosomal dominant disease with high degree of penetrance. But the variability in expression of the syndromic components often results in a delayed diagnosis. [5,6] 35 -50% of the cases may occur due to a spontaneous mutation.[7]

Mutations in PTCH1 gene on long arm of chromosome 9q22.3 have been reported to be responsible for the inheritance of this syndrome.[8] Other sonic hedgehog pathway gene mutations such as PTCH2 (located on chromosome 1p32) and SUFU gene (on chromosome 10q24.32) in the absence of mutated PTCH1 gene have also been reported.[9]

According to the knudson 2-hit hypothesis, in patients with GGS, all the cells carry a native inherited mutation and receive a second hit in the form of a random somatic event like radiation, that results in loss of allele by mitotic dysjunction/deletion/recombination. In patients with sporadic onset of GGS, both the hits come from random somatic events. The former phenomenon, with a single second hit is responsible for BCC, OKCS, medulloblastomas, meningiomas, ovarian/cardiac fibromas and in physical anomolies, while the latter phenomenon with 2-hits is responsible for features like palmar/plantar pits.[6,8]

Initial description of a syndrome with multiple BCCS was done by Jarisch and white in 1894. [10] Brinkley and Johnson reported the case first in 1951.[5] It was Robert J.Gorlin and Robert W.Goltz who established the classical triad of the syndrome with BCC, OKC and bifid ribs in 1960. Rayner et al added the presence of falx calcifications and palmar/plantar pits to the criteria needed for diagnosis.[11]

Evans et al established the major and minor diagnostic criteria for GGS in 1994 which were eventually modified by Kimonos et al in 2004.[12,13]

BCCs are the common manifestations of GGS, occurring predominantly in head and neck followed by trunk and limbs.[14]

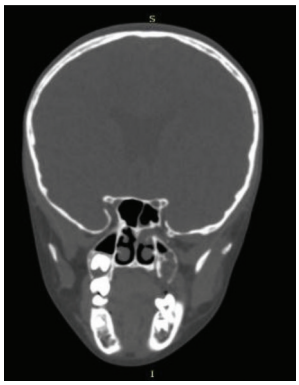


Figure-18: Left hemimaxillary lytic lesion shown on coronal CT bone window image – likely OKC.

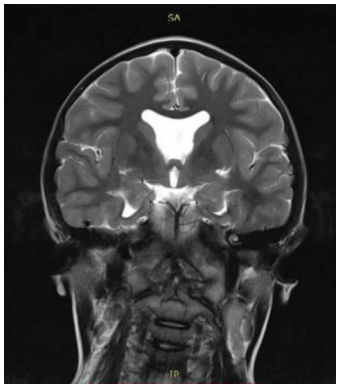


Figure-19: Coronal T2WI representing Agenesis of Septum Pellucidum.

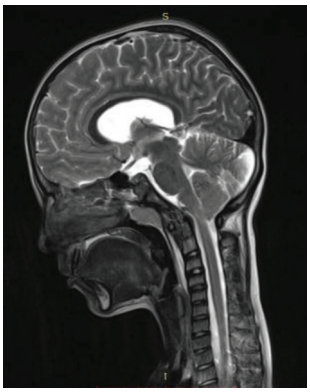


Figure-20: Sagittal T2WI showing Tonsillar ectopia with crowded Foramen Magnum.

BCCS are not the primary features in middle aged patients, particularly after the age of 30 years.[7] Incidence of BCCs in blacks with the syndrome is significantly less (approximately 40%) when compared to the whites (90%) which can be attributed to the protection conferred by melanin.[8] 0.4% of the patients with BCC turn out to be positive for GGS.[8] The spectrum of morbidity due to BCC ranges from cosmetic issues to death from metastasis. Documented metastatic potential is 0.0028 to 0.55%[15] and deaths due to lung metastasis have been reported. Severity of BCC is thus a prognostic factor.

KCOTs are considered the most consistent component of GGS with an incidence of nearly 75 to 100%.[3] Compared to the general population, KCOTs occur a decade earlier in syndromic patients, with peak incidence in 2nd to 3rd decade.[5] Mandible is involved (69%) more than the maxilla (31%).[16] Complications associated with KCOTs are superadded infection, pathological fractures, malignant transformation into squamous cell carcinoma or ameloblastoma.[17] Although pantomographs suffice for initial diagnosis, CT helps in detecting small cysts not evident on X-Ray, can define the extent of the cysts and demonstrate complications such as intra-cystic infection, eroded walls and displacement of teeth.[5] Enucleation and marsupialization[2,18] are the treatment options available with a high rate of recurrence due to the presence of small satellite cysts. Therefore, a biannual follow up for a period of five years followed by annual surveillance is recommended.[19]

A higher incidence of rib anomalies and male predeliction, with a lower incidence of BCC, medulloblastoma, syndactyly/polydactyly, family history, pectus deformity has been noted in India in contrast to the rest of the world, thus depicting a wide ethnic variation in the manifestations of the components of GGS.[2]

A multidisciplinary preventive and therapeautic protocol is deemed necessary, with the involvement of Dermatology, Genetics, Paediatrics, Neurology, Cardiology, Gynecology, Oral and maxillofacial surgery, Otorhinolaryngology, ophthalmology and psychiatry.[6]

An early diagnosis becomes imperative for a multitude of reasons, foremost being Radiation exposure. GGS patients are extremely sensitive to radiation as they tend to accumulate P53 in exposed cells.[20] Development of numerous BCCs in the field of irradiation has been documented in patients undergoing Radiotherapy for medulloblastoma. Radiation induced secondary intracranial neoplasms like meningioma, ependymomas have been reported in several studies.[21] Hence radiotherapy is strictly contraindicated and Judicious imaging is advised in these patients.[6] Limited sun and UV exposure is also a part of their medical management.[2] Regular surveillance is mandatory, particularly in children, in view of development of syndromic CNS, GIT, GUT and skin tumors. Genetic counselling is vital in hereditary cases in view of 50% of the prospective children developing the syndrome. Antenatal screening can detect cardiac fibromas and macrocephaly and can help the obstetrician and pediatrician to plan an effective perinatal care.[2,8] Psychiatric counselling becomes essential to combat the morbidity induced by BCCs and the prolonged treatment.[6]

CONCLUSION

This article emphasizes on the importance of a Radiologist in suspecting sporadic GGS in the presence of triggering imaging features on non-contrast CT (such as bilamellar falx calcifications and OKCs). Further screening with radiography and ultrasound for other components of the syndrome, to meet the laid down criteria and proactive clinical evaluation of the patients for indolent skin lesions, within the imageology department can establish the diagnosis of the syndrome, which can be further confirmed by tissue diagnosis. As BCC is the high-risk component of the syndrome in middle aged patients, an early diagnosis and treatment can drastically decrease the morbidity and mortality. Genetic testing and

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counselling are necessary for at-risk pregnancies and for surveillance of patients for other tumors that are associated with the syndrome. Strict prohibition of radiotherapy and limited sun exposure can curb the occurrence of new BCCs in the field of irradiation. Expertise from multiple specialties and a life-long surveillance is needed to treat these patients.

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