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Quantitative and Qualitative Estimation of Sialic Acid with the reference to Cancer Patients

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ABSTRACT

Bosom disease is the most well-known sort of harmful tumor among ladies and their second driving reason for malignant growth related passings. The most widely recognized technique for screening and determination is mammography. In any case, two fundamental issues have been distinguished. To begin with, the portion of radiation got amid the test keeps the technique from the utilization on ladies who are < 40 years of age. Second, there can be mammogram disappointment attributable to the absence of tumor diverge from the stringy tissue. Along these lines, there is a requirement for screening techniques that will distinguish high-hazard cases. We built up an organic marker test that can recognize them. Expanded dimensions of sialic corrosive (SA) in spit are known to related with bosom disease. In this investigation, we assessed the plausibility of Raman spectroscopy as a technique for evaluation of SA in spit, utilizing citrate-diminished silver nanoparticles (cit-Ag-NPs) as a surface-improved Raman spectroscopy (SERS) substrate. Measurement of SA was cultivated by estimating its force in salivation and contrasting it and an alignment bend of SA models. The mean SA fixation in spit was observed to be essentially higher among 100 bosom malignant growth patients (18.3 ± 9.4 mg•dL– 1; mean ± SD) than among 106 solid controls (3.5 ± 1.0 mg•dL– 1). The SERS test demonstrated affectability of 94% and explicitness 98% for location of patients with bosom malignant growth, accepting that SA focus >7 mg•dL– 1 is a cutoff for positive test outcomes. Our discoveries demonstrate the handiness of this SERS system as a straightforward, helpful, and exceedingly delicate strategy for quantitative investigation of SA in spit. The straightforwardness of this nanotechnological test may help to generously decrease the mortality among patients with bosom malignant growth by furnishing ladies with a basic, noninvasive screening test that can be connected paying little mind to age or thickness of bosom tissue.

1. Introduction

Bosom disease is the most continuous threatening tumor among ladies on the planet, being the reason for in excess of 520,000 passings for every year; it is the second driving reason for malignant growth passings among ladies, being outperformed just by lung disease [1, 2]. Early location of bosom malignant growth, joined with a forceful and auspicious therapeutic intercession, is important to considerably decrease the passing rates among patients with this disease. The real identification techniques for bosom malignant growth, for example, X-beam mammography, ultrasonography, attractive reverberation imaging (MRI), figured tomography (CT), and positron emanation tomography (PET), have significant impediments, which influence their analytic viability. For example, CT and PET sweeps open individuals to a moderately high portion of radiation in correlation with different kinds of demonstrative tests and are probably going to cause new malignant growths in certain patients [3, 4]. X-beam mammography, the most widely recognized and generally financially savvy screening technique, is restricted by low affectability and explicitness. Up to 75% of false positive outcomes and 34% of false negative outcomes have been accounted for in a distributed report on X-beam recognition tests [3]. One of the principle burdens of mammography is that it somewhat expands the danger of radiation-incited bosom disease. This circumstance keeps mammograms from being generally connected to ladies more youthful than 40 years. On account of ladies having thick bosom tissue, a mammogram can fall flat on the grounds that the tumor differentiate is like that of a sinewy tissue. It has been discovered that practically 40% of ladies more established than 40 years have thick tissue. Furthermore, for more youthful ladies under 40, the thick fat bosom tissue covers a large portion of the tumors at their underlying stages, and X-beam mammography is regularly uncertain: when it is basic to distinguish a tumor so as to give legitimate treatment in a convenient way. As a result, bosom malignant growth is frequently identified just at further developed stages when the treatment alternatives are altogether increasingly constrained. The 5-year relative survival rate for ladies with stage 0 or stage I bosom disease is near 100%. Stage II and III bosom malignant growths yield a survival rate of ~93% and 72%, separately, and if the disease has achieved organize IV, prospects for survival among the patients are diminished to just 22% [2]. In this manner, there is a dire requirement for a quick, precise, generally cheap, and noninvasive strategy for early identification of bosom disease paying little heed to age and bosom tissue thickness. Sialic acids are a group of nine-carbon acidic monosaccharides that happen toward the finish of oligosaccharide chains of mucins, glycoproteins, and glycolipids appended to the outside of cells and solvent proteins [5, 6]. N-acetylneuraminic corrosive (Neu5AC) is the overwhelming type of sialic corrosive (SA) and nearly the main structure found in human natural liquids and

tissues [7]. The substance equation C11H19NO9 and structure of sialic corrosive are exhibited in Fig. 1. The SA particle is made out of a pyranose "seat" ring comprising of five carbon molecules and one oxygen iota, or "spine structure", to which a N-acetyl (N– CH3CO), a carboxyl gathering (CO– OH) and a glycerol "tail" are joined (– C3H7O3). Adjusted glycosylation is a widespread element of malignancy cells, and certain glycan structures are notable biomarkers of tumor movement [8, 9]. As of late, raised dimensions of SA have been appeared to be a trademark highlight of salivation of patients with bosom malignancy; subsequently, this measurement has been recommended as a noninvasive biomarker for determination or theranosis of this sort of disease [10, 11]. The utilization of salivation as an indicative medium is profitable in light of the fact that example accumulation is straightforward, noninvasive, and safe. Vinogradova et al. have appeared little measures of watery SA might be effectively distinguished by surface-improved Raman spectroscopy (SERS) estimations with citratereduced silver nanoparticles (cit-Ag-NPs) [11]. These

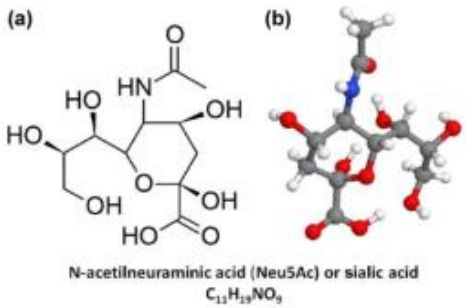

Figure 1 Chemical structure of Neu5Ac or sialic acid. (a) A schematic chemical model; (b) a stereographic model using color spheres; gray: carbon, red: oxygen, blue: nitrogen, white: hydrogen (mirror images).

results propose the likelihood of utilizing SERS for the discovery of SA in human salivation at the sub-atomic dimension to analyze bosom malignancy. The most broadly connected strategies for evaluation of SA in salivation are the acidic ninhydrin strategy portrayed by Yao K. [12] and thiobarbituric corrosive test altered by Skoza L. [13], which have breezed through the trial of time. In concentrates distributed as of late, evaluation of SA in spit keeps on being performed by these techniques [14, 15]. In any case, measurement of SA by SERS is a novel innovation that requires less reagents and could be valuable for clinical determination since it is exceptionally delicate, speedy, and shabby, while the hardware might be compact, and results can be gotten continuously. There are a few investigations utilizing SERS for the imaging of SAs on living cells [16, 17], however as far as anyone is concerned, there are no near reports on SERS in applications concerning its utilization for measurement of SA in human natural liquids or its orderly application to screening for them in human populaces. SERS is a Raman spectroscopic procedure that has appeared of the inelastic dispersing of the active radiation going from 106 to 1016 in enhancement factors from particles adsorbed on metals [18, 19]. Accordingly, SERS has an incredible potential as a molecularly explicit diagnostic apparatus for exceedingly delicate location of powerless Raman sign of proteins or other natural analytes with a little Raman dissipating cross-segment or at exceptionally low focuses, as low as the single-atom level [18– 21]. Besides, colloidal suspensions of metallic nanoparticles, principally silver, gold, or copper are the most widely recognized SERS substrates in light of the simplicity of readiness, long time span of usability, and high Raman signal upgrade factors [19– 21]. A standout amongst the most broadly utilized silver colloids is set up by the decrease of silver nitrate with trisodium citrate [22]. The concoction arrangement process results in silver nanoparticles with a surface secured by a layer of negative citrate particles to repay the positive charge of the NP surface. Cit-Ag-NPs have been shown to be successful at SERS location of decidedly charged analytes [23, 24] yet in addition some adversely charged atoms, including SA [11, 25, 26]. The point of this investigation was to look at the capacity of Raman spectroscopy to gauge convergences of SA in human spit, utilizing a colloidal suspension of cit-Ag-NPs as a SERS substrate. A progression of convergences of SA in water demonstrated a connection with the absolute power of their SERS spectra showing a decent monotonically expanding connection. The got alignment bend was connected to decide SA levels in salivation of solid ladies and patients with bosom malignant growth. Salivary SA fixations were observed to be fundamentally higher in bosom malignancy patients than in controls. These outcomes may prompt the improvement of an adaptable and financially savvy analytic technique for bosom malignant growth based on SERS.

2. Review of literature:

Malignancy may result from any one or a mix of compound, physical, organic and hereditary put-down to singular cells. A significant natural property shared by these specialists is their capacity to make harm or adjustment of cell DNA (Tannock and Hill, 1998). About 80% of human malignant growths are brought about by ecological variables, mainly synthetics (Doll and Peto, 1996).

Cancer of the Breast:

Carcinoma of the bosom, the most widely recognized malignant growth in ladies, is the third most basic disease on the planet, representing the most elevated dismalness and mortality in ladies. It is of genuine concern attributable to the rising frequency of the illness in the last 5-10 years (Parkin et al., 1999). Ladies determined to have bosom disease have relative survival rates of 96%, 79%, 67%, and 60% for 1,5,10 and 15 years individually (Wingo et al., 1998). Many inclining factors have been seen by Willett(1993). They are geographic circulation, hereditary inclination, expanding age, length of regenerative life, equality, age at the season of first youngster, heftiness, exogenous estrogen, oral contraceptives and fibrocystic changes in atypical, epithelial hyperplasia and carcinoma of the contralateral bosom or endometrium. Malignant growth of bosom once in a while happens before the age of 25, yet a consistent ascent has been seen at the season of menopause. The hazard increments with early menarche and late menopause and is more successive with nuiliparous than multiparous ladies. Ladies with first kid when more seasoned than 30 years are at expanded hazard. In light of the level of separation bosom tumors are evaluated as non-metastasising, metastasising and decently metastasising. The American joint board of trustees on disease ordered tumors into various stages as : Stage I - Tumor estimate < 2 cm in distance across, with no nodal inclusion. Stage II - Tumor

estimate < 5cm, portable axillary hub, with no far off metastasis. Stage 111 - skin contribution, pectoral chest divider obsession, axillary hub and inner mammary lymph hub association. Stage IV - any type of malignant growth with or without nodal association, pectoral obsession, yet with scattered metastasis. 2

Symptoms:

Bosom malignant growth presents itself as a dangerous or non harmful singular, unmistakable mass. Destructive bumps are hard, single and sporadic fit as a fiddle. Seeping from areola improve probability of essence of malignant growth. Different side effects which demonstrate its quality incorporate change fit as a fiddle of bosom, rash around the areola, knot in arm pit, unmistakable veins around the bosom, swelling of arms, ulceration of skin and manifestations of auxiliary tumors somewhere else. In cutting edge bosom malignancy in illustration of skin and dimpling, reversal of areola is watched.

Incidence:

Every year, bosom malignancy is analyzed in 910,000 ladies worldwide and 376,000 ladies bite the dust from the malady (WHO , 1997). There is a four to five overlay variety in bosom malignant growth rate rates crosswise over various nations. Age balanced rates for bosom malignant growth are 176% higher in created than in creating countries, with high frequency of articulation in North America and Europe (Parkin, 1998). In the United States, bosom disease represents most astounding occurrence and second most astounding death rate everything being equal. As indicated by the Surveillance, Epidemiology and End Results (SEER), 182,800 new cases and more than 40,800 passings from bosom malignant growth have been accounted for among ladies in the United States (Greenlee et ai., 2000). In Germany, bosom disease is in charge of 25% of female malignant growths and 18% of disease passings (Schleicher and Ammon, 1998).

In spite of the fact that the rate of bosom disease is lower in Asia, the biggest increment in rate rates was recorded in Japan and Singapore. Besides, while no noteworthy change in death rates have been accounted for in the U.S.A. Britain, Wales or Norway, there has been a 50-60% expansion in Japan and Singapore (Ursin et al., 1994). Transient examinations have appeared higher level of bosom malignant growth in South African Indians when contrasted with tribal Indians in the North West. Interestingly, low rates of bosom malignancy have been recorded in Asian Indians from the Indian subcontinent (Walker and Halse, 1999). In India, bosom malignant growth is the second most regular disease among ladies (Schaier and Lubin, 2000). The age balanced rates (AAR) of bosom malignancy in Chennai and Bangalore is 21.7, where as in Mumbai it is 24.4 (Yeole and Jussawala, 1992). A populace based survival contemplate in Chennai has demonstrated that the watched survival rates at 1,3 and 5 years are 80%, 58% and 48% individually, while the relative survival rates are 81%, 61% and 51% (Gajalakshmi et al., 1997). In Bangalore, the watched survival rate was 42.3% and the comparing relative survival rate was 46.8% (Nanda Kumar et al., 1995).

Epidemiological Evidences of Breast Cancer:

The primary perception in regards to a familial relationship with bosom malignant growth was performed over five decades back by Macklin (1954) who, in her spearheading work intended to search for the hereditary premise of human bosom disease, found a fundamentally higher recurrence of prostate malignancy among relatives of ladies with bosom malignancy than among relatives.of control gatherings.

Thiessen (1974), after examination of the familial rate and dispersion of all malignancies in a gathering of 145 bosom disease patients, contrasted and that of 139 randomized control patients, announced that fundamentally higher rates of just uterine, prostatic and bosom malignancy were found among both maternal and fatherly relatives of the bosom disease patients. On this premise, he recommended that the mammary organ is a piece of an incorporated genital organ framework whose various parts share one of a kind natural and neurotic attributes, including hormone responsiveness and malignant growth powerlessness. He likewise guessed the presence of some basic „etiological factor that could work in the advancement of tumors in differing regenerative organs, including bosom and prostate. Gun et al concerning the putative familial bunching of-bosom and prostate malignant growth have given inverse outcomes. In this manner, Isaacs et al (1995) in an investigation of families chose on account of the nearness of prostate malignant growth did not discover expanded dangers for disease at different locales, for example, bosom, ovary or endometrium. Negri et al (1997) did not watch a raised danger of prostate malignant growth in relatives of bosom disease patients. Hence, it appears to be certain that not all information on the potential relationship between bosom malignant growth in females and prostate disease in guys are univocal. In any case, various investigations preformed by various gatherings in populaces of various geographic beginning seem to show that a family ancestry of bosom disease may affect prostate malignant growth and the other way around.

3. Sialic acid with the reference to cancer patients

Molecular Mechanisms and Genetic Abnormalities of Breast Cancer:

Bosom malignant growth movement from ordinary tissue to obtrusive disease happens more than 5-20 years. It is driven by a progression of collecting hereditary changes that might be inherited just as substantial (Hollywood et al., 1995). Two noteworthy classes of qualities, in particular oncogenes and tumor supressor qualities (TSGs) have been ensnared in malignant growth advancement (Macleod, 2000)). Typical mammalian cells contain cell qualities named proto oncogenes that assume a job in development, advancement and separation. Proto oncogene enactment to oncogene prompts uncontrolled ceil development and in the end neoplastic change. Oncogenes might be engaged with various phases of neoplasia. Some are associated with inception, where as others have a job in advancement, movement or metastasis; while a few oncogenes cause uncontrolled development by enacting determined development stimulatory signal transduction pathways, others modify basic hubs in the phone cycle {Park, 1998). Tumor supressor qualities keep up ordinary cell homeostasis by controlling cell multiplication, separation and apoptosis. Misfortune or inactivation of these qualities is

related with the advancement of harm (Macleod, 2000). The TSGs are arranged into two classes. Development inhibitory TSGs (GITSGs) and harm supressor qualities (MSGs) {Islam and Islam, 2000). GITSGs are malignant growth powerlessness qualities that have an opponent impact on cell development. These qualities contend with oncogenes to tie a typical factor to intercede their natural impacts. MSGs can cause tumor supression either by actuating cell separation or customized cell passing (Jansen Durr, 1996). Hereditary precariousness coming about because of mistakes in DNA replication and fix may incline a cell to the threatening phenotype. This can actuate oncogenes or erase districts containing TSGs. Loss of heterozygosity (LOH) is a corridor sign of TSG inactivation. Current proof recommends that intricate connections between different TSGs with oncogene are required for malignant growth movement (Liotta and Liu, 2001). Countless, including oncogenes, tumor silencer qualities or hormonal receptors, might be changed in bosom malignant growth, truth be told, gained or acquired variations from the norm in a wide assortment ofgenes have been ensnared in the pathogenesis of bosom disease (Isaacs, 1995; Jones et al., 1995 ; Kallioniemi and Visakorpi , 1996 ; Walker et al., 1997). Be that as it may, it ought to be underlined that the majority of these hereditary irregularities, incorporating those as of late portrayed in the PTEN/MMAC1 quality (Li et al., 1997 ; Steck et al., 1997 , Liaw et al., 1997), are not elite of bosom malignant growth and speaks to modifications in oneogenes or tumor silencer qualities normally changed in human tumors from various inceptions. All things considered, mutational investigations on certain qualities, including AR quality and those associated with inherited bosom malignancy (BRCA1, BRCA2), have given a few outcomes that might be of importance with regards to putative hereditary irregularities basic to bosom and prostate disease. The enthusiasm for AR as a potential factor basic to the two tumors emerges from late perceptions demonstrating that hereditary irregularities in this hormonal receptor are shared by these two hormonally subordinate tumors, yet in fact in just a little extent of patients (Bentel and Tilley, 1996 , Lobaccaro etal, 1993 ; Hall et al., 1996 ; Zhu et al., 1997).

AR alterations in breast cancer:

The AR is an interpretation factor that assumes a fundamental job in a wide number of natural capacities, from advancement and upkeep of male conceptive capacities to balance of resistant reactions or improvement of neural tissues (Chang et al., 1995). Like other atomic receptors, AR applies its organic impacts in the wake of official of flowing androgens principally transported to target tissues via bearer proteins (Quigley et al., 1995). Androgen restricting actuates a conformational change in the AR that encourages receptor homodimerization, atomic transport, and connection with DNA. The authoritative of the AR to the hormone reaction component (HRE) present in target qualities results in the guideline of their transcriptional movement (Desl Pere et al,, 1992). The structure of the AR is additionally like that of different individuals from the steroid receptor group of legand subordinate translation factors, with a N-terminal transactivating area (exon An), a focal DNAbinding space (exon B and C), and a C-terminal hormone restricting space (exons D through H) (Chang et al., 1988). The principal sign that AR may likewise be adjusted in bosom carcinoma was given by wooster et al, (Wooster et al., 1992), who detailed an AR germline transformation in two siblings with bosom malignant growth and Reifenstein disorder, a fractional androgen heartlessness disorder initially depicted as a X-connected familial disorder of hypospadias, barrenness, and gynecomastia in relationship with typical 17-Ketosteroid discharge and high FSH levels (Quigley et al., 1995). The transformation results in the substitution Arg 607 Gin, inside the area encoding the DNA-restricting space of the receptor. All the more as of late, Lobaccaro et al., (Lobaccaro et al., 1993) distinguished another germline change in the AR quality, in a man with lobular carcinoma of the bosom and incomplete androgen obtuseness disorder. In rundown, a progression of ongoing examinations performed by various gatherings has releaved that acquired and gained AR modifications may happen in bosom carcinomas. 1.8. BRCA1 and BRCA2 changes in bosom disease: Evidence for a hereditary part in bosom malignant growth chance was first noted by Paul Broca over one century prior, when he depicted four ages of bosom malignant growth in his significant other's family (Broca , 1866). From that point forward broad epidemiological investigation of bosom malignant growth cases that have all the earmarks of being bunched in families have been accounted for. The consequences of this investigation recommend that about 5% of bosom carcinomas might be clarified by acquired changes in at least one qualities. In spite of the hereditary heterogenecity of bosom malignancy and the high pervasiveness of sporadic sickness, a few bosom disease helplessness loci have been distinguished (Serova et al., 1997). The first of these qualities, named BRCA1, was mapped in 1990 to chromosome 17q 21 by hereditary linkage examination of substantial families that included numerous instances of earlyonset bosom carcinomas (Hall et al., 1990) and has been as of late recognized by Miki et al (1994) utilizing positional cloning techniques. BRCA1 is made out of 22 coding exons dispersed over more than 100kb of genomic DNA and encodes a 1863 amino corrosive protein, with two RING finger areas as its Nterminal part, which are believed to be engaged with DNA-authoritative or in proteinprotein associations. Furthermore, BRCA1 shares a rationed locale with 53 bpi (a p53 restricting protein) and rad 9 (a yeast protein engaged with the control of the DNA harm - instigated cell cycle capture), which has recommended that BRCA1 is probably going to work in the cell core and might be associated with at least one cell cycle check focuses (Koomn et al., 1996). Transformations in the BRCA1 quality are considered portion of the families defenseless to early beginning bosom malignant growth and for at any rate 80% of families with grouped bosom and ovarian diseases (Couch et al., 1996 , Szabo CI and King MC, 1997). Until this point, germlme BRCA1 transformations have been accounted for in excess of 200 families from various geographic starting points. Germline BRCA1 transformations have additionally been found in young ladies with bosom disease who don't have a place with families with different influenced individuals (Langston et al., 1995). All classes of changes are spoken to in these announced cases, including missense transformations, gibberish changes, cancellations, inclusions, or intronic changes, in spite of the fact that the larger part result in the generation of a truncated protein. The finding of this vast level of loss-of-work transformation is predictable with the theory

that BRCA1 goes about as tumor silencer quality. _lt is additionally momentous that a large portion of the detailed BRCA1 quality changes have been distinguished in a solitary family, yet a modest number have been recognized more than once. Specifically noteworthy is a frameshift change brought about by cancellation of an AG dinucleotide, which has been recognized in excess of 20 groups of Ashkenazi Jewish drops and is evaluated to happen at a recurrence of about 1% in this populace (Struewing et al., 1995; Fitz Gerald et a!., 1996).

The perception that not exactly a large portion of the families with different instances of bosom disease indicated linkage to BRCA1 prompted the recommendation that there was in any event an extra quality related with bosom malignant growth powerlessness. This outcome incited another genomic linkage seek and a second bosom malignant growth vulnerability quality, named BRCA2, was situated on chromosome 13q12 (Wooster et al., 1994) and thusly cloned (Tavtigian et al., 1996). BRCA2 is made out of 27 exons and encodes a protein of 3418 amino corrosive buildups, which does not give off an impression of being altogether like different proteins. Ongoing examinations have demonstrated that BRCA2 articulation is coordinately directed with BRCA1 articulation amid expansion and separation in mammary epithelial cells, recommending that the two qualities may act in a similar pathway (Rajan et al., 1996). So also to BRCA1, BRCA2 connects with Rad 51, giving extra help to the suggestion that these proteins might be fundamental cofactor in the Rod 51 - intervened fix of twofold strand breaks (Sharan et al., 1997). Truth be told, Connor et al (1997) have discovered proof of a DNA fix deformity in mice with a truncating BRCA2 change. Clinical investigations have uncovered that BRCA2 presumably represents an extent of early beginning bosom malignant growth generally equivalent to that subsequent from BRCA1, and it might be of uncommon significance in families with a high occurrence of male bosom disease, yet not in those with different instances of ovarian malignant growth. Mutational examination of the BRCA2 quality in various populaces has uncovered that as in BRCA1, the recognized changes are broadly appropriated all through the coding grouping of the quality, despite the fact that proof of some intermittent transformations has likewise been discovered (Couch et al., 1996 ; Phelan et al., 1996). Additionally of intrigue is the finding that BRCA2 changes in families with the most astounding danger of ovarian malignant growth in respect to bosom disease and grouped in a solitary exon of this quality (Gayther et al., 1997). At long last, and furthermore in the same way as BRCA1, assorted investigations have demonstrated that BRCA2 is a rare focus for substantial inactivation in bosom and ovarian tumors (Miki et al., 1996 ; Teng et al., 1996 ; Friedman et al., 1997)

4. Conclusion

In perspective on its ability for the recognition of SA in human salivation, Raman spectroscopy might be a promising strategy for bosom malignant growth conclusion. Utilizing the alignment bend acquired with reference fluid arrangements, this technique was connected to evaluation of SA in salivation of 106 solid ladies and 100 patients with bosom malignant growth. We found that the SA fixation in the salivation of the control bunch was 3.5 ± 1.0 mg□ dL−1 (middle, Q1, Q3: 3.4, 2.7, 4.5 mg□ dL−1) as opposed to 18.5 ± 9.7 mg□ dL−1 in the bosom malignant growth gathering (middle, Q1, Q3: 16.3, 12.1, 23.3 mg□ dL−1). This technique indicated affectability of 94%, particularity of 98%, and exactness of 92%. Based on these outcomes, we propose that salivary groupings of SA more noteworthy than 7 mg□ dL−1 might be characteristic (with high likelihood) of bosom malignancy or some other disease. We trust that this work has demonstrated the likelihood of building up an adaptable SERS-based technique that might be valuable for the determination of bosom malignancy. The reception of the SERS trial of human salivation by the medicinal network, exploiting the test's straightforwardness, its noninvasiveness, minimal effort, and materialness independent of age or bosom tissue thickness, holds guarantee as a noteworthy achievement in ladies' social insurance. This development may help to generously diminish the mortality brought about by one of the main sources of death among human females around the world.

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