However, there are some serious potential pitfalls related to the concept of interpretive reporting, e.g., training and expertise of the clinical chemist, the type of clinical laboratory, the association of the laboratory with academic institutions, and medicolegal liability.

An excellent example, for which all of these points are relevant, is prenatal testing for congenital anomalies by determination of alpha-fetoprotein in maternal serum (MSAFP). This area of testing is constantly changing, is being studied aggressively, and requires continual surveillance of the literature to keep abreast of the current consensus regarding interpretation of the MSAFP results. The training and expertise of the clinical chemist are important because clinical chemists may have their doctoral training in chemistry, microbiology, endocrinology, or other medically related areas. Endocrinologists or physicians would be most capable for interpreting these data because of their knowledge related to pregnancy.

The type of clinical laboratory may be important because some laboratories specialize in particular areas, i.e., a laboratory specializing in endocrine or reproductive endocrine testing. This type of highly specialized testing would most likely be found in association with an academic institution, which is the next consideration. In an academic setting, new or developing areas of testing are more apt to be found, laboratory directors with highly specialized training are usually present, and colleagues are available for discussion. In the case of MSAFP testing, a laboratory within a department of obstetrics & gynecology has faculty that maintain an academic interest in prenatal screening and make it possible to derive, through discussions of current consensus of the medical literature with regard to this assay.

Finally, medicolegal liability becomes a reality when interpretive reporting is practiced. It may be good that clinical laboratorians are willing to trust their data and interpretations of it to this extent, but they must also realize the potential extent of the commitment being made.

This certainly is not the only scenario under which there can be good interpretive reporting of MSAFP data. Clinical chemists trained in other areas can utilize consultants from a variety of places, including academic centers and private practice. They can also attend seminars and workshops and educate themselves in specific areas.

Dr. Killingsworth’s closing comments suggested that interpretive reporting would help establish a continuing dialog and foster a closer working relationship with physicians and nursing staff. As a director of a clinical reproductive endocrinology laboratory in an obstetrics & gynecology department of a large university medical center, I have always provided interpretive reporting. It makes life much more interesting and provides many more opportunities to interact with colleagues and to utilize your own skills and knowledge.

Reference

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More on Enzymes in Sjögren’s Syndrome

To the Editor:

We read with great interest the paper by Pal et al. (1) showing that 71% of patients with Sjögren’s syndrome (SS) show increased values for immunoactive trypsin (IRT) in serum, and 17% have high concentrations of pancreatic amylase in serum.

We studied 19 patients with SS (9 primary, 10 secondary) and found increased serum IRT in 63%, with absence of the salivary fraction of serum amylase in 29%, unrelated to IRT abnormalities. Similarly to Pal et al. (1), we observed that patients with pancreatic dysfunction tended to have a longer evolution of the syndrome (143 ± 40 months) than those with normal pancreatic function (92 ± 24 months). Pancreatic function was altered irrespective of the type of SS (primary or secondary) and of the degree of histological damage in labial biopsies, but there was an association between pancreatic involvement and salivary gland dysfunction as assessed by scintigraphy.

We also studied pancreatic exocrine function by means of the BT-PABA test (2), and found that four patients with increased IRT had a subnormal urinary PABA excretion, steatorrhea being present in two of them.

Initially we interpreted these findings as did Pal et al. (1), i.e., that pancreatic dysfunction was probably because of the same mechanism leading to impaired salivary gland function, namely, lymph cell infiltration around secretory ducts of the exocrine glands. Such an explanation was supported by prior work from our laboratory (3) showing that this same pattern of exocrine pancreatic dysfunction (high IRT and low PABA) could be detected in a substantial proportion of patients with chronic pancreatitis, in whom it appeared to be related to ductal or ductular obstruction in the face of a preserved exocrine glandular system and was, therefore, reversible with resolution of the obstruction (3).

However, when three of our patients with SS and impaired pancreatic function died and we had a chance to undertake histological studies of their pancreas, we found only mild fibrosis and minimal interstitial lymph cell infiltration. The pancreas of one patient also disclosed slight fatty change, while in another there were inconspicuous dilatations of medium-size ductules in addition to signs of necrotizing vasculitis in different stages of evolution.

On the basis of the evidence reported above, we believe that the probable cause of pancreatic dysfunction in SS should be sought in immunological alterations developing in ductular and acinar cells of the pancreas, a hypothesis already put forward by Ludwig et al. (4, 5).

References

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